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Third Edition

ADULT-GERONTOLOGY PRACTICE GUIDELINES

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This book is dedicated to my family and friends for always understanding and being there during the development of this book.

To Kaitlin and Carsen—Reach confidently towards your dreams and never look back!

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INSTRUCTOR RESOURCE

- Mapping to AACN Essentials: Core Competencies for Professional Nursing Education are available to qualified instructors by emailing textbook@springerpub.com

SECTION I

GUIDELINES

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CHAPTER 1

NORMAL PHYSIOLOGIC CHANGES IN THE AGING ADULT

L. Douglas Smith Jr.

INTRODUCTION

A. As we age, our bodies progress through a continuum of predictable changes to basic biologic processes affecting our ability to interact with the environment. Aging is a complex process that begins at conception and progresses through death. Aging is a heterogeneous process; no person ages the same as another, and organs within the same individual experience age-related changes at different times due to the unique confluence of genetic makeup, lifestyle choices, and environmental exposure through the life span. No singular theory adequately explains the holistic nature of the aging process—aging is the result of the complex interplay of many factors (e.g., biologic, psychologic, sociologic).

B. Routine age-related change is not synonymous with pathology. Ongoing discoveries suggest that many chronic maladies historically associated with aging (e.g., joint pain) may result from long-lived lifestyle choices more than changes related to age. The concept of successful aging considers an aged individual with good physical and cognitive function free of chronic disease. Still, aging is an inevitable process that results in predictable changes in physiologic function; the following is a brief discussion of common changes with age.

BRAIN AND NERVOUS SYSTEM

A. Anatomic changes in the central and peripheral nervous systems result in functional alterations in the autonomic and somatic nervous systems. Cerebral blood flow decreases, and there is a loss of compensatory mechanisms for normal fluctuation in blood pressure. Neuronal shrinkage and loss (most notable in the cerebellum and cerebral cortex) decrease overall brain volume. Accumulation of neurofibrillary tangles and neuritic plaques occurs routinely—although less than in Alzheimer disease. Significant decreases in the ability to synthesize and degrade neurotransmitters and a loss of myelin sheath result in impaired electrical transmission.

B. Observable effects due to anatomic changes in the nervous system vary between individuals. Muscular atrophy and strength decreases are seen due to decreased innervation and resting neurologic tone. Fine motor coordination and agility decrease; tremors may develop, making it difficult to perform activities of daily living. Changes to autonomic sensory neurons result in impaired proprioception, balance, and coordination, leading to a higher risk of falls. As a result of decreased nerve conduction speed, autonomic and somatic reflexes slow and contribute to delayed reactions times to touch and pain.

COGNITION

A. Cognitive impairment has long been thought normal in the aging process; however, this paradigm provides an overly simplistic understanding of aging on cognition. In a successfully aged person, parameters of cognitive function, including performance of well-practiced skills, retention of general knowledge, and recognition of familiar objects, remain stable over the lifetime. Changes in cognitive function associated with normal aging begin near the seventh decade of life and include decline in executive function, decreased attention span, difficulty reasoning in unfamiliar situations, and reduced processing of new information; still, successfully aged adults remain capable of functioning in society well beyond their 70th birthday.

SLEEP

A. Aging alters circadian patterns, resulting in changes in sleep and wakefulness. Changes in the thalamus, limbic, and reticular activating systems controlled by the hypothalamus result in a shift in normal rhythmic functions, creating sleep latency (delay in onset of sleep), reduced sleep efficiency (more time in bed when not asleep), increased nocturnal and early morning awakenings, and increased daytime sleepiness and napping. In addition, time spent in the deep and rapid eye movement stages of sleep decreases. These changes occur due to normal aging but may indicate underlying pathology (e.g., snoring or sleep apnea) or adverse drug effects.

EYES

A. Age-related changes to the eye and vision are typical and well-documented. Atrophy of periorbital fatty tissue and decreased resting tone result in ptosis or other malposition of the eyelids. Lacrimal gland changes result in reduced quantity and quality of tear production. The conjunctiva thins and may become yellow. In some adults, the cornea develops a noticeably yellow ring of fatty deposits known as arcus senilis. The iris stiffens, affecting its ability to change size, resulting in pupils that are smaller and more sluggishly responsive to light. The lens yellows and becomes opaque, scattering available light, while the retina becomes thinner due to changes ▶

in retinal photoreceptors and retinal nerve fiber thickening. Examination with the ophthalmoscope reveals narrowed and straightened blood vessels and gray and narrowed spots near the macula.

B. These anatomic differences result in commonly cited changes in vision. Dry and burning eyes result from reduced tear production, while the displacement of the lacrimal punctum may result in ineffective tear drainage and complaints of watery eyes. Changes to the iris and lens may result in difficulty reading up close (presbyopia) and other decreases in visual acuity. In addition, aged adults often experience reduced color and contrast discrimination and glare sensitivity.

C. Aging adults are at risk of three particular eye-related conditions due to normal aging. First, cataracts may develop from excessive protein accumulation in the lens over time. Cataracts appear as an opacity in the lens and interfere with the red reflex. Glaucoma results from increased intraocular pressure and may result in the gradual loss of peripheral vision. Finally, macular degeneration results from the breakdown of cells in the macula and leads to central vision loss and blindness while leaving the peripheral vision intact.

EARS

A. Ears are complex sensory organs responsible for input to the brain for hearing and balance. The ear's anatomy consists of three parts (external, middle, and inner), all of which experience changes during aging.

HEARING

A. Age-related changes affecting hearing include narrowing of the auditory canal, thickening of canal-lining hairs, and atrophy of the cerumen glands, resulting in thicker cerumen. Conductive hearing loss occurs as the tympanic membrane stiffens and calcification of ossicular joints occurs in the middle ear. Sensorineural hearing loss results from the loss of cochlear and auditory center innervation and stiffening of the basilar membrane. These age-related changes result in presbycusis, the gradual loss of hearing in both ears. Hearing loss occurs gradually and often presents as reduced ability to hear high-frequency sounds or impaired speech recognition in noisy settings. While hearing impairments may not be life-threatening, they can be disabling and negatively impact the quality of life.

BALANCE

A. The vestibule and semicircular canals of the inner ear, along with proprioceptive neurons of the central and peripheral nervous systems, coordinate balance. Sensory hair cell loss in the vestibule and changes in semicircular canal innervation impact balance in the aged adult. Aged adults often present with postural sway, complaints of vertigo, and have an increased risk of injury due to falls.

SMELL AND TASTE

A. Taste and smell are highly integrated senses allowing for sensory evaluation of the environment. Changes in their function carry significant implications for ingestion of food, personal safety, and personal hygiene. Age-related changes to these senses are incompletely understood but typically attributed to changes in the oral mucosa and nasal cavity (decrease in olfactory nerve fibers and taste buds), damage to cells throughout the life span (viral infections or environmental toxins), medication use, and diminished levels of neurotransmitters. The loss of smell impedes distinguishing spoiled foods, determining body odor, and identifying smoke in the environment. In addition, taste changes are likely to decrease interest in food and lead to weight loss.

CARDIOVASCULAR SYSTEM

A. As the cardiovascular system ages, expected changes in structure and function occur, including modest left atrial enlargement and hypertrophy and stiffening of the left ventricle, resulting in prolonged contraction time. Time in diastole lengthens to allow the stiffened ventricle longer to relax. Calcification and annular thickening of the aortic and mitral valves occur. There is a loss of pacemaker cells and fibrosis along the cardiac conduction system, leading to decreased responsiveness to adrenergic stimulation. Composition of the blood vessel changes to include increased collagen and decreased elastin, resulting in stiffer, less responsive blood vessels and coronary arteries.

B. Functional changes result from these structural changes. In general, the aged heart takes more time to recover systolic and conduction function between each beat. This is not significant while at rest; however, when stressed or at exercise, aged adults experience a decrease in maximum heart rate, cardiac output, and activity tolerance. While heart chambers enlarge, the overall heart size does not change. Valvular changes may result in nonconcerning systolic murmurs on auscultation. Due to differences in cardiac conduction, the aged adult may experience a higher rate of premature ectopic beats and are at a higher risk for atrial fibrillation. As the blood vessels stiffen, they become less responsive to baroreceptor signals and adrenergic stimulation, and the systolic blood pressure increases over time while the diastolic blood pressure remains consistent, resulting in widening pulse pressure.

RESPIRATORY SYSTEM

A. It is difficult to distinguish changes in the respiratory system solely linked to advanced age from those connected to environmental exposures over time. Changes in the respiratory status result from both changes in the lung and its ability to perform gas exchange and changes in structures assisting in ventilation. Rib and vertebrae osteoporosis and rigidities of the costal cartilage lead to limitations in thoracic movement and decreased chest wall compliance. The diaphragm flattens and becomes less efficient, resulting in pulmonary overdistention, and the recruitment of accessory muscles for adequate ventilation increases metabolic demands. Muscle weakness leads to a less vigorous cough. Age-related reductions in tracheobronchial cilia and immunoglobulin A reduce the ability to filter inhaled air and neutralize inhaled viruses.

B. Changes in the lung affect both ventilation and gas exchange. Enlargement of alveolar ducts from loss of elastic tissue results in a one-third decrease of surface area available for gas exchange. Lessened elasticity results in decreased vital capacity and increased residual volume. Changes in cardiovascular function and enlargement of the pulmonary artery lead to ventilation–perfusion mismatch; the weakened respiratory muscles become less able to move air into dependent alveoli where perfusion is greatest. Reduced effectiveness of gas exchange leads to rising carbon dioxide levels ▶

and decreasing oxygen levels in the blood, predisposing the aged adult to hypoxia and hypercapnia with less respiratory reserve capacity than when younger.

GASTROINTESTINAL SYSTEM

A. Age-related changes in the gastrointestinal (GI) system occur, although the effect on well-being is minimal. The GI tract includes all organs responsible for ingestion, digestion, absorption of nutrients, and excretion of solid waste from the body. GI-specific alterations exist; however, many changes associated with the GI system result from changes in other systems. For example, changes in the nervous system impact peristalsis and transit time, while changes in the cardiovascular system decrease mesenteric blood flow and absorption of nutrients. Specific age-related changes in the GI tract include gingival retraction and loss of teeth, decreased volume of saliva, decreased tone at the lower esophageal sphincter, reduced motility, atrophy of gastric mucosa, reduction of digestive enzyme excretion, decreased anal sphincter tone, and increased transit time (ingestion to excretion). Constipation and reflux gastritis are common complaints associated with age-related changes of the GI system.

METABOLISM AND BODY WEIGHT

A. Nutrients absorbed by the gastrointestinal system nourish the body's cells and provide energy for normal metabolic functions. As we age, the metabolism slows and the body requires less energy. In addition, hormonal changes cause the body to increase body fat stores and create less muscle mass, further decreasing the metabolic rate and increasing the chance for obesity. Throughout life, adults are encouraged to exercise most days of the week and monitor daily dietary intake to maintain a healthy body mass index between 18.5 and 24.9 kg/m^2.

ENDOCRINE SYSTEM

A. The endocrine system is a complex array of interlinked organs and glands closely linked to the nervous system. Because of the interconnectedness, it is challenging to identify gland-specific changes that occur over time. Decreases in endocrine function result in problems associated with metabolism, electrolytes, glucose, water, and minerals. Diabetes mellitus, hypothyroidism, osteoporosis, adrenal insufficiency, and various forms of hypopituitarism are some of the most common disease states associated with decreased endocrine function.

MUSCULOSKELETAL

A. The bones, muscles, and joints experience age-related changes. Calcium and mineral loss, in addition to inadequate intake of calcium and vitamin D, excessive alcohol and tobacco use, and decreased weight-bearing activity, lead to weakening of bones over time. Aging increases the chance of fracture and reduces repair speed when a fracture occurs. Joint stiffness and pain result from structure change, inflammation, and space narrowing. Muscle mass and strength decline due to hormonal changes and lack of physical activity. Muscle weakness, poor posture, joint compression, and brittle bones often reduce height by as much as 2 in. by the eighth decade of life.

GENITOURINARY SYSTEM

A. Aging of the renal system generally results in decreased efficiency of the urinary system. Kidney mass decreases and fibrotic changes occur in the parenchyma. Loss of nephrons in the renal cortex primarily affects those nephrons most important to maximal urine concentration and results in about 50% decrease in functional glomeruli in adults aged into the seventh decade of life. The nephrons remaining suffer a reduced filtering ability. Renal blood flow decreases in response to stiffened and thickened blood vessels. The loss of nephrons and decrease in blood flow result in reductions in glomerular filtration rate and creatinine clearance, leading to decreased ability to concentrate urine, manage electrolyte balance, and excrete toxic waste products. The ureters, bladder, and urethra also undergo age-related changes and include decreases in tone, elasticity, capacity, and sphincter tone, contributing to frequent urination, urinary urgency, and urinary incontinence. Females are at increased risk of urinary infections and males often experience difficulty with urination secondary to prostate enlargement.

REPRODUCTION AND SEXUALITY

SEXUALITY

A. Sexuality and intimacy are essential aspects of health and well-being throughout the life span. Decreasing hormones in both aged adults create significant and distressing changes to sexuality. There is a less rapid and extreme vascular arousal response to stimulation for both sexes. Time to orgasm increases, as does the refractory period after orgasm. Males may experience erectile dysfunction, premature ejaculation, less forceful ejaculations, and enlarging prostates. Females may experience anorgasmia, problems with arousal, and painful intercourse.

MENOPAUSE

A. Females reach menopause, the cessation of menses, commonly between their mid-40s and late 50s, with the average age at 51 years. Before menopause, the ovarian function declines, and irregular and lighter menses occur. These changes occur when the ovaries cease producing progesterone and estrogen. When this happens, reproduction is no longer possible. In addition, the hormonal changes decrease blood supply to the vagina and contribute to decreased vaginal secretion and lubrication during intercourse. Other changes associated with menopause include weakened pelvic muscles, thinning of the vaginal epithelium, and alkalinization of the vagina. These changes may lead to pain during sexual activity and increase the risk of infection.

BREAST

A. Changes to breast tissue occur as both males and females age. Postmenopausal females experience breast tissue atrophy due to decreases in sex hormones. Fibrous connective tissue replaces thinning tissue. As breast elasticity decreases, the breast decreases in size and may sag. Declining testosterone levels in males may lead to gynecomastia.

BLOOD AND BLOOD COMPONENTS

HEMATOPOIESIS

A. Outside the influence of pathology, the hematopoietic system maintains adequate function through the life span.

Baseline production and turnover of red blood cells and platelets do not change significantly; however, as with other systems, the hematopoietic ability to compensate for a loss (e.g., from phlebotomy) is less robust. Notably, platelets become hyperresponsive to thrombotic stimulators and several clotting factors increase, resulting in a slight but consistent decrease in bleeding time.

IMMUNE SYSTEM

A. The immune system weakens with aging due to a weakened ability to make antibodies to disease. The thymus gland, responsible for T-cell activating hormones, atrophies and weakens. Peripheral T-cells decrease, increasing the risk of developing infections and making infections more severe. Aged clients greatly benefit from adherence to immunization schedules recommended by the Centers for Disease Control and Prevention.

SKIN, HAIR, AND NAILS

A. Normal skin aging leads to atrophy, decreased elasticity, and impaired reparative responses. The epidermis becomes thin, flattens, and loses elasticity, leading to fine lines and wrinkles. The epidermis plays a crucial role in vitamin D synthesis, and as the epidermis thins the aged person's ability to synthesize vitamin D decreases. Skin hydration is affected as sweat, oil, and sebaceous glands become less active. The loss of subcutaneous fat tissues throughout the body reduces insulation against cold, and the loss of protective fat pads in the feet increases the risk of a foot injury. Wound healing is delayed with age. Hair changes are related to decreased follicle density and function. Hair growth slows and hair thins. Hair loss occurs most commonly in the scalp, axilla, and pubic areas. Hair color may gray and females may begin to grow facial hair. Fingernails become thin and tear easily, while toenails grow thick.

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CHAPTER 2

HEALTHY LIVING FOR THE ADULT-GERIATRIC CLIENT

Ann McQueen Blair and Kathleen Bradbury-Golas

HEALTH MAINTENANCE DURING THE LIFE SPAN

A. Health maintenance involves identifying individuals at risk for health problems and encouraging behaviors that reduce these risks. An important aspect of health maintenance is client education, including teaching individuals about their risk factors for disease and ways to modify their behaviors to reduce their risks of comorbidities. Client teaching guides may be downloaded by the practitioner, filled according to the client's evaluation and needs, and given to the client.

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ADULT RISK ASSESSMENT FORM

A. The Adult Risk Assessment Form (Exhibit 2.1) should be used for all adult clients to evaluate their risk for particular diseases. The practitioner should interview the client, assessing for the risk factors listed on the Adult Risk Assessment Form. The family history of first-degree relatives (parents, siblings, and children) should also be discussed because many diseases are related to genetic factors. Keep a copy of the Adult Risk Assessment Form in the front of the client's chart

EXHIBIT 2.1 Adult Risk Assessment Form

Name: _____ DOB: _____ Chart #: _____

Allergies: _____

Occupation: _____

Family History **First-degree relatives with remarkable diseases (e.g., hypertension, DM, CAD, cancer, and thyroid)**			
1.		6.	
2.		7.	
3.		8.	
4.		9.	
5.		10.	

CAD, coronary artery disease; DM, diabetes mellitus.

Assess the client for the following personal risk factors:

A. Coronary heart disease:
1. High-fat/high-cholesterol diet.
2. Obesity.
3. Elevated cholesterol level.
4. Stroke.
5. Hypertension.
6. Tobacco use.

B. Lung cancer:
1. High-fat/high-cholesterol diet.
2. Tobacco use.

C. Cervical cancer:
1. Early age of first intercourse.
2. Multiple sexual partners.
3. History of human papillomavirus.

D. Breast cancer:
1. Nulliparous.
2. Primigravida after the age of 35.
3. High-fat diet.

E. Colon cancer:
1. History of polyps.
2. High-fat diet.

F. Osteoporosis:
1. Less than 1 g of calcium per day.
2. History of tobacco or alcohol use.
3. Sedentary lifestyle.
4. Thin, Caucasian.
5. Female gender.

The contributions of Cheryl A. Glass to this chapter in prior editions are acknowledged here.

EXHIBIT 2.1 Adult Risk Assessment Form (*continued*)

G. **Glaucoma/visual impairment:**
   1. Family history of glaucoma.
   2. Diabetes mellitus.

H. **Sexually transmitted infections (STIs)/HIV:**
   1. Alcohol and drug use or abuse.
   2. Multiple sexual partners.
   3. Homosexual or bisexual partner.
   4. History of intravenous drug use/needle sharing.
   5. History of blood transfusion.
   6. Exposed to or history of STI.
   7. Exchanging sex for drugs or money.

I. **Substance abuse:**
   1. Alcohol or drug use history including "street drugs" and opioids.
   2. Family history of substance abuse.
   3. Stress or poor coping mechanisms.
   4. Administer the **CAGE** Assessment: Have you ever tried to **C**ut down on your alcohol/drug use? Do you get **A**nnoyed if someone mentions your use is a problem? Do you ever feel **G**uilty about your use? Do you ever have an **E**ye-opener first thing in the morning after you have been drinking or using the night before?

J. **Accidents and suicide:**
   1. Previous suicide attempt.
   2. Family history of suicide.
   3. Alcohol use.
   4. Substance use.
   5. History of depression.
   6. High-stress or "hot-reactor" personality.
   7. Male gender.
   8. Poor coping mechanisms or stress.

K. **Safety:**
   1. Does not use seat belt or car seat.
   2. Drinks and drives.
   3. Drives over the speed limit.
   4. Does not wear safety helmet if driving motorcycle.
   5. Inadequate number of smoke detectors or none in the home.

and update yearly or as needed. When complete, this tool can guide the practitioner in determining the assessment needs of each client. If using electronic medical records, a special section should be identified for risk assessment.

ADULT PREVENTIVE HEALTHCARE

A. Exhibits 2.2 and 2.3 help the practitioner identify changes in the adult client's risk factor status, make recommendations for health maintenance (e.g., immunizations, laboratory work, physical exams), and educate the client on prevention. The guide can be used as a quick reference for the practitioner to evaluate the client's adherence to preventive measures. Keep a copy of this flow sheet and guide in the front of the client's chart where they can be reviewed routinely and updated as necessary. If using electronic medical records, a special section should be identified as routine health maintenance.

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ADULT-GERIATRIC SCREENING RECOMMENDATIONS

A. Health promotion includes utilization of preventive screen testing. Multiple guidelines are available for clinicians to utilize during guidance and discussion of recommendations with clients. Professional societies, the U.S. Public Health Service, and other organizations evaluate the strength of evidence and the weight of clinical research, statistical analysis, and the risk and benefits of testing to make recommendations for screening. Most of the recommendations for this section are from the U.S. Preventive Services Task Force (USPSTF) guidelines.

B. Eligibility and Medicare coverage for screening are summarized in Table 2.1.

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ABDOMINAL AORTIC ANEURYSM

A. The Centers for Disease Control and Prevention (CDC) notes abdominal aortic aneurysms (AAAs) were the primary cause of death in 59% of deaths in 2019. The main risk factors for development of AAAs are age, sex, smoking, and family history. A history of smoking occurs in 75% of those with AAA. About two-thirds of people who have an aortic dissection are male. Older males who have smoked are at the highest risk of developing an AAA. Dissections and ruptures are the cause of most deaths from aortic aneurysms. An AAA is often asymptomatic; however, the symptoms may include a sharp, sudden pain in the chest; throbbing or deep pain in the back or side, and/or pain in the buttocks, groin, or legs; trouble breathing or swallowing; or syncope. Nearly two-thirds of aneurysms leading to surgery were detected as incidental findings on imaging studies performed for other indications. On physical examination, a pulsatile mass in the epigastrium where the aorta bifurcates at the umbilicus may be palpated.

B. There are several types of aneurysm: thoracic aneurysms, abdominal aneurysms, peripheral aneurysms, and those that occur in the brain, which may cause a stroke. An AAA is an enlarged ballooning area that is more than 50% of the normal diameter of the aorta. The U.S. Preventive Services Task Force (USPSTF) notes that people who are screened are about twice ▶

EXHIBIT 2.2 Adult Preventive Healthcare Flow Sheet

Immunization Schedule				
Immunization or Positive Titer	**Date**	**Date**	**Date**	**Date**
Tdap (tetanus, diphtheria, and pertussis) booster				
MMR				
TB (yearly as indicated)				
HepB				
Influenza (yearly)				
Pneumococcal (PCV15, PCV20, PPSV 23)				
Varicella (14–49y)				
Zoster recombinent (>50y)				
COVID-19				
Other				

Assess clients for the following behaviors:

Risk Assessment			
Date	**Date**	**Date**	**Date**

Clients should be educated about any behavior modifications that can reduce their risk factors for health problems. The practitioner should note the date as well as the type of counseling given to the client.

Client Education				
Behavior Modification	**Date**	**Date**	**Date**	**Date**
Diet/exercise				
Tobacco/alcohol				
Injury prevention				
Skin protection				
Hormone therapy				
Safe sexual practices				
Occupational hazards				
Self-exam: testicular				

HepB, hepatitis B; MMR, measles, mumps, rubella; TB, tuberculosis.

as likely to have AAA surgery within 3 to 5 years compared with people who are not screened. The 2019 USPSTF guideline on Abdominal Aortic Aneurysm: Primary Care Screening recommendations for AAA screening include the following:

1. Males 65 to 75 years of age who have ever smoked (100 cigarettes in a lifetime) should have a onetime ultrasound screening for AAA even if they are asymptomatic.

2. Males ages 65 to 75 years who have never smoked can be selectively offered screening by clinicians rather than routinely screening all males in this group. Screening should be based on medical history, family history, other risk factors, and client preferences.

3. The current evidence is insufficient to assess the balance of benefits and harms for the USPSTF to recommend that females ages 65 to 75 who have ever smoked be screened for AAA.

4. The USPSTF recommends that females who smoke or have never smoked should not undergo AAA screening. AAA is uncommon in older females and is very rare in females who have never smoked. The Society for Vascular ▶

EXHIBIT 2.3 Adult Health Maintenance Guide

Name: _____ DOB: _____ Chart #: _____

Allergies: _____

Occupation: _____

The following tests should be performed according to the individual client's risk factors as part of preventive healthcare. The practitioner should fill in the date and result of each test and highlight any remarkable results.

Test	Date	Result	Date	Result	Date	Result	Date	Result
Height								
Weight								
BMI								
BP								
Dermatology exam								
Oral cavity exam								
Echocardiogram								
TSH								
Lipid profile								
Urinalysis								
Digital rectal exam								
Hemoccult								
Colonoscopy								
PSA								
Testicular exam								
Pelvic exam/Pap smear								
Clinical breast exam								
Mammogram								
STI/HIV								
Foot examination for diabetics								
Additionally, the practitioner should ensure that the client receives other healthcare as needed. Assess client's adherence to other preventive healthcare exams.								
Dental exam								
Vision/glaucoma exam								

BMI, body mass index; BP, blood pressure; PSA, prostate-specific antigen; STI, sexually transmitted infection; TSH, thyroid-stimulating hormone.

Surgery (SVS) 2018 guidelines recommend males and females older than 75 years with a history of tobacco use who are in otherwise good health and have not previously been screened with ultrasound have a onetime ultrasound screen for AAA.

5. The ultrasound is the gold standard modality for screening. CT scans are not recommended for first-line screening due to the high cost and radiation exposure.

6. Aneurysm size:

a. Small aneurysms have a diameter of < 3.0 cm.

b. Medium aneurysms have a diameter between 3.1 and 5.4 cm.

i. The SVS guidelines recommend observation for asymptomatic AAA < 5.4 cm in diameter.

c. Large aneurysms have a diameter ≥5.4 cm.

i. For asymptomatic clients, the risk of AAA rupture generally does not exceed the risk associated with elective AAA repair until aneurysm diameter exceeds 5.5 cm.

d. Very large aneurysms have a diameter ≥6.0 cm.

TABLE 2.1 MEDICARE COVERAGE AND ELIGIBILITY FOR SCREENING: CURRENT AS OF 2018

Test/Screen	Medicare Part B Coverage Eligibility
Preventive visit	"Welcome to Medicare" preventive visit available for Medicare coverage within the first 12 months of obtaining Medicare Part B • Measurement of height, weight, and blood pressure • Calculation of BMI • Simple vision test • Review of potential risk for depression and level of safety • Offer to talk about creating advance directives • Written plan on which screening, shots, and other preventive services are needed
Yearly "Wellness" visits	Annual "Wellness" visits for Medicare patients with Part B for longer than 12 months; used to develop or update a personalized prevention plan to prevent disease and disability based on current health and risk factors • Completion of an HRA • Review of medical and family history • Development or update of the list of current providers and prescriptions • Measurement of height, weight, blood pressure, and other routine measurements • Evaluation for cognitive impairment • Personalization of health advice • Listing of risk factors and treatment options • Development of screening schedule for appropriate preventive services
AAA screening	Onetime AAA ultrasound if the client meets the eligibility requirements; requires referral Risk factors for eligibility: • Family history of AAA • Male 65–75 years of age and have smoked at least 100 cigarettes/lifetime
Bone mass measurement	Covered screen once every 24 months for clients who meet the eligibility criteria: • Females whose PCP determines both of these (based on medical history and other findings): ▪ Estrogen deficient ▪ At risk of osteoporosis • X-rays showing possible osteoporosis, osteopenia, or vertebral fractures • Clients taking prednisone or steroid-type drugs or planning to begin this treatment • Clients who have been diagnosed with primary hyperparathyroidism • Clients being monitored to evaluate whether the osteoporosis drug therapy is effective Covered more often if medically necessary
Alcohol misuse screenings and counseling	Covered screen once per year for clients who meet the eligibility criteria: • Adults with Part B (including pregnant females). ▪ Use alcohol ▪ Do not meet the medical criteria for alcohol dependence • Four brief face-to-face counseling sessions per year if the following apply: ▪ Alcohol misuse determined by the PCP ▪ Competent and alert during counseling • Counseling must be provided: ▪ By a qualified PCP ▪ In a primary care setting
Cardiovascular disease (behavioral therapy)	One visit per year in PCP office or primary care clinic • Check blood pressure • Discuss aspirin use (if appropriate) • Discuss dietary information
Cardiovascular disease screening	Screening blood tests every 5 years • Cholesterol • Lipids • Triglycerides
Cervical and vaginal cancer screenings	Pap tests, pelvic exams, and clinical breast exams covered by Part B • Once every 24 months for all females • Once every 12 months if one of the following applies: ▪ High risk for cervical or vaginal cancer ▪ Childbearing age and have had an abnormal Pap test in the past 36 months

(*continued*)

TABLE 2.1 MEDICARE COVERAGE AND ELIGIBILITY FOR SCREENING: CURRENT AS OF 2018 (*CONTINUED*)

Test/Screen	Medicare Part B Coverage Eligibility
Colorectal screening	FOBT: • Coverage with a referral from a physician, physician assistant, nurse practitioner, or clinical nurse specialist Multitarget stool DNA test: • Covered once every 3 years if **all conditions** are met: ▪ 50–85 years of age ▪ No signs or symptoms of colorectal disease including, but not limited to: – Lower gastrointestinal pain – Blood in stool – Positive guaiac fecal occult blood test – Fecal immunochemical test ▪ At average risk for developing colorectal cancer, meaning: – No personal history of the following: • Adenomatous polyps • Colorectal cancer • Inflammatory bowel disease, including Crohn disease and ulcerative colitis – No family history of the following: • Colorectal cancers or adenomatous polyps • Familial adenomatous polyposis • Hereditary nonpolyposis colorectal cancer Screening flexible sigmoidoscopy: • Once every 48 months for most clients 50 years or older • If not high risk, is covered 120 months after a previous screening colonoscopy Screening colonoscopy: • Once every 24 months if at high risk for colorectal cancer • If not at high risk, is covered once every 120 months, or 48 months after a previous flexible sigmoidoscopy • A screening colonoscopy can become a diagnostic colonoscopy when: ▪ Results in biopsy or removal of a lesion or growth ▪ The biopsy or removal happens during the same visit as the screening colonoscopy Barium enema: • When utilized instead of a flexible sigmoidoscopy or colonoscopy: ▪ Coverage every 48 months if 50 years or over ▪ Coverage every 24 months and are at high risk for colorectal cancer
Dental	Routine dental care or dental procedures such as cleanings, fillings, tooth extractions, dentures, dental plates, or other dental devices not covered by Medicare
Depression screening	One depression screening per year covered by Medicare; screening must be done in a primary care setting that can provide follow-up treatment and/or referrals
Diabetes screening	Screening to test for diabetes covered by Medicare • Based on the screening test, may be eligible for two diabetes screenings each year • Screenings covered if any of the following risk factors exist: ▪ Diagnosed with prediabetes ▪ Hypertension ▪ History of abnormal cholesterol and triglycerides ▪ Obesity ▪ History of high glucose • Covers these tests if two or more of the following apply: ▪ Age 65 or older ▪ Overweight ▪ Family history of diabetes (parents, brothers, sisters) ▪ History of gestational diabetes or delivery of a baby weighing more than 9 lbs • If covered with Medicare Part B and have prediabetes, the client may qualify for diabetes prevention program services to help make health behavior changes that can prevent or delay the onset of diabetes. The program begins with at least 16 core sessions over a 6-month period, which are offered in a group setting. After the core sessions, additional monthly sessions in the diabetes prevention sessions include the following: ▪ Tips on how to get more exercise ▪ Strategies for weight control ▪ Support from people with similar goals and challenges ▪ A lifestyle coach, specially trained in motivation

(*continued*)

TABLE 2.1 MEDICARE COVERAGE AND ELIGIBILITY FOR SCREENING: CURRENT AS OF 2018 (*CONTINUED*)

Test/Screen	Medicare Part B Coverage Eligibility
DSMT	Outpatient DSMT to teach coping and management of diabetes • May cover up to 10 hours of initial DSMT; training may include 1 hour of individual training and 9 hours of group training • May also qualify for up to 2 hours of follow-up training each year if it takes place in a calendar year after the year of initial training • Training includes the following: ▪ Tips for eating healthy, being active, taking drugs, and reducing risks • Must have a written order from a PCP or other healthcare provider for certain clients with Part B who are at risk for complications from diabetes • Some exceptions apply: ▪ Group sessions are not available or if the provider notes special needs that would be better met in individual training sessions ▪ If living in a rural area, may get DSMT services from a practitioner, such as a registered dietitian in a different location through telehealth
Hepatitis C screening test	Covers one hepatitis C screening test if a PCP or practitioner orders it Also covers yearly repeat screening for certain clients at high risk who meet one of the following conditions: • High risk because they use or used illicit injection drugs • Had a blood transfusion before 1992 • Born between 1945 and 1965
HIV screening	Voluntary HIV screenings, one test once every 12 months Covered for clients who meet the criteria: • 15–65 years old • Younger than 15 or older than 65 and at increased risk • Pregnant ▪ Covered up to three times during a pregnancy ▪ Interval testing during pregnancy not defined by CMS
Immunizations	Flu shot: • Normally covers one flu shot per flu season given in the fall or winter HepB: • Clients at high or medium risk for HepB: ▪ Some risk factors noted as increased risk defined by Medicare: hemophilia, ESRD, diabetes, healthcare workers who have frequent contact with blood or bodily fluids, and other factors that increase risk for HepB (check with PCP to see if at high or medium risk) Pneumococcal shot: • One pneumococcal shot to prevent infections • A different second shot covered by Part B if it is given 1 year (or later) after the first shot • Discuss with the PCP or other healthcare providers to evaluate need for one or both shots
Lung cancer screening	A lung cancer screening with LDCT once per year for clients who meet all of the following conditions: • Age 55–77 • Asymptomatic (no signs or symptoms of lung cancer) • Either a current smoker or have quit smoking within the last 15 years • Tobacco smoking of at least 30 "pack-years" (an average of one pack a day for 30 years) • Written order from their PCP Before the first lung cancer screening, an appointment needs to be scheduled to: • Discuss the benefits and risks of lung cancer screening • Decide whether lung cancer screening is right for the client
Mammography (medical)	Screening mammogram once every 12 months (11 full months must have passed since the last screening) Diagnostic mammogram when medically necessary • Covers females with Medicare 40 years of age or older • One baseline mammogram for females with Medicare 35–39 years of age

(*continued*)

TABLE 2.1 MEDICARE COVERAGE AND ELIGIBILITY FOR SCREENING: CURRENT AS OF 2018 (*CONTINUED*)

Test/Screen	Medicare Part B Coverage Eligibility
Nutrition therapy services	MNT services and certain related services: • Can be provided by a registered dietitian or nutrition professional who meets certain requirements • Can only be referred by a PCP • MNT services may include the following: ▪ An initial nutrition and lifestyle assessment ▪ Individual and/or group nutrition therapy services ▪ Follow-up visits to check on progress managing diet • In a rural area, may be provided through telehealth; a registered dietitian or other nutrition profession, in a different location, would provide the service • MNT covered by Medicare as part of overall dialysis care if the dialysis is done in a dialysis facility • Eligibility includes clients with Part B who meet at least one of the following conditions: ▪ Have diabetes ▪ Have kidney disease ▪ Have had a kidney transplant in the last 36 months
Obesity screening and counseling	Behavioral counseling for weight loss covered by Medicare; counseling may be covered if given in a primary care setting (such as a PCP's office) • Counseling required in a primary care setting so that it can be coordinated with other care and a personalized prevention plan • Covers all clients with a BMI of 30 or more
Prostate cancer screening	All males older than 50 (beginning the day after their 50th birthday) Digital rectal examination: • Once every 12 months PSA test: • Once every 12 months
STI screening and counseling	Screening for chlamydia, gonorrhea, syphilis, and HepB • Once every 12 months • At certain times during pregnancy Up to two individual 20- to 30-minute, face-to-face, high-intensity behavioral counseling sessions covered by Medicare each year for sexually active adolescents and adults at increased risk for STIs if referred by a PCP
Smoking and tobacco use cessation	Counseling for up to eight face-to-face visits in a 12-month period performed by a qualified PCP or other Medicare-recognized practitioner All clients with Part B who use tobacco eligible for counseling
Vision	Routine eye examinations for eyeglasses or contact lenses not covered by Medicare Covers AMD testing Vision screen for glaucoma: • Glaucoma test every 12 months for clients at high risk for glaucoma: ▪ Diabetes ▪ Family history of glaucoma ▪ African Americans and those 50 years or older ▪ Hispanic Americans and those 65 years or older Vision screen for retinopathy: • Yearly exam for diabetic retinopathy • Clients on Medicare who have diabetes

Note: Consult the Medicare website for full coverage information and costs: www.medicare.gov/coverage/preventive-and-screening-services.html.

AAA, abdominal aortic aneurysm; AMD, age-related macular degeneration; BMI, body mass index; CMS, Centers for Medicare and Medicaid Services; DSMT, diabetes self-management training; ESRD, end-stage renal disease; FOBT, fecal occult blood test; HepB, hepatitis B; HRA, health risk assessment; LDCT, low-dose CT; MNT, medical nutrition therapy; PCP, primary care physician; PSA, prostate-specific antigen; STI, sexually transmitted infection.

7. Management consists of ongoing clinical evaluation and AAA surveillance and risk modification. Aneurysm surveillance from the SVS supports the following surveillance intervals:

a. Initial ultrasound screening for aortic diameter >2.5 cm but <3.0, rescreening after 10 years.

b. For AAA 3.0 to 3.9 cm, imaging at 2- to 3-year intervals.

c. For AAA 4.0 to 5.4 cm, imaging at 6- to 12-month intervals.

d. For AAA 5.0 to 5.4 cm, imaging at 6-month intervals, with surgical consult/referral for possible elective repair.

8. Aneurysm diameter is the strongest predictor of aneurysm rupture, with risk increasing markedly at aneurysm diameters >5.4 cm. Rapid expansion, which is thought to increase the risk of rupture, is defined as an increase in maximal aortic diameter ≥5 mm over a 6-month period of time or >10 mm over 1 year, using the same radiographic method of measurement. ▶

9. AAA surgical repair:

a. The decision for elective repair of asymptomatic AAA relies primarily on determining risk of rupture based on aneurysm diameter >5.4 cm. However, the diameter threshold for repair is not absolute. An elective AAA repair should also take into account the natural history of AAA and factors that increase the risk of aneurysm expansion or rupture.

b. Repair is indicated in clients with AAA who are symptomatic (tenderness or abdominal or back pain, evidence for embolization, rupture), have an AAA ≥5.4 cm, or an AAA that has expanded >0.5 cm within a 6-month interval.

c. There are two types of surgical repair:

i. Open surgical repair is the only proven intervention that has decreased AAA mortality. The open surgical repair of a large AAA has a mortality rate of 4% to 5%, with nearly one-third having pulmonary and cardiac complications.

ii. Endovascular aneurysm repair (EVAR) with stent grafts is the alternative surgical procedure to open repair. A late conversion to an open repair may be required. The overall complication rate for EVAR, including access-related issues, is approximately 10%.

d. An individualized approach to the client when choosing between open and EVAR repair needs to take into account the client's age, risk factors for perioperative morbidity and mortality, anatomic factors, and experience of the surgeon.

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BONE MASS DENSITY MEASUREMENT FOR OSTEOPOROSIS

A. Osteoporosis and low bone mass are currently estimated to be a major public health threat for almost 44 million U.S. females and males 50 years of age and older. The incidence of fragility fractures increases exponentially with age, although the increase begins approximately 10 years later in males. However, the mortality rate associated with hip fractures, as well as vertebral and other major fractures, is higher in males than in females. Falls cause nearly 90% of osteoporotic fractures. Older adult clients have increased susceptibility to fractures because of other medical problems, side effects of medications, and older adults' tendency to fall. Falls cause nearly 90% of osteoporotic fractures. Fractures may cause chronic pain and disability and affect the functional ability and quality of life: Mortality and morbidity are highest with a hip fracture. For an in-depth review of osteoporosis, refer to the "Osteoporosis/Kyphosis/Fracture" section of Chapter 22.

B. The Fracture Risk Assessment Tool (FRAX) calculates the absolute fracture risk or estimates the probability of fracture. The algorithms from the FRAX calculate the probability of hip fracture and the 10-year probability of a major osteoporotic fracture (clinical spine, forearm, hip, or shoulder fracture). The FRAX assessment includes questions about age, smoking, family history of hip fracture, long-term glucocorticoid use, and low body weight. The FRAX score is used to drive decisions for testing and prescribing osteoporosis medications.

C. The Foundation for Osteoporosis Research and Education (FORE) FRAX score 10-Year Fracture Risk Calculator at riskcalculator.fore.org or at the University of Sheffield, Great Britain website at www.sheffield.ac.uk/FRAX. The National Osteoporosis Foundation (NOF) Clinician's Guide to Prevention and Treatment of Osteoporosis is available at bonehealthandosteoporosis.org.

Screening through the identification of risk factors does not diagnose osteoporosis but identifies people who require further evaluation.

D. Bone mass measurement screening is done through several tests:

1. A standard x-ray is inadequate to screen for osteoporosis.

2. The gold standard test for measurement of bone mineral density (BMD) is the use of the central dual-energy x-ray absorptiometry (DXA) of the hip and lumbar spine.

a. The central DXA of the radius bone and the peripheral bone density of the heel or another bone may be required for the morbidly obese client.

3. Quantitative ultrasonography (QUS) of the calcaneus is also utilized for measurement.

a. The measurement cutoffs for the QUS and the DXA are not interchangeable.

b. A limitation of ultrasound is that it does not reliably exclude or confirm DXA-determined osteoporosis.

c. Because of limited precision and slow rate of change of bone mass at peripheral sites, quantitative heel ultrasound cannot be reliably used to follow clients who are treated for osteoporosis.

d. An individual with a high-risk ultrasound finding needs a confirmatory DXA to determine the need for treatment based on established guidelines and as a baseline for monitoring therapy.

4. The peripheral quantitative CT (pQCT) is available but is not recommended for screening. QCT measures volumetric bone density of the spine and hip and can analyze cortical and trabecular bone separately. The application of T-scores to predict the risk of fracture has not been validated with QCT. Additionally, the test is more costly and results in greater exposure to radiation than DXA.

E. The recommendations for who to screen and screening intervals of 1 to 2 years vary by different organizations, including the NOF, the World Health Organization, and the American College of Obstetricians and Gynecologists (ACOG). ▶

The U.S. Preventive Services Task Force (USPSTF) guideline on osteoporosis screening is currently being updated (public comment closed August 2021); the USPSTF guideline is current as of July 2015. The USPSTF recommends screening for osteoporosis by sex and age:

1. Females:
   a. Age 65 years or older.
   b. The USPSTF does not define the upper age limit for screening in females because the risk of fractures increases with age.
   c. According to the draft 2018 recommendation, the USPSTF recommends screening for osteoporosis with bone measurement testing to prevent osteoporotic fractures in postmenopausal females younger than 65 years who are at increased risk of osteoporosis, as determined by a formal clinical risk assessment tool. The NOF, International Society for Clinical Densitometry (ISCD), Endocrine Society, and Canadian Osteoporosis Society recommend bone density testing for females older than 50 years with any clinical risk factors for fractures.
2. Males:
   a. Routine screening is currently a widespread practice.
   b. The USPSTF concludes that the current evidence is insufficient to assess the balance of benefits and harms of screening for osteoporosis to prevent osteoporotic fractures in males. The USPSTF found inadequate evidence that drug therapies reduce the risk of fracture in males who have no previous osteoporotic fracture.
3. The NOF, ISCD, Endocrine Society, and Canadian Osteoporosis Society recommend bone density testing for all males age 70 years or older and for males 50 to 69 years based on their risk factor profile.

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CARDIOVASCULAR DISEASE AND CARDIAC SCREENING (CARDIAC CALCIUM SCORE)

A. The U.S. Preventive Services Task Force (USPSTF) has multiple screening and prevention recommendations for cardiac and cardiovascular disease (CVD). Refer to Chapter 13 for an in-depth review of cardiac conditions.

1. Screening recommendations:
   a. Screen for high blood pressure (BP) in adults 18 years of age and older.
      i. The recommendation includes measurements outside of the clinical setting for diagnostic confirmation before starting treatment.
      ii. Annual screening is recommended for adults age 40 years or older and for those who are at increased risk for high BP. Persons at increased risk include those who have high normal BP (130–139/85–89 mmHg), those who are overweight or obese, and African Americans.
      iii. Adults age 18 to 39 years with normal BP (<130/85 mmHg) who do not have other risk factors should be rescreened every 3 to 5 years. The USPSTF recommends rescreening with properly measured office BP and, if BP is elevated, confirming the diagnosis of hypertension (HTN) with ambulatory blood pressure measurement (ABPM).

The USPSTF recommends ABPM as the reference standard for confirming the diagnosis of HTN. Ambulatory BP monitoring devices record BP at regular intervals over 12 to 24 hours while the client goes about their normal activities and while sleeping. Elevated ambulatory systolic BP is consistently and significantly associated with increased risk for fatal and nonfatal stroke and cardiovascular (CV) events, independent of office BP. Ambulatory BP monitoring can be performed every year in high-risk persons and every 3 to 5 years in those at low risk (adults 18–39 years of age with no risk factors).

   b. **Lipid screening guidelines have currently been updated.** There are no sex-specific recommendations for screening for lipid disorders. The following are the age specifics for screening:
      i. The USPSTF recommends lipid screening in both males and females who are at increased risk for coronary heart disease (CHD), males aged 35 years and older, or females aged 45 years and older. Risk factors include dyslipidemia, diabetes, high BP, and smoking.
         1) 10-year CV event risk of ≥10%: Low- to moderate-dose statins are recommended.
         2) 10-year CV event risk of 7.5% to 10%: The recommendation is for selectively offering treatment using an individualized approach.
      ii. Lipid screening can also be completed in clients 20 to 35/45 years of age with increased risk for CHD. Currently, no guidelines support routine screening of lipids in young adults aged 20 to 35 years without risks of CHD.
      iii. For adults 76 years of age and older with no history of CVD, there is insufficient evidence to make a recommendation.
   c. Screen for CHD with EKG: The USPSTF found inadequate evidence to determine whether adding resting or exercise EKG to conventional risk factor assessment leads to improved risk stratification of persons to inform treatment decisions.
      i. Adults at low risk of CVD events: The USPSTF recommends against screening with resting or exercise EKG for prediction of CVD events in asymptomatic adults at low risk for CVD events.
      ii. Adults at intermediate or high risk of CVD events: Currently, there is insufficient evidence to assess the balance of benefits and harms of screening with resting or exercise EKG to prevent CVD events in asymptomatic adults at intermediate or high risk for CHD events.
   d. The current evidence is insufficient to assess the benefits and harms of screening for peripheral ▶

artery disease and CVD risk assessment with the ankle-brachial index (ABI) in adults.

i. There are no studies that provide evidence about the intervals for screening for peripheral arterial disease (PAD) with the ABI in asymptomatic clients.

ii. Evidence shows that low-dose aspirin treatment in asymptomatic clients with low ABI does not improve health outcomes and may increase bleeding.

iii. There are no trials that provide evidence on other interventions to reduce CVD events or interventions that might delay the onset of lower extremity symptoms.

e. The USPSTF recommends against screening for asymptomatic carotid artery stenosis in the general adult population.

f. The evidence is insufficient to assess the balance of benefits and harms of using nontraditional risk factors to screen asymptomatic males and females with no history of CHD to prevent CHD events. The nontraditional risk factors included in this recommendation include the following:

i. High-sensitivity C-reactive protein (hs-CRP).
ii. ABI.
iii. Leukocyte count.
iv. Fasting blood glucose level.
v. Periodontal disease.
vi. Carotid intima media thickness (CIMT).
vii. Coronary artery calcification (CAC) score on electron-beam CT (EBCT).
viii. Homocysteine level.
ix. Lipoprotein(a) level.

2. The American College of Cardiology (ACC) notes the CAC, a high-tech screen for CVD, is considered a second-tier assessment technique. Cost remains one concern, as well as the radiation risk. It is a good predictor of future cardiac disease events, which includes stroke. The calcium burden measured by CAC probably equals about one-fifth of the total atherosclerosis burden. The ACC/American Heart Association (AHA) guideline on the assessment of cardiovascular risk states that "[i]f, after quantitative risk assessment, a risk-based treatment decision is uncertain, assessment of ≥1 of the following—family history, hs-CRP, CAC score, or ABI—may be considered to inform treatment decision making."

3. Prevention recommendations with aspirin:

a. The magnitude of the CV risk reduction with aspirin use depends on an individual's initial risk for CVD events. Risk assessment for CVD should include evaluation of the following risk factors: age, sex, race/ethnicity, total cholesterol level, high-density lipoprotein (HDL) cholesterol level, systolic BP, HTN treatment, diabetes, and smoking. An online version of the ACC/AHA risk calculator can be found at too ls.acc.org/ascvd-risk-estimator-plus/#!/calculate/estimate/.

b. The recommendations for use of aspirin in the primary prevention of CVD (which are currently being updated [open for public recommendations until November 2021]) and colorectal cancer (CRC) are noted in Table 2.2.

i. The decision to initiate low-dose aspirin use for the primary prevention of CVD in adults 40 to 59 years of age who have a 10% or greater 10-year CVD risk should be an individual one. Clients who are not at risk for bleeding may benefit the most from aspirin use.

ii. The USPSTF recommends against initiating low-dose aspirin use for the primary prevention of CVD in adults age 60 years or older. Aspirin use may also result in small to moderate harms, including gastrointestinal (GI) bleeding and hemorrhagic stroke.

iii. The USPSTF concludes with moderate certainty that the benefit of aspirin use for the primary prevention of CVD events, combined with the reduced incidence of CRC, outweighs the increased risk of bleeding by a moderate amount in adults age 50 to 59 years who have a 10-year CVD risk of 10% or greater.

iv. The USPSTF concludes with moderate certainty that the benefit of aspirin use for the primary prevention of CVD events, combined with the reduced incidence of CRC, outweighs the increased risk of bleeding by a small amount in adults age 60 to 69 years who have a 10-year CVD risk of 10% or greater.

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TABLE 2.2 PROPOSED USPSTF ASPIRIN USE TO PREVENT CARDIOVASCULAR DISEASE AND COLORECTAL CANCER: PREVENTIVE MEDICATION FOR ADULTS (AS OF OCTOBER 2021)

Age Range	Recommendation
Adults ages 40 to 59 years with a 10% or greater 10-year CVD risk	The decision to initiate low-dose aspirin use for the primary prevention of CVD in adults ages 40 to 59 years who have a 10% or greater 10-year CVD risk should be an individual one. Evidence indicates that the net benefit of aspirin use in this group is small. Persons who are not at increased risk for bleeding and are willing to take low-dose aspirin daily are more likely to benefit.
Adults age 60 years or older	The USPSTF recommends against initiating low-dose aspirin use for the primary prevention of CVD in adults age 60 years or older.

Notes: This recommendation applies to adults who are at increased CVD risk and at average risk for CRC. Persons who are at increased CVD risk and are known to be at increased risk for CRC (e.g., persons with a family or personal history of CRC or familial adenomatous polyposis) should consult their healthcare provider.

CRC, colorectal cancer; CVD, cardiovascular disease; USPSTF, U.S. Preventive Services Task Force.

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COLORECTAL SCREENING

A. Refer to the "Colorectal Cancer Screening" section of Chapter 14 and further review of other gastrointestinal (GI) conditions.

B. The American Cancer Society (ACS) released their 2018 updated guidelines for colorectal cancer (CRC) screening for adults with average risk. New data indicate an increasing incidence of CRC among individuals younger than age 50 years. The incidence has increased by 51% since 1994.

1. Regular CRC screening with either a high-sensitivity stool-based test or a structural (visual) examination should begin at age 45 years among adults with average risk for CRC.

2. Adults aged 50 years and older should be screened regularly for CRC.

3. Among adults with average CRC risk who are in good health and have a life expectancy of more than 10 years, CRC should be continued through age 75 years.

4. CRC screening decisions should be individualized for clients age 76 through 85 years according to client preferences, life expectancy, health status, and history of screening.

5. Continuing CRC screening is discouraged for individuals older than 85 years.

C. The following are the U.S. Preventive Services Task Force (USPSTF) recommendations for screening for CRC by age:

1. Adults age 45 to 49: The USPSTF recommends screening for CRC in adults aged 45 to 49 years.

2. Adults age 50 to 75 years:

A. The USPSTF recommends screening for CRC starting at age 50 years and continuing until age 75 years.

B. The risks and benefits of different screening methods vary.

3. Adults age 76 to 85 years:

A. The decision to screen for CRC in adults age 76 to 85 years should be an individual one, taking into account the client's overall health and screening history.

B. Adults in this age group who have never been screened for CRC are more likely to benefit.

C. Screening would be most appropriate among adults who are healthy enough to undergo treatment if CRC is detected and who do not have comorbid conditions that would significantly limit their life expectancy.

D. CRC prevention plays an important role in the overall health benefit of aspirin, but this benefit is not apparent until 10 years after aspirin therapy is started. Clients need to take aspirin for at least 5 to 10 years to realize this potential benefit, and persons with shorter life expectancy are less likely to benefit. Thus, aspirin use is more likely to have an effect when it is started and used between the ages of 50 and 59 years, with those of higher cardiovascular disease (CVD) risk. The majority of CRCs occur in people older than 50. The USPSTF guidelines recommend the use of aspirin only for adults age 50 to 59. The American College of Gastroenterology and the USPSTF agree that clients at an increased risk of CVD often have other issues associated with increased inflammation, such as diabetes, obesity, or hypertension. Those other issues increase the risk of CRC. See Table 2.2 for the USPSTF recommendation for aspirin use to prevent CVD and CRC.

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DENTAL CARE

A. Research has linked infections from poor oral care from untreated cavities and gum disease to preterm birth, heart disease, diabetes, and respiratory diseases. Primary care providers are on the front line for performing oral examinations. Alcohol, drug use, smoking cigarettes, using pipes, and smokeless tobacco have been identified as among the major causes of poor oral health and partly responsible for the increase in risk of oral cancer.

B. Medicare does not cover routine dental care or dental procedures such as cleanings, fillings, tooth extractions, dentures, or other dental devices. Medicare Part A (hospital insurance) can pay for emergency or complicated dental procedures, even though dental care is not covered. Medicaid coverage for dental care varies by state. Medicare Advantage plans now cover some dental care. The Affordable Care Act (ACA) expansion of medical insurance includes coverage for dental services in children.

C. Primary care providers should provide education on good oral hygiene, including brushing, flossing, seeing a dentist at least yearly, and eating a healthy diet. The U.S. Preventive Services Task Force (USPSTF) has, however, found inadequate evidence that oral screening examinations accurately detect oral cancer or that treatment of screen-detected oral cancer improves morbidity or mortality.

D. Many dental schools provide care at reduced costs based on an individual's income. The U.S. Department of Health and Human Services Health Resources and Services Administration (HRSA) has health centers in both cities and rural areas that provide care based on income. The HRSA maintains a website to find the nearest The HRSA-funded health centers at findahealthcenter.hrsa.gov/tool.

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DEPRESSION SCREENING

A. Screen for depression, regardless of risk factors, in the general adult population, age 18 years and older, including pregnant and postpartum adults. Among older adults, risk factors for depression include disability and poor health status related to medical illness, complicated grief, chronic sleep disturbance, loneliness, and history of depression. However, the presence or absence of risk factors alone cannot distinguish clients with depression from those without depression.

B. Screening is recommended by the U.S. Preventive Services Task Force (USPSTF) when staff-assisted depression care supports are in place to ensure accurate diagnosis, appropriate treatment, and appropriate follow-up.

C. There is little evidence regarding the optimal timing for screening. The optimum interval for screening for depression is also unknown. Screening all adults who have not been screened previously and using clinical judgment in consideration of risk factors, comorbid conditions, and life events are used to determine if additional screening of high-risk clients is warranted.

D. There are multiple depression screening tools:

1. The Patient Health Questionnaire (PHQ-9) is the most common screening tool used to identify depression. The tool is located at www.drugsandalcohol.ie/26814/1/Patient%20Health%20Questionnaire.pdf.

2. The Hamilton Rating Scale for Depression (HAM-D) should be administered by a healthcare professional. The tool is located at www.mdcalc.com/hamilton-depression-rating-scale-ham-d.

3. The Cornell Scale for Depression in Dementia (CSDD) is located at www.windstonehealth.com/wp-content/uploads/2015/05/Cornell.pdf.

4. The Geriatric Depression Scale (GDS) is located at www.grepmed.com/images/264/depression-geriatrics-diagnosis-geriatric-scale. The GDS (short form) is located at geriatrictoolkit.missouri.edu/cog/GDS_SHORT_FORM.PDF.

5. The Edinburgh Postnatal Depression Scale (EPDS) is located at www.fresno.ucsf.edu/pediatrics/downloads/edinburghscale.pdf.

E. All positive screening results should lead to additional assessment that considers the severity of depression and comorbid psychological problems (e.g., anxiety, panic attacks, or substance abuse), alternate diagnoses, and medical conditions.

F. Medicare Part B covers an annual depression screening for Medicare beneficiaries in primary care settings when staff-assisted depression care supports are in place to ensure accurate diagnosis, effective treatment, and follow-up, thereby facilitating and coordinating referrals to mental health treatment. Medicare does not identify a specific depression screening tool.

G. The March 2016 Institute for Clinical System Improvement (ISCI) Adult Depression in Primary Care Guideline provides practitioners with current information related to screening for depression, including an algorithm for adult depression in the primary care setting and the criteria for a major depressive episode from the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (*DSM-5;* American Psychiatric Association, 2013).

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DIABETES SCREENING

A. The incidence of type 2 diabetes mellitus (DM) is one of the major health problems facing the United States today and will continue to be an issue as the population ages and becomes more obese. The tests to be used for the diagnosis of diabetes are also utilized for screening for DM: fasting plasma glucose, the 2-hour plasma glucose after a 75-g glucose tolerance test, and the hemoglobin A1C tests (see the "Diabetes Mellitus" section of Chapter 24).

B. The U.S. Preventive Services Task Force (USPSTF) recommends screening in the primary care setting for abnormal blood glucose as part of cardiovascular risk assessment in adults 35 to 70 years of age who are overweight or obese and do not have obvious symptoms of diabetes. Clinicians should offer or refer clients with abnormal blood glucose to intensive behavioral counseling interventions to promote a healthful diet and physical activity. Their recommendation further states that persons who have a family history of diabetes or a history of gestational diabetes or polycystic ovarian syndrome (POS) or are members of certain racial/ethnic groups (e.g., African Americans, American Indians or Alaskan Natives, Asian Americans, Hispanics or Latinos, or Native Hawaiians or Pacific Islanders) may be at increased risk for diabetes at a younger age or at a lower body mass index (BMI). Clinicians should consider screening earlier in persons with one or more of these characteristics.

C. The following are the American Diabetes Association (ADA) screening recommendations:

1. Screening for prediabetes and type 2 DM with an informal assessment of risk factors or validated tools should be considered in asymptomatic adults.

2. Client screening should begin at age 45 years for all people. Consider testing for prediabetes in asymptomatic adults of any age with a BMI of ≥25 kg/m^2 or 23 kg/m^2 (in Asian Americans) who have one or more additional risk factors for diabetes:

a. A1C ≥5.7, impaired glucose tolerance (IGT), or impaired fasting glucose (IFG) on previous testing.
b. First-degree relative with diabetes.
c. High-risk race/ethnicity (e.g., African American, Latino, Native American, Asian American, and Pacific Islander).
d. Females who were diagnosed with gestational DM (GDM).
e. History of cardiovascular disease (CVD).

▶

f. Hypertension (>140/90 mmHg) or receiving therapy for hypertension.
g. High-density lipoprotein (HDL) <35 mg/dL and/or a triglyceride level >250 mg/dL.
h. Females with POS.
i. Physical inactivity.
j. Other clinical conditions associated with insulin resistance (e.g., severe obesity, acanthosis nigricans).

D. If the test results are normal, the ADA retesting for prediabetes and for type 2 diabetes at least at 3-year intervals is reasonable.

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HEPATITIS C SCREENING

A. The U.S. Preventive Services Task Force (USPSTF) recommends offering a onetime screening for hepatitis C virus (HCV) infections to asymptomatic adults born between 1945 and 1965 without known liver disease or functional abnormalities. However, the guidelines were augmented in 2020 to include the following: (a) hepatitis C screening at least once in a lifetime for all adults aged ≥18 years, except in settings where the prevalence of HCV infection is <0.1%; and (b) hepatitis C screening for all pregnant females during each pregnancy, except in settings where the prevalence of HCV infection is <0.1%.

B. The USPSTF also recommends screening for HCV infection in persons at high risk for infection:

1. The most important risk factor for HCV infection is past or current injection drug use. Intranasal drug use is also an additional risk factor.
2. Receipt of a blood transfusion before 1992.
3. Long-term hemodialysis and hemophilia.
4. Percutaneous exposures, including unregulated tattoo.
5. High-risk sexual behaviors (multiple sex partners, unprotected sex, sex with an HCV-infected person or injection drug user, and men having sex with men [MSM]).
6. Birth to an HCV-infected mother.
7. Incarceration.

C. For those with reactive test results, the anti-HCV test should be followed with an additional, supplemental, or confirmatory test for the presence of the virus (see the "Hepatitis C" section of Chapter 14).

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HIV SCREENING

A. HIV testing is a covered benefit from the Centers for Medicare and Medicaid Services (CMS) as a voluntary test every 12 months for people at increased risk and anyone asking for HIV testing. CMS also covers testing for HIV in pregnancy up to three times. The current U.S. Preventive Services Task Force (USPSTF) recommendation is that screening for HIV infection could begin at age 15 to 65 years. Teens younger than age 15 and adults older than 65 also should be screened if they are at risk for HIV infection (see the "Human Immunodeficiency Virus" section of Chapter 20). The USPSTF also recommends that all pregnant females, including those in labor who do not know their HIV status, be screened for HIV infection. High-risk factors for HIV are risky sexual practices (men having sex with men [MSM], sexual partners with HIV, bisexual, exchanging sex for drugs or money) and infected intravenous (IV) drug users. For persons over age 65, new sexual partners are at an increased risk.

B. Testing intervals are not defined by the CMS or by the USPSTF.

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IMMUNIZATIONS

A. The Centers for Disease Control and Prevention (CDC) recommendations for immunizations are presented in Figure 2.1.

B. Immunizations for travel outside of the United States are recommended based on the client's health status and vaccine history, as well as the area(s) of travel. The presence of the risk of becoming ill from poor sanitary conditions, food and water sources, and endemic diseases is considered in the CDC's recommendations for travel immunizations. The CDC has an interactive tool for immunization recommendations for travel located at wwwnc.cdc.gov/travel/destinations/list.

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Recommended Adult Immunization Schedule
for ages 19 years or older

UNITED STATES
2022

How to use the adult immunization schedule

1. Determine recommended vaccinations by age **(Table 1)**
2. Assess need for additional recommended vaccinations by medical condition or other indication **(Table 2)**
3. Review vaccine types, frequencies, intervals, and considerations for special situations **(Notes)**
4. Review contraindications and precautions for vaccine types **(Appendix)**

Vaccines in the Adult Immunization Schedule*

Vaccine	Abbreviation(s)	Trade name(s)
Haemophilus influenzae type b vaccine	Hib	ActHIB® Hiberix® PedvaxHIB®
Hepatitis A vaccine	HepA	Havrix® Vaqta®
Hepatitis A and hepatitis B vaccine	HepA-HepB	Twinrix®
Hepatitis B vaccine	HepB	Engerix-B® Recombivax HB® Heplisav-B®
Human papillomavirus vaccine	HPV	Gardasil 9®
Influenza vaccine (inactivated)	IIV4	Many brands
Influenza vaccine (live, attenuated)	LAIV4	FluMist® Quadrivalent
Influenza vaccine (recombinant)	RIV4	Flublok® Quadrivalent
Measles, mumps, and rubella vaccine	MMR	M-M-R II®
Meningococcal serogroups A, C, W, Y vaccine	MenACWY-D MenACWY-CRM MenACWY-TT	Menactra® Menveo® MenQuadfi®
Meningococcal serogroup B vaccine	MenB-4C MenB-FHbp	Bexsero® Trumenba®
Pneumococcal 15-valent conjugate vaccine	PCV15	Vaxneuvance™
Pneumococcal 20-valent conjugate vaccine	PCV20	Prevnar 20™
Pneumococcal 23-valent polysaccharide vaccine	PPSV23	Pneumovax 23®
Tetanus and diphtheria toxoids	Td	Tenivac® Tdvax™
Tetanus and diphtheria toxoids and acellular pertussis vaccine	Tdap	Adacel® Boostrix®
Varicella vaccine	VAR	Varivax®
Zoster vaccine, recombinant	RZV	Shingrix

*Administer recommended vaccines if vaccination history is incomplete or unknown. Do not restart or add doses to vaccine series if there are extended intervals between doses. The use of trade names is for identification purposes only and does not imply endorsement by the ACIP or CDC.

Recommended by the Advisory Committee on Immunization Practices (www.cdc.gov/vaccines/acip) and approved by the Centers for Disease Control and Prevention (www.cdc.gov), American College of Physicians (www.acponline.org), American Academy of Family Physicians (www.aafp.org), American College of Obstetricians and Gynecologists (www.acog.org), American College of Nurse-Midwives (www.midwife.org), and American Academy of Physician Associates (www.aapa.org), and Society for Healthcare Epidemiology of America (www.shea-online.org).

Report

- Suspected cases of reportable vaccine-preventable diseases or outbreaks to the local or state health department
- Clinically significant postvaccination reactions to the Vaccine Adverse Event Reporting System at www.vaers.hhs.gov or 800-822-7967

Injury claims

All vaccines included in the adult immunization schedule except pneumococcal 23-valent polysaccharide (PPSV23) and zoster (RZV) vaccines are covered by the Vaccine Injury Compensation Program. Information on how to file a vaccine injury claim is available at www.hrsa.gov/vaccinecompensation.

Questions or comments

Contact www.cdc.gov/cdc-info or 800-CDC-INFO (800-232-4636), in English or Spanish, 8 a.m.–8 p.m. ET, Monday through Friday, excluding holidays.

Download the CDC Vaccine Schedules app for providers at www.cdc.gov/vaccines/schedules/hcp/schedule-app.html.

Helpful information

- Complete Advisory Committee on Immunization Practices (ACIP) recommendations: www.cdc.gov/vaccines/hcp/acip-recs/index.html
- *General Best Practice Guidelines for Immunization* (including contraindications and precautions): www.cdc.gov/vaccines/hcp/acip-recs/general-recs/index.html
- Vaccine information statements: www.cdc.gov/vaccines/hcp/vis/index.html
- Manual for the Surveillance of Vaccine-Preventable Diseases (including case identification and outbreak response): www.cdc.gov/vaccines/pubs/surv-manual
- Travel vaccine recommendations: www.cdc.gov/travel
- Recommended Child and Adolescent Immunization Schedule, United States, 2022: www.cdc.gov/vaccines/schedules/hcp/child-adolescent.html
- ACIP Shared Clinical Decision-Making Recommendations: www.cdc.gov/vaccines/acip/acip-scdm-faqs.html

U.S. Department of Health and Human Services
Centers for Disease Control and Prevention

Scan QR code for access to online schedule

CS310021-A

FIGURE 2.1 Recommended immunization schedule for adults aged 19 years or older (2022).

Source: From Centers for Disease Control and Prevention. (2021). Recommended immunization schedule for adults aged 19 years or older, United States, 2021. https://www.cdc.gov/vaccines/schedules/downloads/adult/adult-combined-schedule.pdf.

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MAMMOGRAPHY

A. Screening mammography guidelines:

1. The 2016 U.S. Preventive Services Task Force (USPSTF) screening mammography recommendations apply to asymptomatic females age 40 years or older who do not have preexisting breast cancer or a previously diagnosed high-risk breast lesion and who are not at high risk for breast cancer due to a known underlying genetic mutation (such as a *BRCA1* or *BRCA2* gene mutation or other familial breast cancer syndrome) or a history of chest radiation at a young age.

a. The USPSTF concludes that the current evidence is insufficient to assess the benefits and harms of digital breast tomosynthesis (DBT) as a primary screening method for breast cancer.

b. In addition to false-positive results and unnecessary biopsies, all females undergoing regular screening mammography are at risk for diagnosis and treatment of noninvasive and invasive breast cancer that would otherwise not have become a threat to their health, or even apparent, during their lifetime (known as "overdiagnosis"). Beginning mammography screening at a younger age and screening more frequently may increase the risk of overdiagnosis and subsequent overtreatment.

i. Females age 40 to 49 years: The decision to start screening mammography in females prior to age 50 years should be an individual one. Females who place a higher value on the potential benefit than the potential harms may choose to begin biennial (every other year) screening between the ages of 40 and 49 years. Females with a parent, sibling, or child with breast cancer are at higher risk for breast cancer and thus may benefit more than average-risk females from beginning screening in their 40s.

ii. Females age 50 to 74 years: Biennial screening mammography is recommended.

iii. Females age 75 years or older: The current evidence is insufficient to assess the balance of benefits and harms of screening mammography in females age 75 years or older.

2. The American College of Obstetricians and Gynecologists (ACOG) 2017 recommendations for screening mammography include the following:

a. Ages 40 to 49: Offer screening mammography in the context of a shared, informed decision-making approach that recognizes the uncertainty of additional benefits and harms of clinical breast examination beyond screening mammography.

b. Ages 40 to 49: Initiate after counseling, if client desires.

c. Ages 50 to 74: Recommend screening by no later than age 50, annual or biennial.

d. Continue until 75 years. Beyond age 75, the decision to discontinue should be based on client autonomy and shared decision-making process that includes a discussion of the client's health status and longevity.

3. The 2021 American Cancer Society (ACS) mammography guideline recommendations are for clients at average risk for breast cancer. Females at high risk, due to family history, a breast condition, or other reasons, need to begin screening earlier and more often.

a. Females should have the opportunity to begin annual screening between the ages of 40 and 44 years yearly.

b. Females with an average risk of breast cancer should undergo regular screening mammograms yearly at ages 45 to 54. ▶

TABLE 2.3 CERVICAL CANCER SCREENING GUIDELINES FOR *AVERAGE-RISK* FEMALES

	Recommending Body		
	ASCCP, SGO, and ASCP 2021 Guidelines	**USPSTF 2021 Guidelines**	**ACOG 2021**
Statement about annual screening	Females of any age should not be screened annually by any screening method.	Cervical cancer screening visits are an opportunity to discuss other health problems and preventive measures.	Clients should be counseled that annual well-woman visits are recommended even if bimanual pelvic exam with cervical cancer screening is not performed at each visit. The components of the examination may vary depending on the client's age, risk factors, and physician preference. The visit is valuable in promoting prevention practices, recognizing risk factors for disease, identifying medical problems, administration of immunizations based on age and risk factors, and establishing a clinician–client relationship.
Females aged <21 years	No screening is recommended, regardless of age at sexual initiation or other risk factors.	No screening is recommended for females <21.	No screening is recommended for females <21, regardless of age at sexual initiation and other behavior-related risk factors.

(*continued*)

TABLE 2.3 CERVICAL CANCER SCREENING GUIDELINES FOR *AVERAGE-RISK* FEMALES (*CONTINUED*)

	Recommending Body		
	ASCCP, SGO, and ASCP 2021 Guidelines	**USPSTF 2021 Guidelines**	**ACOG 2021**
Females aged 21–29 years • Cytology (conventional or liquid based) alone	Cervical cytology every 3 years is recommended. HPV cotesting should not be used in females <30 years.	Cervical cytology alone **every 3 years** is recommended.	Cervical cytology **every 3 years** is recommended. Liquid-based and conventional methods of cervical cytology collection are acceptable for screening. HPV cotesting should not be used in females <30 years.
Females aged 30–65 years • Cytology (conventional or liquid based) alone OR • hrHPV cotest (cytology and HPV test administered together)	hrHPV cotesting is recommended **every 5 years**.	Cervical cytology alone is recommended **every 3 years.** OR alternatively hrHPV testing alone is recommended **every 5 years**.	Cervical cytology test alone **every 3 years** is acceptable. **OR alternatively Pap test + hrHPV cotesting every 5 years is preferred over cytology alone every 3 years.** **The FDA approved primary hrHPV testing alone every 5 years.**
When to stop screening for average-risk females	Females >65 years with adequate screening history do not need further screening.	For females >65 years, no screening is recommended for those who have had adequate prior screening and are not otherwise at high risk for cervical cancer.	• For females >65 years, no screening is recommended for those who have had adequate prior screening and are not otherwise at high risk for cervical cancer; and if no history of cervical changes **AND** either three negative Pap test results in a row or two negative cotest results in a row within the past 10 years, with the most recent test performed within the past 5 years.
Primary hrHPV testing alone	2021 ASCCP and SGO issued agreement with ACOG guidelines for use of HPV test for primary testing.	For average-risk females aged 30–65, hrHPV testing **every 5 years** is an alternative to cytology alone **every 3 years.**	Primary hrHPV testing is recommended every 5 years for females aged 30–65.
Screening posthysterectomy	Females who have had a total hysterectomy (removal of the uterus and cervix) should stop screening. Females who have had a supracervical hysterectomy (cervix intact) should continue screening according to guidelines.	No screening is recommended for females who have had a hysterectomy with removal of the cervix and do not have a history of high-grade precancerous lesions (e.g., cervical intraepithelial neoplasia grade 2 or 3) or cervical cancer.	• For females who have had a total hysterectomy with removal of the uterus and cervix and no history of cervical cancer or cervical change, no screening with routine cytology screening, and HPV testing should be discontinued and not restarted for any reason. • For females who had a hysterectomy in which the cervix was removed **AND** have a history of cervical cancer or moderate to severe cervical changes, the recommendation is to continue screening for 20 years after surgery.
Screening among those immunized against HPV 16 and 18	Females of any age with a history of HPV vaccination should be screened according to the age-specific recommendation for the general population.	The possibility that vaccination might reduce the need for screening with cytology or hrHPV testing has not been established; therefore, females who have been vaccinated should continue to be screened as recommended until further evidence accrues.	Females who have been vaccinated against HPV still need to be screened according to the same age-specific guidelines.

ACOG, American College of Obstetricians and Gynecologists; ASCCP, American Society for Colposcopy and Cervical Pathology; ASCP, American Society for Clinical Pathology; FDA, Food and Drug Administration; HPV, human papillomavirus; hrHPV, high-risk HPV testing; SGO, Society of Gynecologic Oncology; USPSTF, U.S. Preventive Services Task Force.

c. Females 55 years and older should transition to biennial screening or have the opportunity to continue screening annually.

d. Females should continue screening mammograms as long as their overall health is good and they have a life expectancy of 10 years or longer.

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PAP SMEAR/PELVIC EXAMINATION

A. Human papillomavirus (HPV) increases the risk of cancer, including cervical, vulvar, vaginal, penile, and anal cancer, as well as cancer of the oropharynx. Since cervical cancer is believed to be caused by sexually transmissible HPV infections, clients who have not had sexual exposures are likely at low risk. Females older than 21 years who have not engaged in sexual intercourse may not need a Pap test depending on circumstances. The decision should be made at the discretion of the client and their physician. Females who have had sex with females are still at risk of cervical cancer.

B. Persistent infection of the uterine cervix with high-risk types of human papillomavirus (hrHPV) is required for the development of invasive cervical cancer. Infection with hrHPV is common, especially with sexually active young females; most infections are transient and spontaneously clear without clear clinical consequences. The American College of Obstetricians and Gynecologists (ACOG) joins the American Society for Colposcopy and Cervical Pathology (ASCCP) and the Society of Gynecologic Oncology (SGO) in endorsing the U.S. Preventive Services Task Force (USPSTF) 2021 cervical cancer screening clinical guidelines, recommending screening strategies with cytology or cotesting (cytology in combination with hrHPV) based on age. The guidelines recommend screening of females 21 to 65 years of age with cytology alone or cotesting with hrHPV based on age-specific guidelines. The American Cancer Society (ACS) recommends cervical cancer screening with an HPV test alone every 5 years for everyone with a cervix from age 25 until age 65. If HPV testing alone is not available, people can get screened with an HPV/Pap cotest every 5 years or a Pap test every 3 years. See Table 2.3 for cervical cancer screening recommendations for average-risk clients. Females who are infected with HIV or are otherwise immunocompromised (e.g., solid organ transplants), females previously treated for cervical intraepithelial neoplasia 2 (CIN2) or CIN3 or cancer, and females exposed to diethylstilbestrol (DES) before birth need more frequent screening.

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PROSTATE CANCER SCREENING

A. Prostate cancer is one of the most common cancers in males. It is a slow-growing cancer with a lifetime risk of 11%, and the lifetime risk of dying from prostate cancer is 2.5%. The median risk of death from prostate cancer is 80 years. The risk factors for prostate cancer include:

1. Age: males older than age 50.

2. Race/ethnicity: African American (4.2%), Hispanic (2.9%), Caucasian (2.3%), and Asian and Pacific Islander males (2.1%).

3. Males who have a family history of prostate cancer.

B. Smoking is associated with a high risk of prostate cancer mortality. The U.S. Preventive Services Task Force (USPSTF) 2018 recommendations for prostate-specific antigen (PSA) screening states the following:

1. The use of digital rectal examination as a screening modality is not recommended.

2. Males age 55 to 69 years:

a. The decision to undergo periodic PSA-based screening for prostate cancer should be an individual one. Before being screened, clients should have an opportunity to discuss the potential benefits and harms with their clinician and to incorporate their values and preference in the decision.

b. In determining whether this service is appropriate in individual cases, clients and clinicians should consider the balance of benefits and harms on the basis of family history, race/ethnicity, comorbid conditions, client values, and other health needs.

c. Clinicians should not screen clients who do not express a preference for screening.

3. Males 70 years and older: The USPSTF recommends against PSA-based screening for prostate cancer, noting adequate evidence from randomized clinical trials (RCT) is consistent with no benefit of PSA-based screening for prostate cancer due to a lack of evidence on the benefits. ▶

4. Harms of screening include frequent false-positive results with subsequent overdiagnoses, which can result in overtreatment, including invasive procedures, chemotherapy, and radiation.

C. Screening for prostate cancer with PSA has been controversial because of the potential for overdiagnosis and overtreatment. The PSA may be elevated with prostate problems other than cancer, such as enlarged prostate, prostatitis, urinary tract infection, or recent ejaculation. Further testing for suspected prostate cancer includes a digital rectal examination (not a current recommendation), transrectal ultrasound, and/or a needle biopsy.

D. Active surveillance with continued observation and physical examinations has been the more common treatment choice over the past several years among males with low-risk prostate cancer and may reduce the chance of overtreatment. Active surveillance includes periodic PSA tests, physical examination, and repeated prostate biopsy, an often repeated rectal examination. Treatment includes prostatectomy surgery, radiation, cryotherapy, chemotherapy, immunotherapy, or androgen deprivation therapies. Side effects of these procedures include urinary incontinence, erectile dysfunction, and bowel dysfunction. The use of androgen deprivation therapy for localized prostate cancer is associated with erectile dysfunction, gynecomastia, and hot flashes.

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SEXUALLY TRANSMITTED INFECTION SCREENING

A. Taking a thorough sexual history is the basis of identifying high-risk sexual behavior, screening, counseling, and treating sexually transmitted infections (STIs). Older adults should continue to be screened throughout their lifetime for STIs; there is no cutoff on the age of transmission of STIs. All persons who seek evaluation and treatment for STIs should be screened for HIV. Screening should be routine, regardless of whether the client reports any specific behavioral risk for HIV. The Centers for Disease Control and Prevention (CDC) recommends that persons 13 to 64 years of age be screened for HIV in all healthcare settings. Persons should be notified that testing will be performed, but retain the option to decline or defer testing. Guidelines and recommendations for HIV/AIDS for clinicians are available at the CDC website located at www.cdc.gov/hiv/guidelines/index.html. For more discussion on sexual health and treatment guidelines for STIs and HIV see the "Sexual Health Issues in the Aging Population" of Chapter 15; the full CDC STI guidelines in Chapter 18; and the "Human Immunodeficiency Virus" section of Chapter 20.

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VISION SCREENS

A. Age-related changes affect the sense of sight and require screening for refractory errors, macular degeneration, glaucoma, and cataracts (see Chapter 8, "Eye Guidelines").

B. Multiple medical and systemic conditions including genetic, cardiovascular, endocrine, gastrointestinal (GI), nutritional, and metabolic disorders increase the need for a comprehensive dilated eye examination. The American Optometric Association recommends an examination every 2 years for risk-free adults between 18 and 60 years. For those at risk between 18 and 60 years of age, eye examinations are recommended every 1 to 2 years. For adults who are 61 years of age and older who are risk-free, an annual eye examination is recommended. A comprehensive eye examination for at-risk adults should occur annually or as recommended. The American Academy of Ophthalmology recommends a baseline comprehensive exam at age 40. This recommendation is for all adults with no signs or risk factors for eye disease. Based on the results of the initial screening, an ophthalmologist will prescribe the necessary intervals for follow-up exams.

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PRETRAVEL HEALTH SCREENINGS

A. Pretravel consultation offers a dedicated time to prepare travelers for the health concerns that might arise during their trips. The objective is to perform an individual risk assessment, educate the traveler regarding anticipated health risks and methods for prevention, and provide immunizations for vaccine-preventable diseases and medications for prophylaxis, self-treatment, or both. Travel medicine specialists have in-depth knowledge of immunizations, risks associated with specific destinations, and the implications of traveling with underlying conditions. Therefore, a comprehensive consultation with a travel medicine expert is indicated for any traveler with a complicated health history, special risks (such as traveling at high altitudes or working in refugee camps), or exotic or complicated itineraries.

B. Effective pretravel consultations require attention to the health background of the traveler and incorporation of the itinerary, trip duration, travel purpose, and activities, all of which determine health risks.

1. Risk assessment:
   a. Medical history including underlying conditions, allergies (especially any pertaining to vaccines, egg, or latex), and medications. ▶

b. Special conditions including pregnancy, breastfeeding, disabilities, immunocompromising conditions or medications, seizure disorder, recent myocardial infarction (MI), surgery, and history of Guillain–Barré syndrome.
c. Immunization history: routine and travel vaccines.
d. Prior travel experience including malaria chemoprophylaxis, experience with altitude, and illness related to prior travel.

2. Trip details:
a. Itinerary: countries and specific regions, including the travel order of countries, as well as rural or urban travel.
b. Timing: trip duration, season of travel, time to depart.
c. Reason for travel: tourism, business, visiting friends/relatives, missionary/aid work, adoption, or seeking healthcare (medical tourism).
d. Travel style: independent travel or package tour, traveler risk tolerance, general hygiene standards at the destination, modes of transport, accommodations, and propensity for "adventurous" eating.
e. Special activities: disaster relief, medical care (providing or receiving), high altitude, diving, cruise ship, rafting or other water exposure, cycling, extreme sports, spelunking, and anticipated interactions with animals domestic and wild.

C. Assess individual risks: Many elements merit consideration in assessing a traveler's health risks (see Table 2.1). Certain travelers may confront special risks. Recent hospitalization for serious problems may lead the advisor to recommend delaying travel. Air travel is contraindicated for certain conditions, such as less than 3 weeks after an uncomplicated MI and less than 10 days after thoracic or abdominal surgery. The travel health provider and the traveler should consult with the relevant healthcare providers most familiar with the underlying illnesses.

D. Manage risk.

1. Immunizations are a crucial component of pretravel consultations, and the risk assessment forms the basis of recommendations for travel vaccines.

2. For travelers going to malaria-endemic countries, it is imperative to discuss malaria transmission, ways to reduce risk, and recommendation for chemoprophylaxis and continuation protocol of the regimen post return.

3. Travelers with underlying health conditions require attention to their health issues as they relate to the destination and activities. Travelers should be counseled on how they can find reputable medical facilities at their destination, such as using the International Society of Travel Medicine (ISTM) website (www.istm.org) or the American Society of Tropical Medicine and Hygiene website (www.astmh.org) to find suitable clinics. Any allergies or serious medical conditions should be identified on a bracelet or a card to expedite medical care in emergency situations.

4. The pretravel consultation also provides another setting to remind travelers of basic health practices during travel, including frequent handwashing, COVID-19 prevention precautions including wearing face masks and sanitizing surfaces, as well as wearing seat belts, and using car seats for infants and children. General issues such as preventing injury and sunburn also deserve mention. Written information is essential to supplement oral advice and enable travelers to review the instructions from their clinic visits; educational material is available at the Centers for Disease Control and Prevention (CDC) Travelers Health web page (www.cdc.gov/travel). Advice on self-treatable conditions may minimize the need for travelers to seek medical care while abroad.

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CHAPTER 3

ADULT-GERIATRIC ASSESSMENTS

L. Douglas Smith Jr.

COMPREHENSIVE ASSESSMENT IN GERONTOLOGY

A. Adults 65 years and older will account for 20% of the total U.S. population within 10 years. Older individuals with increasingly complex chronic health conditions will account for an ever-increasing proportion of the adult and geriatric clinician's client population. While changes associated with the aging process *may* lead to progressive functional and cognitive decline, normal aging and the development of chronic disease are not synonymous. Still, the deterioration associated with aging makes it challenging to identify developing pathologies, leaves older adults vulnerable to disability and diminished quality of life, and makes it challenging to navigate the healthcare environment. The comprehensive geriatric assessment is a holistic approach to evaluating the aged person's status to develop an individualized plan of care. Personalization is imperative; the senior client may desire the care trajectory to prioritize aspects other than health outcomes and lengthened survival.

B. The evidence-based model for comprehensive geriatric assessment involves an interprofessional team performing a holistic review of the client's physical health, functional ability, psychological well-being, and socioenvironmental status. The geriatric assessment can be time- and labor-intensive. Strategies to reduce the burden of the evaluation include expanding the traditional disease-oriented visit to incorporate elements of the comprehensive assessment over multiple visits, electronic completion of screening forms before arrival, the delegation of tasks to other members of the interprofessional team, and client selection. Although parts of the assessment may be pertinent, the complete evaluation is inappropriate for clients with advanced dementia, terminal illness, and functional dependence. There is no particular evidence-prescribed instrument used; many validated tools exist. The assessment should be comprehensive while covering the four domains of physical health, functional ability, psychological well-being, and socioenvironmental status. The following includes only some of the available methods used for the assessment.

PHYSICAL HEALTH

A. The geriatric assessment does not replace the traditional physical examination; rather, it enhances the diagnostic process by elucidating information relevant to standard medical management, which clients may underreport. Therefore, clinicians must be attuned to the client's medical history and complaints while expanding the scope of the examination to search for conditions routine in older adults.

VISION AND HEARING

A. Vision and hearing impairments are common in older adults, although clients frequently fail to report these problems due to the association with normal aging. These problems may significantly impact the quality of life. Referral to a specialist (ophthalmologist or audiologist) may be appropriate, although the primary provider can initiate basic screening and workup. Assessment of visual acuity occurs with the Snellen eye chart or by asking the client questions such as "Are you having problems reading the newspapers or books?" or "Are you able to recognize faces from afar?" A hearing assessment using the whisper test or asking questions such as "Do you have problems hearing?" should occur. Family involvement with these screening questions may be invaluable as sensory loss may be less noticeable to the client. Positive screens indicate the need for further workup and intervention.

NUTRITION STATUS

A. Poor nutrition status in older adults is associated with increased health service use and mortality. Aged clients may be at increased risk for nutritional deficiency due to a host of physical and functional changes associated with normal aging. Poor nutrition may lead to unintentional weight loss or body mass index (BMI) below acceptable standards (BMI should be above 23 kg/m^2). Nutritional screening occurs using questions about weight loss or a validated instrument such as the Mini Nutritional Assessment (available at www.mna-elderly.com/sites/default/files/2021-10/mna-mini-english.pdf).

IMMUNIZATIONS

A. Aged clients are particularly at risk for preventable disease, and compliance with the recommended schedule from the Advisory Committee on Immunization Practices is encouraged. Clinicians should ensure age-appropriate immunization status for influenza, tetanus, diphtheria, pertussis, pneumococcus, and herpes zoster. Geriatric providers should monitor communications regarding ongoing immunization for coronavirus.

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POLYPHARMACY

A. There is no single definition of polypharmacy, although it is often defined as using five or more medications daily. Older persons are often prescribed multiple medications by different healthcare providers, increasing their risk of drug–drug interactions and adverse drug events.

1. The older adult population takes an average of 18 prescription drugs per year.

2. One in four people aged 65 years and older receive at least one of 20 potentially drugs inappropriate for older adult clients.

3. The amount and frequency of alcohol use should be included in medication reviews:

4. An important step in evaluating *potential* polypharmacy is the assessment of new symptoms. Typical signs and symptoms suggesting polypharmacy include confusion, gait disturbance, depression, delirium, dementia, anger, forgetfulness, falls, insomnia, drowsiness, and incontinence.

5. The strongest predictor of a potentially harmful medication is the number of drug prescriptions. Exposure to multiple drugs puts clients at additive risk of each single drug and increases the risk of geriatric syndromes and their sequela for falls, depression, and cognitive impairment. Refer to Appendix C for the American Geriatrics Society's (AGS) Beers criteria for potentially inappropriate medication use in older adults. The updated Beers criteria article is available at doi.org/10.1111/jgs.15767.

6. Prescribers with insufficient geriatric care knowledge may be a significant factor in polypharmacy when medications are not thoroughly evaluated for efficacy, safety, and interactions.

7. A potential source of therapeutic duplication may occur during a transition in care between providers, consultants, or discharge from hospitalization.

8. Evaluate issues related to the client's ability to remember medication names, the reason for prescription, how often to administer, and the ability to afford medications.

9. The best method of detecting potential problems with polypharmacy is to have clients bring in all of their medications, prescription and nonprescription, in their bottles. There are several approaches to the evaluation of polypharmacy issues:

a. Schedule a "brown bag" visit to review medications and the client's understanding (see Exhibits 3.1 and 3.2).

b. A senior care pharmacist may be available in your community for a referral. Senior care pharmacists are skilled healthcare professionals who have specialized training in the medication-related needs of seniors. Typically, the senior care pharmacist does a comprehensive consultation through a one-on-one review of all medications. ▶

EXHIBIT 3.1 Checklist for Brown Bag Medication Checkup

What to check for:

A. Indication for each medication.
B. Drug interactions between medications and food.
C. Adverse drug reactions (especially those to report or how to manage them).
D. Expiration dates of medications.
E. How the client is taking their medications.
F. Physical disabilities.
G. Monitoring tests.
H. How the medication is being stored.
I. Cultural, reading level, capability.
J. Allergies.

Make sure the client understands:

A. What each medication is used for.
B. How to take each medication (e.g., with or without food, number of times per day).
C. What to expect from the medication.
D. Side effects (especially those to report or how to manage them).

Take-home messages for the client:

A. Stress the importance of proper disposal of expired or discontinued medications.
B. Clarify questions on any remaining prescriptions.
C. Explain the importance of proper storage with certain medications (e.g., nitroglycerin).
D. Remind the client to communicate with their doctor or pharmacist about any side effects of prescription medications, OTCs, or herbal supplements.
E. Suggest medication schedules and the importance of compliance.
F. Reinforce lifestyle modifications to promote well-being.

OTC, over-the-counter.

Source: Permission to reprint from the Ohio Pharmacists Association. Ohio Patient Safety Institute: www.OPSI-Medication-Safety-gatefold-0319.pdf.

EXHIBIT 3.2 Medication Review Form—Brown Bag Program

Date: _____

Patient name: _____

Sex: M F

Telephone number:_____ Age: _____

Reported medicine allergies: _____

Please list ALL medications that you are currently taking (prescriptions, OTC medications, other):

_____

_____

_____

_____

_____

_____

_____

For Pharmacist Use

	Medicine #1	Medicine #2	Medicine #3	Medicine #4	Medicine #5
Drug name					
Dosage					
SIG on label					
MD name					
Expiration date					
Reported medical problem					
How long taken?					
Still taken?	Yes/No	Yes/No	Yes/No	Yes/No	Yes/No
Date of last MD visit					
Client knows purpose of drug	Yes/No	Yes/No	Yes/No	Yes/No	Yes/No
Compliance with SIG	Yes/No/Partial	Yes/No/Partial	Yes/No/Partial	Yes/No/Partial	Yes/No/Partial
Side effects?					
OTCs					
Improper administration	Yes/No	Yes/No	Yes/No	Yes/No	Yes/No
Overuse/underuse	Yes/No	Yes/No	Yes/No	Yes/No	Yes/No
Expired	Yes/No	Yes/No	Yes/No	Yes/No	Yes/No
Follow-up needed?	Yes/No	Yes/No	Yes/No	Yes/No	Yes/No

OTC, over-the-counter; SIG, prescription directions.

Source: Permission to reprint from the Ohio Pharmacists Association. Ohio Patient Safety Institute: www.OPSI-Medication-Safety-gatefold-0319.pdf.

c. The Institute for Safe Medication Practices (ISMP) has published the **List of Confused Drug Names,** which includes look-alike and sound-alike drugs. The *List of Confused Drug Names* is located at www.ismp.org/Tools/confuseddrugnames.pdf.

d. Client teaching: *See Client Teaching Guide for this chapter, "Safety Issues: Medication Safety."*

10. Suggestions for safeguards to reduce risk in errors when writing a prescription include the following:

a. Use both the brand and generic names on prescriptions and labels.

b. Include the purpose of the medication on prescriptions and labels.

c. The Food and Drug Administration (FDA) approves of and the ISMP recommends using "tall man," or mixed case, letters for writing prescriptions. Tall man lettering involves highlighting the dissimilar letters in two drug names to aid in distinguishing between the two drugs. See Table 3.1 for an example of using "tall man" letters for medications on the *List of Confused Drug Names*.

11. Look for drug duplication and drug–drug interactions with prescribed medications, herbs, vitamins, and over-the-counter (OTC) drugs. Multiple websites are available for evaluation of drug interactions:

a. www.drugs.com/drug_interactions.html.

b. reference.medscape.com/drug-interactionchecker.

TABLE 3.1 EXAMPLES OF "TALL MAN" LETTERS TO PREVENT CONFUSION AND DECREASE LOOK-ALIKE PRESCRIPTION ERRORS

Drug Name/Tall Man	Confused Drug Name/Tall Man
Bupropion/BuPROPion	Buspirone/BusPIRone
Clomiphene/ClomiPHENE	Clomipramine/ClomiPRAMINE
Dobutamine/DOBUTamine	Dopamine/DOPamine
Glipizide/GlipiZIDE	Glyburide/GlyBURIDE
Nicardipine/NiCARdipine	Nifedipine/NIFEdipine
Prednisone/PrediSONE	Prednisolone/PrednisoLONE

Source: U.S. Food and Drug Administration. (2020). Name differentiation project. Retrieved from https://www.fda.gov/Drugs/DrugSafety/MedicationErrors/ucm164587.htm.

c. www.rxlist.com/drug-interaction-checker.htm.
d. www.rxlist.com/grapefruit-page3/supplements.htm.

DEPRESCRIBING

A. Deprescribing is the planned and supervised process of dose reduction or stopping medications that may cause harm or no longer provide a benefit. The strongest predictor of a potentially harmful medication is the total number of prescription drugs. The decision to stop a medicine should be weighed against the benefit. The clinician, the individual, and the family are vital partners in making an informed decision to deprescribe. Deprescribing is a trial, and medications may be restarted when appropriate.

B. Clients may combine prescribed medications with OTC medications/supplements in ways that may be harmful. Minimizing the prescribed drugs helps mitigate this risk.

C. Age-related changes occur in pharmacodynamics resulting in drug–receptor affinity, altering effective and toxic concentrations. Pharmacokinetic parameters, including drug absorption and bioavailability, distribution, and elimination, are affected by age. There is an age-related decline in serum albumin concentrations. Drug excretion by the kidneys declines; therefore, drugs excreted mainly by the kidneys may accumulate and significantly prolong the duration of action. Many older adult clients have renal impairment, but serum creatinine may not rise due to reduced muscle mass. There is no significant age-related decline in liver capacity to detoxify, metabolize, and eliminate drugs. However, medications metabolized by cytochrome P450 or similar liver enzymes may result in competitive inhibition of each other, resulting in slow clearance, drug accumulation, and toxicity.

D. Drug overdose and toxicity is a leading cause of acute confusion, particularly in older people. The risk of drug-related cognitive impairment increases with the number of medications prescribed.

E. Polypharmacy increases the risk of drug–food interaction when a food interferes with the drug's absorption or effect. For example:

1. A high-fiber diet decreases digoxin bioavailability, leading to subtherapeutic serum concentrations and an increased risk of treatment failure.

2. Potassium-rich foods may increase the risk of hyperkalemia when potassium-sparing diuretic or angiotensin-converting enzyme (ACE) inhibitors are prescribed.

F. Poor nutritional status may interfere with drug efficacy and excretion, leading to a higher risk of drug toxicity. Evaluate if the client has difficulties chewing or is altering medications by crushing because of swallowing problems. Some medicines should not be crushed because it changes the absorption or stability of the drug, causes an irritant effect, or has an unacceptable taste.

G. Medications designed to release slowly should not be crushed or chewed since the total dose will be released more rapidly than intended. A resource guide for crushing medications is available at www.ismp.org/recommendations/do-not-crush. Modified-release medications have abbreviations:

1. Long-acting (LA).
2. Controlled-release (CR).
3. Extended-release (XR).
4. Slow-release (SR).
5. Controlled delivery (CD).
6. Prolonged-release (XL).
7. When crushed, enteric-coated (EN or EC) medication loses the protection to the stomach or assurance of the drug's delivery beyond the stomach.
8. Film-coated (FC) and sugar-coated medications may require that coating to prevent rapid degradation of the medicine or to mask the taste.
9. A tablet may have a scoreline to facilitate half doses, but this does not necessarily mean it can be safely crushed.
10. *Drugs with a small therapeutic window, such as digoxin, carbamazepine, theophylline, and phenytoin, should not be crushed because of the increase in toxicity.*

H. The Beers criteria from the AGS (see Appendix C) is the most widely used geriatric resource for evaluating the safety and efficacy of medication use and includes recommendations based on a review of the literature regarding the quality of evidence, frequency and severity of harm, and availability of better treatment classes of medications, and makes recommendations for use or avoidance in older adults:

1. Benzodiazepines: Do not combine two or more other central nervous system active drugs due to increase in delirium and fall risks. Alternatives for geriatrics with anxiety include selective serotonin reuptake inhibitor (SSRI; except for paroxetine and not in clients at risk or with history or risk of falls) or the serotonin-norepinephrine reuptake inhibitor (SNRI) buspirone. Consider nonpharmacologic interventions or low-dose trazodone or melatonin for older adults with insomnia.

▶

2. Proton pump inhibitors (PPIs): Use for ≥8 weeks may be considered if the client has H2-blocker failure and a high risk of ulcer. Recommend referral to a gastroenterologist to collaborate management or deprescribing regimen if the older adult continues to use PPI for 8 to 12 weeks' duration.

3. Antihyperglycemics: Avoid thiazolidinediones (TZDs) or closely monitor geriatrics with fluid retention or risk of heart failure.

4. Antipsychotics: Geriatrics are particularly vulnerable to experience orthostatic hypotension, bradycardia, and urinary and bowel retention.

5. Cholinesterase inhibitors (ChEIs): Geriatrics are particularly vulnerable to experience orthostatic hypotension, bradycardia, and urinary and bowel retention.

I. Criteria for evaluating appropriate prescribing include the **S**creening **T**ool of **O**lder **P**eople's potentially inappropriate **P**rescriptions (STOPP) and the **S**creening **T**ool to **A**lert doctors to **R**ight **T**reatments (START). STOPP/START is an educational tool for pharmacists and prescribers. STOPP/START prescription criteria consist of a set of inappropriate combinations of medicines and disease (STOPP) and a set of recommended treatments for given conditions (START):

1. The STOPP/START Toolkit is available for use at www.cgakit.com/m-2-stopp-start.

2. The 10 most common drug classes implicated in hospital admissions for adverse drug reactions (ADRs), in the order of ADR-related admissions, are nonsteroidal anti-inflammatory drugs (NSAIDs), including aspirin, diuretics, anticoagulants, ACE inhibitors and angiotensin II receptor blockers, antidepressants, beta-blockers, opiates, digoxin, prednisolone, and clopidogrel.

3. Anticholinergic medication use is of specific concern in older people. Figure 3.1 shows the increasing adverse side effects of anticholinergic drugs. Anticholinergic drugs are mentioned explicitly in STOPP for their risk of toxicity in combination with tricyclic antidepressants, phenothiazines, benzodiazepines, neuroleptics, and antihistamines. The dangers of geriatric syndromes related to incontinence, physical impairments (gait speed and handgrip strength), falls, cognitive decline, and delirium increase incrementally as the client's anticholinergic burden increases. Cholinergic deficits may accompany aging and play a central role in the pathophysiology of Alzheimer disease.

J. Another resource for deprescribing guidelines is the Canadian Deprescribing Network (CaDeN). Their website (deprescribing.org/resources) links to free and readily available deprescribing brochures and resources. In addition, there are algorithm resources for tapering and deprescribing benzodiazepines, antipsychotics, PPIs, antihyperglycemics, ChEIs, and memantine for Alzheimer, dementia, or Parkinson, which are noted in Appendix D.

K. One of the deprescribing research links noted on the www.deprescribing.org site is Medstopper, a web-based educational tool that assists in stopping or tapering medications. The tool identifies medicines with the highest stopping priority, tapering suggestions, links to available risk calculators, and mentions whether there are Beers/STOPP criteria for the medications. Medstopper is available at medstopper.com.

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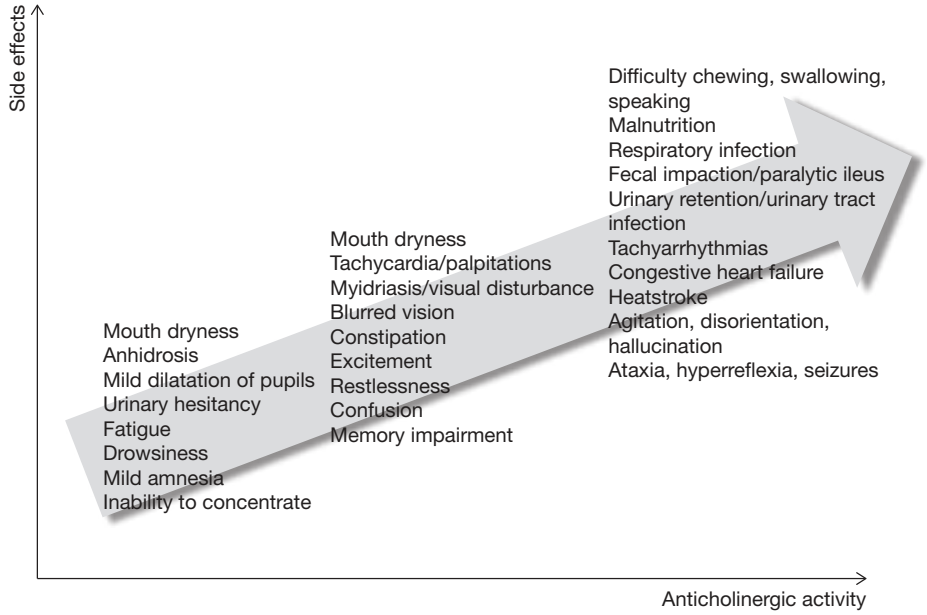


FIGURE 3.1 The increasing adverse side effects from anticholinergic drugs.

Source: Collamati, A., Martone, A. M., Poscia, A., Brandi, V., Celi, M., Marzetti, E., Cherubini, A., and Landi, F. (2016). Anticholinergic drugs and negative outcomes in the older population: From biological plausibility to clinical evidence. *Aging Clinical Experimental Research, 28*(1), 25–35. doi:10.1007/s40520-015-0359-7.

FUNCTIONAL ABILITY

A. Age-related physiologic changes put aged clients at risk for declines in physical function and increased risk for falls. Determination of functional ability and fall risk occurs with assessments and screening tools. A basic evaluation includes an evaluation of a person's dependence/independence in the performance of activities of daily living (ADL), including personal self-care and community interactions. If the client cannot perform ADLs independently, identify family and other support for referrals.

ACTIVITIES OF DAILY LIVING

A. ADL evaluation of basic self-care activities includes the following:

1. Dressing: Use an open-ended question, such as "Did you dress yourself today?" Evaluate if the client can dress independently, choose weather-appropriate clothing, use buttons and zippers, and bend, grasp, and stand.

2. Eating: Evaluate if the client can use utensils, cut food, and swallow without choking.

3. Ambulating: Observation of the client entering the room and movement during the office visit evaluates if the client can walk independently, has gait abnormalities, or requires assistive devices.

4. Toileting: Evaluate if the client is continent of urine and bowel, or uses disposable undergarments, bedpan, or bedside commode. What is needed for mobility to get to the toilet? Is the client able to self-clean?

5. Hygiene: Evaluate the client's need for assistance with bathing, showering, shaving, brushing teeth, brushing hair, and other hygiene tasks.

B. The Barthel Index of ADLs is an example of an ADL assessment tool. It is located at www.sralab.org/sites/default/files/2017-07/barthel.pdf.

INSTRUMENTAL ACTIVITIES OF DAILY LIVING

A. The assessment of instrumental activities of daily living (IADL) (e.g., executive function) includes determining the ability to care for self and interact with the community. In addition, IADL consists of the ability of the client to shop and prepare food, handle finances and medications, communicate using the telephone, and perform tasks of housekeeping and laundry. The Lawton and Brody Instrumental Activities of Daily Living Scale is one of the most widely used and referenced tools (www.alz.org/careplanning/downloads/lawton-iadl.pdf).

B. Occupational and physical therapy referrals should be considered.

DRIVING ASSESSMENT FOR OLDER ADULTS

A. With the aging population, clinicians will increasingly evaluate the need for client to stop driving. The fourth edition of the American Geriatrics Society's *Clinician's Guide to Assessing and Counseling Older Drivers* (available at geriatricscareonline.org/ProductAbstract/clinicians-guide-to-assessing-and-counseling-older-drivers-4th-edition/B047#) provides decision support for clinicians.

1. Consult the Department of Motor Vehicles (DMV) in your state for information related to regulations for older drivers, including the requirement for a medical evaluation before license renewal, shorter renewal periods for aged drivers, and passing inperson vision, road, or written tests for renewal.

2. The decision to "take the keys away" is one of the significant changes impacting adult independence and self-worth. Changes in eyesight, hearing, reflex response, judgment, "pedal confusion," physical conditions, medications, and memory all impact deciding when to have the client stop driving. Family members look to healthcare providers to assist in determining whether their loved one has the capacity and ability to continue driving. The assessment begins by asking the client open-ended questions:

a. How comfortable are you driving?
b. Are you able to drive in the evening?
c. Do you drive in the rain or other bad weather?
d. Do you drive when it is rush hour?
e. Do you avoid driving on the interstate?
f. Do you ever get lost?
g. Have you ever "lost" your car? Have you had problems misplacing your keys, purse, or wallet?
h. Have you had any accidents, including minor "fender benders"?
i. Have you had any recent traffic tickets or warnings?
j. Do other drivers honk their horns when you are driving or stopped at a stop sign or signal light?
k. Do you have anyone to help you with errands?
l. Do you have any other optional transportation available, such as family and friends, senior citizen van services, taxi services, or a church van?

3. Interview family members:

a. Assess if they discussed the issue of the need to stop driving. The same questions asked to the client can be asked of the family. A three-module educational tool is available at the AARP website (www.aarp.org/auto/driver-safety/we-need-to-talk). The modular seminar covers the following subjects: "meaning of," "observing driving skills," and "planning conversations."
b. Have they been driven by their loved one, and what did they observe when they were passengers?
c. Have they noted any memory issues?
d. How active is their loved one? Have they noted any physical limitations around the home?
e. Has the family seen any dents or scraped paint on the car?

4. Evaluate alcohol consumption.

5. During a "brown bag" visit, review all medications to evaluate polypharmacy issues, drug duplication, and side effect profiles that impact driving.

6. Rereview all medical diagnoses. The client may be seeing multiple doctors, and records may not be current.

a. Vision issues: glaucoma, cataracts, macular degeneration, retinitis pigmentosa, diabetic retinopathy, abnormality of the field of vision (entire spatial area, including central and peripheral vision), visual acuity (able to distinguish details and shapes of objects, contrast sensitivity), color vision deficit, need for corrective glasses or contacts.
b. Hearing: hearing loss.
c. Neurologic disorders: Parkinson disease, multiple sclerosis, epilepsy (last seizure), syncope, Alzheimer disease, stroke (hemiparesis), cognitive impairment, and Huntington disease.
d. Heart/vascular: hypertension, unstable angina, arrhythmia, and aortic aneurysm (nonsurgical treatment).
e. Respiratory issues, including sleep.
f. Sleep issues: hypersomnia, excessive daytime sleepiness, obstructive sleep apnea.
g. Diabetes: poorly controlled diabetes/hypoglycemia. ▶

h. Psychiatric disorders and substance abuse: aggressiveness, behavioral disorders, depression, anxiety, abuse or dependence with alcohol, cannabis, and benzodiazepine use.
i. Physical limitations: amputation, range of motion, manual dexterity, arthritis, difficulties with ADL, hemiparesis.
j. Dementia/cognitive impairment.

7. Referrals:
a. Functional assessment by an occupational therapist.
b. Physical therapy.
c. Medical specialty consultation.
d. On-road assessment by the DMV.

GAIT AND MOBILITY

A. In addition to ADLs, gait speed predicts functional decline and mortality in older adults. Assessing gait speed may identify clients who need further evaluation. Additionally, it helps identify frail clients who might not benefit from the treatment of chronic asymptomatic diseases.

1. Mobility and balance are indicators of the ability to live independently.

2. The Timed Up and Go (TUG) test is a basic mobility assessment evaluating static and dynamic balance. The TUG test assesses the ability to stand from a sitting position, walk, turn, stop, and sit (see Exhibit 3.3). The provider administering the test needs a chair with armrests and a stopwatch. For client with assistive devices (e.g., cane), the device is used during the trial.
a. The client should be wearing regular shoes.
b. The test starts when the client begins rising from the chair and ends when the client is seated.
c. The client should be seated in a resting position with both arms on the chair armrests. Then, the client stands up from the chair, walks approximately 10 feet (3 m), turns around, and returns to sit down in the chair.
d. Verbal cueing through the test may be needed for a person with cognitive impairment.
e. Evaluation:
i. The expected time to complete the TUG test is between 7 and 10 seconds, indicating no balance or mobility issues.
ii. An older adult who takes 12 or more seconds to complete the TUG test is at high risk for falling.
iii. Time greater than 20 seconds indicates the need for assistance and further evaluation.
iv. Taking 30 or more seconds indicates a high risk for falls and higher dependence needs.
f. The TUG test can be repeated periodically to assess the decline in gait, mobility, and balance.

3. The Tinetti Assessment Tool measures both balance and gait. This tool is located at www.whca.org/files/2013/04/TINETTI_assessment_tool.pdf.
a. Balance:
i. Individual scores are given for tasks including sitting balance, rising, standing, balance when the examiner nudges the client, balance with eyes closed, balance ability when turning 360°, and safety when sitting down.
ii. The maximum score for the balance portion of the Tinetti test is 16 points.
b. Gait:
i. Individual scores are given for tasks/items, including the initiation of gait, step length and height of raising the feet to walk, symmetry of stepping, continual stepping, walking straight or a deviation of the path, trunk movement, and walking stance. ▶

EXHIBIT 3.3 The Timed Up and Go (TUG) Test

Patient: _____ **Date**: _____

Time: _____ a.m./p.m.

The TUG test

A. Purpose: To assess mobility.
B. Equipment: A stopwatch.
C. Directions: Clients wear their regular footwear and can use a walking aid if needed. Begin by having the client sit back in a standard armchair and identify a line 3 m or 10 ft away on the floor.

Instructions to the client:

A. When I say **"go,"** I want you to:
1. Stand up from the chair.
2. Walk to the line on the floor at your normal pace.
3. Turn.
4. Walk back to the chair at your normal pace.
5. Sit down again.
B. On the word **"go,"** begin timing.

Stop timing after the client has sat back down and record:

Time: _____ seconds.

An older adult who takes ≥12 seconds to complete the TUG test is at high risk for falling.

Observe the client's postural stability, gait, stride length, and sway.

Check all that apply: ☐ Slow tentative pace ☐ Loss of balance ☐ Short strides ☐ Little or no arm swing ☐ Steadying self on walls ☐ Shuffling ☐ En bloc turning ☐ Not using assistive device properly

Note: For relevant articles, go to www.cdc.gov/steadi/index.html.

ii. The maximum score of the gait portion of the Tinetti test is 12 points.

c. The maximum score for both the balance and gait tests of the Tinetti combined is 28 points:

i. Scores below 19 are a general indicator of high risk for falls.

ii. Scores between 19 and 24 indicate the client has a risk for falls.

4. The 30-second chair stand test is used to test leg strength and endurance (Exhibit 3.4).

5. Consider a physical therapy and/or an occupational therapy referral for gait and/or balance problems.

EXERCISE

A. Physical activity is a crucial step older adults can take to improve health. Physical activity provides many benefits: preventing muscle and bone loss, reducing the risk of falling and fractures, preventing or delaying conditions including diabetes and heart disease, reducing symptoms of anxiety and depression, and allowing prolonged independence. The second edition of the *Physical Activity Guidelines for Americans* was released in 2018 and provides evidence-based guidance to improve health through physical activity (available at health.gov/sites/default/files/2019-09/Physical_Activity_Guidelines_2nd_edition.pdf). The benefits of physical activity outweigh the possibility of adverse outcomes.

B. The National Center on Health, Physical Activity and Disability (NCHPAD) website (www.nchpad.org/Disability~and~Aging) provides information regarding exercise for clients with disabilities and conditions from acquired brain injury, arthritis, chronic fatigue syndrome, rheumatoid arthritis, and stroke.

C. Preexercise medical evaluation:

1. Before beginning a sports or vigorous exercise program, older adults should undergo screening, emphasizing the detection of cardiovascular risks.

2. When screening is suspicious for clinical pathology, additional testing is performed.

D. Key guidelines for adults 18 to 64 years:

1. Some physical activity is better than none, and adults who participate in any amount of physical activity gain some health benefits. All adults should avoid inactivity.

2. For substantial health benefits, adults should do at least 150 minutes (2 hours and 30 minutes) a week of ▶

EXHIBIT 3.4 The 30-Second Chair Stand Test

Patient: _____ **Date:** _____

Time: _____ **a.m./p.m.**

Purpose: To test leg strength and endurance.

Equipment:

A. A chair with a straight back, without armrests (seat 17-in. high).
B. A stopwatch.

Instructions to the client:

A. Sit in the middle of the chair.
B. Place your hands on the opposite shoulder crossed at the wrists.
C. Keep your feet flat on the floor.
D. Keep your back straight and keep your arms against your chest.
E. On **"go,"** rise to a full standing position and then sit back down again.
F. Repeat this for 30 seconds.

On **"go,"** begin timing.

If the client must use their arms to stand, stop the test. Record **0** for the number and score.

Count the number of times the client comes to a full standing position in 30 seconds.

If the client is over halfway to a standing position when 30 seconds have elapsed, count it as a stand.

Record the number of times the client stands in 30 seconds.

Number: _____ **Score** _____

A below-average score indicates a high risk for falls.

Chair Stand: Below-Average Scores

Age	Male	Female
60–64	<14	<12
65–69	<12	<11
70–74	<12	<10
75–79	<11	<10
80–84	<10	<9
85–89	<8	<8
90–94	<7	<4

Note: For relevant articles, go to www.cdc.gov/steadi/index.html.

moderate-intensity or 75 minutes (1 hour and 15 minutes) a week of vigorous-intensity aerobic activity. Aerobic activity should be performed in episodes of at least 10 minutes and spread throughout the week.

a. Aerobic physical activities intense enough and performed long enough to maintain or improve an individual's cardiovascular fitness include biking slowly, general gardening, using a manual wheelchair, walking briskly, and water aerobics. Vigorous activities are basketball, jumping rope, running, swimming laps, martial arts, and dancing.

b. The talk test can be used to evaluate moderate or vigorous activities. If individuals can talk but not sing, they are participating at a moderate level. If only a few words can be said without stopping to catch a breath, it is considered vigorous activity.

3. For additional and more extensive health benefits, adults should increase their aerobic physical activity to 300 minutes (5 hours) a week of moderate-intensity or 150 minutes a week of vigorous-intensity aerobic physical exercise.

4. For additional benefits, adults should also do muscle-strengthening activities that are of moderate or high intensity and involve all major muscle groups on 2 or more days a week.

E. Key guidelines for older adults:

1. The key guidelines for adults also apply to older adults. In addition, the following guidelines are only for older adults:

a. When older adults cannot do 150 minutes of moderate-intensity aerobic activity a week due to chronic conditions, they should be as physically active as their condition allows.

b. Older adults at risk for falls should do exercises that focus on balance.

c. Older adults with chronic conditions should understand how their conditions affect their ability to do regular physical activity safely.

F. Key messages for people with chronic medical conditions:

1. Adults with chronic conditions obtain important health benefits from regular physical activity.

2. When adults with chronic conditions do activity according to their abilities, physical activity is safe.

3. Adults with chronic conditions and symptoms should consult their healthcare provider about the types and amounts of activity appropriate for them.

G. Key guidelines for adults with disabilities:

1. Adults with disabilities who can should get at least 150 minutes a week of moderate-intensity or 75 minutes a week of vigorous-intensity aerobic activity, or an equivalent combination of moderate- and vigorous-intensity aerobic exercise. Aerobic activity should be performed in episodes of at least 10 minutes, and preferably it should be spread throughout the week.

2. Adults with disabilities who can do so should also do muscle-strengthening activities of moderate or high intensity that involve all major muscle groups on 2 or more days a week as these activities provide additional health benefits.

3. When adults with disabilities cannot meet the guidelines, they should engage in regular physical activity according to their abilities and avoid inactivity.

4. Adults with disabilities should consult their healthcare provider about the appropriate amounts and types of physical activity for their abilities.

5. *How to Exercise If You Have Limited Mobility: Chair Exercises and Fitness Tips for People With Injuries or Disabilities* is available at www.helpguide.org/articles/healthy-living/chair-exercises-and-limited-mobility-fitness.htm.

H. Key guidelines for safe physical activity:

1. To do physical activity safely and reduce the risk of injuries and other adverse events, people should:

a. Understand the risks and be confident that physical activity is safe.

b. Choose to do physical activity appropriate for their fitness level and health goals.

c. Increase physical activity over time whenever more activity is necessary to meet goals. Inactive people should "start low and go slow" by gradually increasing how often and how long activities are done.

d. Protect themselves by using appropriate safety gear and equipment, looking for safe environments, following rules and policies, and making sensible choices about when, where, and how to be active.

FALL ASSESSMENT

A. Among older Americans, falls are the number one cause of injuries and death from injury. Many people who fall, even if they are not injured, become afraid of falling. This fear may cause a person to cut down on their activity and become sedentary, which in turn increases weakness and risk of falls:

1. One in four people age 65 and older fall each year.

2. One out of five falls causes serious injuries such as fractures or traumatic brain injuries (TBIs).

3. Less than half of clients who have fallen talk to their healthcare provider about it.

4. The Centers for Disease Control and Prevention (CDC) also notes the following statistics:

a. Over 95% of hip fractures are caused by falls.

b. Males are more likely to die from a fall.

c. Older adults who fall are more likely to be admitted to a long-term facility for a year or longer.

B. Assess whether the client had some physical feeling precipitating the fall, such as shortness of breath, vertigo, cardiac pain/palpitations, or other signs. Evaluate if other factors, such as alcohol, medicines, and over-the-counter meds, may have been a contributing factor.

1. Did the client lose consciousness? Was the client able to get up independently?

2. What injuries occurred in previous fall(s)?

3. The interaction of multiple risk factors causes most falls. The more risk factors a person has, the greater the risk for falls. Therefore, the clinician's first focus for fall prevention is the evaluation of modifiable intrinsic and extrinsic risk factors (Table 3.2).

4. Client teaching: *See Client Teaching Guide for this chapter, "Safety Issues: Fall Prevention."*

C. The CDC has developed the Stopping Elderly Accidents, Deaths, and Injuries (STEADI) program. STEADI uses established clinical guidelines and strategies to help providers address fall risk and identify modifiable risk factors. The core elements of STEADI include screening older clients for fall risk, assessing their modifiable fall risk factors, and intervening to ▶

TABLE 3.2 INTRINSIC AND EXTRINSIC FALL RISK FACTORS

Intrinsic Risk Factors (Client Conditions)	Extrinsic Risk Factors (Environment)
History of falls (greatest risk factor) **Age >65**	Inappropriate or lack of footwear
Muscle weakness	
Gait and balance problems	
Poor vision/cataracts	Improper use or lack of assistive devices
Postural hypotension	Psychoactive medications
Fear/anxiety associated with falls Dizziness/vertigo/vestibular disorders	Environment: throw rugs, wet floors, slippery or uneven surfaces, cluttered room, lack of stair handrails, poor stair design, lack of bathroom grab bars, dim lighting or glare, obstacles, and tripping hazards (loose cords or wires)
Chronic conditions including stroke, Parkinson disease, depression, dementia, arthritis, osteoporosis, incontinence (urgent trips to the bathroom), diabetes (proprioception from diabetic peripheral neuropathy), and reduced proximal muscle strength and reaction due to vitamin D deficiency	

reduce risk using clinical and community strategies. Information about the STEADI program is located at www.cdc.gov/steadi/index.html. In addition, the CDC's National Center for Injury Prevention and Control (NCIPC) has developed an Algorithm for Fall Risk Assessment and Interventions (see Figure 3.2). The fall risk algorithm is also located at www.cdc.gov/steadi/pdf/STEADI-Algorithm-508.pdf. The *Stay Independent* brochure is found at ocagingservicescollaborative.org/wp-content/uploads/2014/07/Stay-Independent-Fall-Risk-Assessment.pdf. The Fall Risk Checklist (Exhibit 3.5) is one toolkit resource of the CDC NCIPC STEADI program that is available for clinicians.

D. When you see clients 65 and older, make the following three questions a routine part of your exam:

1. Have you fallen in the past year?
2. Do you feel unsteady when standing or walking?
3. Do you worry about falling?

If your clients answer "yes" to any of these key screening questions, they are considered at increased risk of falling. Further assessment is recommended.

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PSYCHOLOGICAL WELL-BEING

A. The psychological well-being of aged adults is at risk due to the functional decline and the risk of cognitive defects associated with normal aging or pathology. Depression in adults may present atypically, and aged adults should be screened for depression. Clients should also be screened for changes in cognition and the development of dementia.

AFFECTIVE ASSESSMENT

A. Depression in older adults frequently presents atypically. It may present as apathy and cognitive impairment, disengagement with activities previously found enjoyable, or as weight loss. On the other hand, it may present as simply as adults answering questions they know the answer to as "I don't know." Beyond reductions in functional status and psychological well-being, depression results in a higher risk of suicide, increased use of healthcare services, and increased mortality in the aged adult. There are multiple validated screening tools for depression in the aging adult. Stanford University offers the Mood Assessment Scale and the Geriatric Depression Scale (GDS), both available in various languages.

1. The GDS in multiple languages is located at web.stanford.edu/~yesavage/GDS.html.
2. A web-based version of the GDS is available at www.medafile.com/GDS15.htm, and there are multiple smartphone apps with the GDS available for free.
3. Initial assessment utilizing the five-item version of the GDS can identify those at high risk for depression. This scale can be used rapidly in the office setting with yes/no responses. Two positive of five indicators suggest the full 15-point GDS should be performed.
4. Appropriate referral for psychological counseling and medication management should be considered.

COGNITIVE/DEMENTIA ASSESSMENT

A. Vitamin B_{12} deficiency is a common comorbidity in older clients, and routine assessment of vitamin B_{12} levels in older clients diagnosed with dementia is recommended.

B. Many families voice early concerns about the cognitive changes they see in their older parents and family members. Typical changes that occur with aging compared with the "10 Alzheimer warning signs" are noted in Table 3.3. ▶

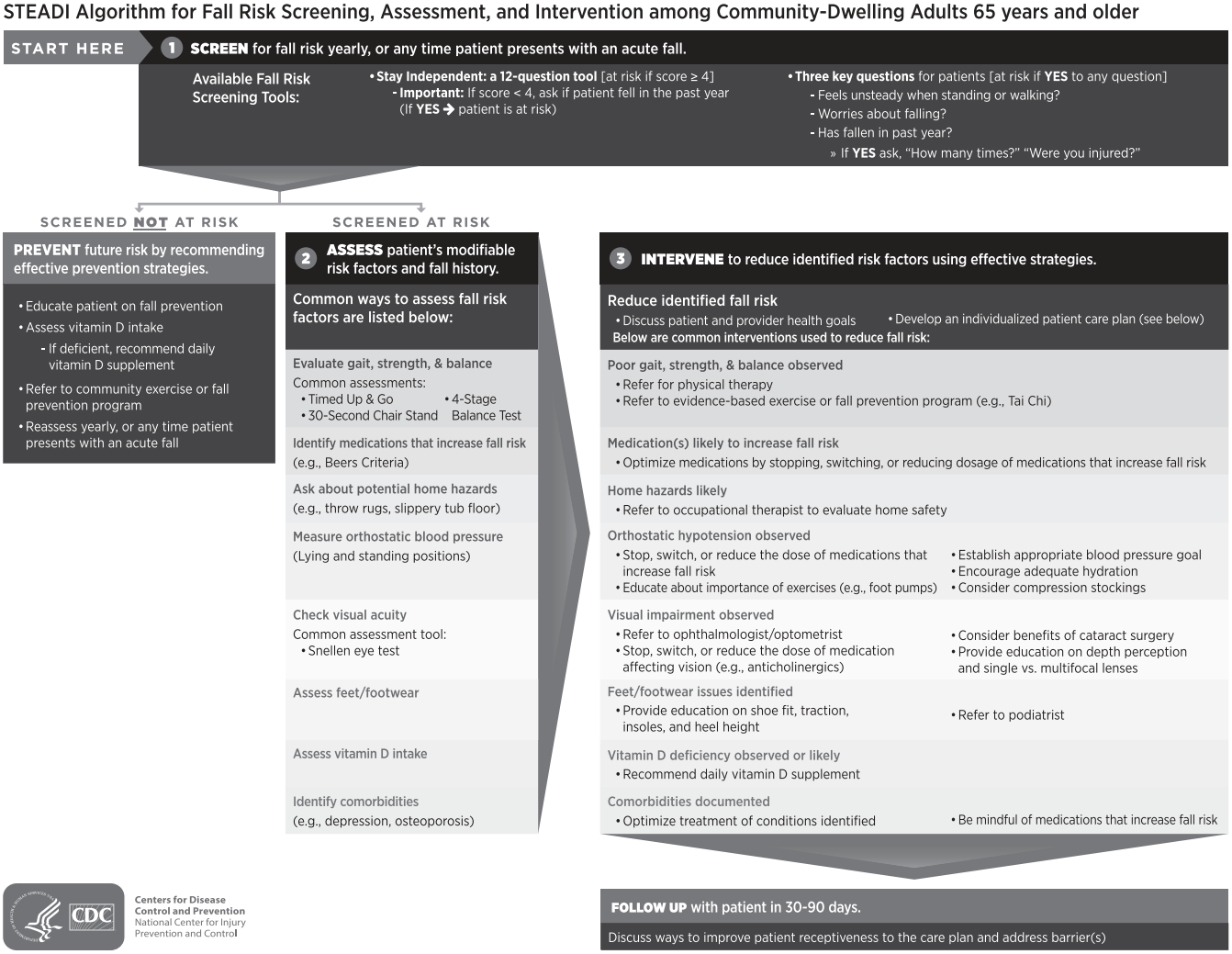


FIGURE 3.2 Centers for Disease Control and Prevention algorithm for fall risk screening, assessment, and intervention. STEADI, Stopping Elderly Accidents, Deaths, and Injuries.

Source: Centers for Disease Control and Prevention. (2019). *The Stay Independent* brochure. https://www.cdc.gov/steadi/pdf/STEADI-Algorithm-508.pdf.

C. Alzheimer disease is the most common form of dementia. Alzheimers.net notes that Alzheimer disease has seven distinct stages. For full descriptions of "What Are the 7 Stages of Alzheimer's Disease?" visit www.alzheimers.net/stages-of-alzheimers-disease.

1. Stage 1: no impairment.
2. Stage 2: very mild decline (losing keys, forgetting familiar names).
3. Stage 3: mild decline (memory and concentration problems).
4. Stage 4: moderate decline (difficulty planning dinner or paying bills on time).
5. Stage 5: moderately severe decline (unable to recall events or remember important dates).
6. Stage 6: severe decline (needs help getting dressed, personality begins to change).
7. Stage 7: very severe decline (trouble eating, swallowing, speaking, and all motor function).

D. There is no "gold standard" tool to evaluate cognitive function and impairment. An initial evaluation provides a baseline for serial surveillance and may also indicate the need for further evaluation. A yearly assessment can determine and identify a gradual decline over time. Assessments that show decline or impairment require further evaluation. The National Institute on Aging notes someone with Alzheimer disease may experience the following symptoms:

1. Getting lost.
2. Trouble handling money and paying bills.
3. Repeating questions.
4. Taking longer to complete routine daily tasks.
5. Poor judgment.
6. Losing things or misplacing them in odd places.
7. Difficulty remembering newly learned information.
8. Unfounded suspicions about family, friends, and professional caregivers.
9. Difficulty speaking, swallowing, and walking.

E. The cognitive assessment does not need to be lengthy. The Memory Impairment Screen (MIS), the General Practitioner Assessment of Cognition (GPCOG), and the Mini-Cog are the most useful in the primary care setting. These assessments require 5 minutes to administer and are very similar. ▶

EXHIBIT 3.5 Fall Risk Checklist

Patient: _____ **Date:** _____

Time: _____ **a.m./p.m.**

Fall Risk Factor Identified	Factor Present?		Notes
Fall history			
Any falls in the past year?	☐ Yes	☐ No	
Worries about falling or feels unsteady when standing or walking?	☐ Yes	☐ No	
Medical conditions			
Problems with heart rate and/or rhythm?	☐ Yes	☐ No	
Cognitive impairment?	☐ Yes	☐ No	
Incontinence?	☐ Yes	☐ No	
Depression?	☐ Yes	☐ No	
Foot problems?	☐ Yes	☐ No	
Other medical conditions (specify)?	☐ Yes	☐ No	
Medications			
Any psychoactive medications, medications with anticholinergic side effects, and/or sedating OTCs (e.g., Benadryl, Tylenol PM)?	☐ Yes	☐ No	
Gait, strength, and balance			
TUG test ≥12 seconds?	☐ Yes	☐ No	
30-second chair stand test below-average score? (See the following table.)	☐ Yes	☐ No	
Four-stage balance test full-tandem stance less than 10 seconds?	☐ Yes	☐ No	
Vision			
Acuity less than 20/40, or no eye exam in more than 1 year?	☐ Yes	☐ No	
Postural hypotension			
A decrease in systolic BP ≥20 mmHg, or a diastolic BP of ≥10 mmHg, or light-headedness or dizziness from lying to standing?	☐ Yes	☐ No	
Other risk factors (specify)			
	☐ Yes	☐ No	
	☐ Yes	☐ No	

Chair Stand: Below-Average Scores

Age	Male	Female
60–64	<14	<12
65–69	<12	<11
70–74	<12	<10
75–79	<11	<10
80–84	<10	<9
85–89	<8	<8
90–94	<7	<4

BP, blood pressure; OTC, over-the-counter; TUG, Timed Up and Go.

TABLE 3.3 TEN ALZHEIMER WARNING SIGNS

Issue	Common Symptoms of Early-Stage Alzheimer Disease	Typical of Aging
Memory loss	Forgetting new information and asking for the same information over and over	Forgetting names or appointments occasionally and remembering them later
Difficulty performing familiar tasks	Difficulty planning or completing everyday tasks, losing track of steps involved in preparing a meal or making a phone call	Occasionally forgetting why you came into a room or what you were planning to say
New problems with writing or speaking	Forgetting simple words or substituting unusual words, making speech or writing hard to understand	Sometimes having trouble finding the right word
Confusion with time and place	Getting lost in familiar places, forgetting where you are and how you got there, and how to get back home	Forgetting the day of the week or where you are going
Poor or decreased judgment	Dressing inappropriately for the weather, giving away large sums of money to telemarketers	Making a questionable or debatable decision from time to time
Problems with abstract thinking	Unusual difficulty performing complex mental tasks, especially relating to numbers	Finding it challenging to balance a checkbook
Misplacing things and losing the ability to retrace steps	Putting things in unusual, inappropriate places	Misplacing keys or a wallet, but being able to retrace steps to find it later
Changes in mood or behavior	Rapid mood swings, extreme confusion, anxiety, suspiciousness, dependence on a family member	Occasionally feeling sad or moody
Trouble understanding visual images and spatial relationships	Change in visual processing, difficulty reading, judging distance, and determining color or contrast, which may cause problems with driving	Vision changes related to cataracts
Withdrawing from social activities	Avoiding being social due to the changes experienced, becoming passive, sitting in front of the television for hours, sleeping more than usual, or not wanting to perform ADL	Sometimes feeling weary of work or social obligations

ADL, activities of daily living.
Source: Reprinted with permission from a Place *for* Mom Connecting Families to Senior Living. (Copyright © 2018). 10 Alzheimer's warning signs. Retrieved from https://www.aplaceformom.com/planning-and-advice /articles/alzheimers-warning-signs.

TABLE 3.4 SCORING THE MINI-COG ASSESSMENT TEST FOR COGNITIVE IMPAIRMENT

Three recalled words	Negative for cognitive impairment
One to two recalled words + normal CDT	Negative for cognitive impairment
One to two recalled words + abnormal CDT	Positive for cognitive impairment
Zero recalled words	Positive for cognitive impairment

CDT, clock-draw test.

F. The MIS is available on the Alzheimer's Association website at www.alz.org/media/Documents/memory-impairment-screening-mis.pdf; the MIS assessment includes:

1. Evaluation of the client's ability to recall four words: checkers, saucer, telegram, and red cross.

2. If necessary, provide category cue words for any of the four words not recalled freely.

G. The Mini-Cog assessment is a short testing tool used to detect cognitive impairment and the need for further evaluation (Table 3.4). The Mini-Cog assessment tool can be found at the Alzheimer's Association website at www.alz.org/media/Documents/mini-cog.pdf. Administration of the Mini-Cog involves three steps:

1. Ask the client to remember three unrelated words (there are six versions of word lists available). Then, ask the client to repeat the words to ensure understanding.

2. Administer the clock-draw test (CDT). See *Section II: Clock-Draw Test* for a copy of the CDT and instructions.

3. Ask the client to recall the three words from step 1.

H. An interactive program test for the GPCOG is located on the following website: www.gpcog.com.au.

I. The Mini-Mental State Examination (MMSE) or the Folstein test is a useful screening tool for cognitive impairment (available at www.fammed.usouthal.edu/Guides&JobAids/Geriatric/MMSE.pdf). It can be used to classify the severity of cognitive impairment. The 30-item tool takes 5 to ▶

10 minutes to administer. Scores greater than or equal to 24 (out of 30) indicate normal cognition. Below this, mild impairment (19–23 points), moderate impairment (10–18), or severe impairment (≤9 points) can be gauged.

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SOCIOENVIRONMENTAL STATUS

A. Although factors associated with the social and environment of the aged person may not heavily impact the medical status of aged adults, they do affect the client's overall health and should be assessed. Social support systems and environmental adaptions often play a prominent role in determining whether the aged adult can stay at home or if other arrangements need to be considered. Assessment of the geriatric client's financial status allows for referral to government assistance programs that may ease the burden of staying home or acquiring assistive services when required.

PHYSICAL ENVIRONMENT

A. Due to functional and cognitive decline, the aged client may be at higher risk of environmental hazards. It is crucial to screen for use of smoke and carbon monoxide detectors. Especially when physical disability is present, assessment for stairs and access ramps is essential. Physical and occupational therapy may provide insight into the need for assistive devices such as grab bars, shower seats, lift chairs, and instructing on removing rugs to prevent falls. Identification of the mode and method of communicating in the case of emergency or accident should be clear, and consideration for a wearable alert device made when needed.

SOCIAL

A. The strength of the social support network may play a significant role in determining the client's independence. For example, identifying who the aged adult may call if assistance is needed with routine tasks is necessary or if they are unable to provide care for themselves. Discussing the social system also allows for identifying friends and loved ones the aged adult enjoys being around. These questions also will enable a transition to discuss healthcare and financial proxies if the senior adult cannot make decisions.

CULTURAL DIVERSITY AND SENSITIVITY

A. Culture is more than nationality or race. It also includes the aged, the illiterate or those lacking education, and persons with physical limitations. Culture influences a person's reasoning, decisions, and actions. It is the accumulation of learned beliefs, values, habits, and practices.

B. Cultural diversity exists when groups of different cultures coexist within an environment. Knowing that cultural differences exist and not assigning values between cultures reflects cultural sensitivity. However, significant differences may exist in how healthcare is perceived and practiced because of the differing values and beliefs regarding health and illness inherent among people of varying cultural backgrounds.

C. Contributing factors to cultural diversity:

1. Increasing immigration.
2. Efficiency in transportation and travel.
3. Increase in the homeless and poor population.
4. Increase in divorce rate and single parenting.
5. Grandparents raising grandchildren.
6. Substance abuse.
7. Violence.
8. Transgender sex changes.
9. LGBT acceptance.
10. Illiteracy.
11. Increase in non-English-proficient healthcare providers.
12. Federal regulations.
13. Physical limitations (disabilities).

D. Cultural sensitivity is the responsibility of all healthcare providers. Each office visit is an opportunity to gain more knowledge about a client's health beliefs and practices. Inadequate awareness of the client's health beliefs could lead to mistrust. This may result in barriers including inappropriate delivery of care, increased cost, and noncompliance, and may eventually lead to more barriers to healthcare access. In addition, those with physical disabilities often have unpleasant experiences with healthcare encounters, affecting their decisions to seek appropriate care. Title VI of the Civil Rights Act is particular about providing services less than the existing standard of care to anyone based on race, age, sex, color, national origin, or financial status.

E. Thoughtful considerations:

1. The provision of care without being sensitive to the needs of a culturally diverse client may suggest that the healthcare provider's values and beliefs are superior to those of the client's and may lead to a disparity in care. Implementing evidence-based guidelines to a culturally diverse population whose healthcare practices are not always based on scientific studies remains a constant challenge. The limited client involvement in care may result in noncompliance, placing clients at greater risk for health-related complications. The delay in the provision of healthcare can result in life-threatening complications.
2. Numerous resources are available throughout the literature and the internet. Preferences for educational/assessment tools are within the healthcare provider's prerogative.
3. The following are guidelines for promoting cultural sensitivity in the clinical setting:
    a. Provide a cultural diversity self-assessment.
        i. Online internet self-assessment tools, for example, the Centers for Disease Control and Prevention website (see the following section).
        ii. Download self-assessment tools from public sources (see the following section).
        iii. Use existing self-assessment tools and make necessary changes to fit the need (Exhibit 3.6).
    b. Identify the need of the population served.
        i. Understand the community and its health status.
        ii. Evaluate resources, attitudes, and barriers inside the communities and practice location.
            1) Access to resources.
            2) Notification of assistance.

3) Range of assistance options:
a) Transportation.
b) Communication; consider an interpreter (personal vs. automation):
i) Identify bilingual staff.
ii) Use a Health Insurance Portability and Accountability Act (HIPAA)-compliant medically trained translation service.
iii) Provide multilingual written materials.
c) Education (meaningful/multilingual).

c. Educate staff to cultural diversities.
i. Assessments should include client's health values and beliefs (Exhibit 3.7).
ii. Communication should be meaningful.
1) Be precise and clear.
2) Use plain language.
3) Observe facial expressions and body language.
4) Use short sentences to explain lengthy information.

d. Schedule longer appointments if needed.
e. Clarify the limitations of healthcare providers.
f. Identify alternatives offered by healthcare providers.

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EXHIBIT 3.6 Cultural Diversity and Sensitivity Self-Evaluation Form

Using a scale of 1 to 5, with 1 = never and 5 = always, please answer the following questions:

Question	Response Value
1. I am comfortable with my culture and can compromise on situations without sacrificing my integrity.	
2. I think about what I say and how it may affect people with different beliefs and practices.	
3. I am aware that others may stereotype me, and I am willing to proactively get involved and share my beliefs and practices.	
4. I evaluate what the real reasons are when I encounter a conflict with the aged, the disabled, or persons of a different culture.	
5. I am aware of sensitive issues when I am around women and persons of different beliefs and races.	
6. I ask for clarification when I do not understand what others mean.	
7. I am aware of my assumptions about others who are culturally or racially different than myself and I am okay with it.	
8. I object when others tell ethnic jokes.	
9. I listen without interrupting when someone is speaking.	
10. I am comfortable forming friendships with people of different cultures.	
11. I find ways to learn more about different cultures and how to communicate effectively.	
12. I realize that flexibility and empathy allow me to evaluate persons of different cultures without imposing any judgments, which allows me to collaborate effectively.	
13. I recognize that there are ways other than mine.	
14. I accept people for who they are regardless of color, educational achievement, financial status, age, physical limitations, or gender.	
15. I don't mind apologizing if I have wronged or offended someone.	
16. I respect that others may have a different interpretation of personal space.	
17. I treat people differently than the biases and prejudices of members of my culture.	
18. I do not look down on people who do not speak English fluently or may have an accent.	
19. I understand that there are many ways to communicate.	
20. I use simple and common phrases when around older adults, those with physical limitations, or someone of a different culture who may not speak my language proficiently.	

Add up all of the numbers for a total score.

Total score: outstanding: 95–100; good: 85–94; average: 75–84; needs improvement: 74 or lower.

(This tool is intended for personal use only. It is designed to be performed as a personal self-assessment. No reliable test that measures stability, equivalence, and homogeneity has been done.)

EXHIBIT 3.7 Sample: History Form

Client: Personal, Social, and Family Information

Name _____

Date of birth _____

1. Today's date _____
2. Age _____
3. Gender: M or F. Is your answer to question number 3 based on a transgender sexual change? If no sexual change has taken place, skip questions 4 and 5.
4. Date of procedure _____
5. Type of procedure _____

Medications prescribed _____

_____

_____

Sexual orientation: heterosexual _____ gay/lesbian _____ other _____

Proficient in speaking English YES NO

Proficient in reading English YES NO

Ability to read lips YES NO

Preferred spoken language

_____

Most comfortable language when speaking

_____

Most comfortable language when reading

_____

Preferred greeting Mr. Mrs. Ms. First name

_____

Type of nonverbal communication used

_____

Eye contact

_____

Need of interpreter

_____

Relation to interpreter

_____

Quiet/use of silence

_____

Use and definition of time

_____

Use of any common signs (okay, pain, clapping)

_____

Use of comfort space

_____

Tactile use

_____

Use of cultural jargon or slang that may affect evaluation

_____

_____

Perception of pain

_____

EXHIBIT 3.7 Sample: History Form (*continued*)

Cultural _____ Ethnicity _____

Family role and function

_____

Work

_____

Leisure activities

_____

Friends _____ Others _____

Country of origin

_____

Country of birth

_____

Years in the United States

_____

Did you grow up in a city _____ town _____ suburb _____ rural _____

Ethnicity

_____

Major support group

_____

Dominant members of the family

_____

Decision-makers for the family

_____

Previous work history

_____

Present work history

_____

Education

_____

Describe the importance of religion

_____

Religious beliefs/practices

_____

Religious association

_____

Cultural/religious practices/restrictions

_____

Meaning and use of religious symbols

_____

Interaction with family/significant other—describe

_____

_____

Role of father _____Role of mother _____

Role of older sibling/siblings

_____

EXHIBIT 3.7 Sample: History Form (*continued*)

Grandparents' role

Number of dependents

Children/grandchildren

Are there any with physical limitations (deaf, mute, or blind)? Yes _____ No _____

If you answered yes to the above, please identify what type

Means of communication

Expectation from this visit _____

Food preferences _____

Beliefs on health promotion

Family history

Skin color/hair structure _____

Reason for Visit

Chief complaint

Perceived cause

Reasons for cause

Symptoms of illness

Onset and severity (pain scale)

Effects of illness on ADL

Fear of the unknown about illness

Treatment expectations and results

Beliefs/practices about illness

Health promotion beliefs and practice

Types of healing practices

Client's appearance

Common diseases and disorders

EXHIBIT 3.7 Sample: History Form (*continued*)

Beliefs and practices regarding traumatic events

Beliefs and practices for preventive health

Surgical history

Other medical history

Any additional information that may improve client care

ADL, activities of daily living.

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ADVANCED CARE PLANNING

A. The assessment allows a time to review the aged adult's preferences for medical care. The discussion should involve the client's preference for the trajectory of care (e.g., life-sustaining at all costs or prioritizing quality of life and functional independence) and identification of healthcare proxies and surrogate decision-makers. The time may also be used to prepare advance directive documents or provide a referral to services to assist with these tasks. Resources such Prepare for Your Care (www.prepareforyourcare.org) and the Stanford Letter Project (med.stanford.edu/letter.html) may be helpful to the aged adult when preparing for these discussions.

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CLIENT TEACHING GUIDE

SAFETY ISSUES: FALL PREVENTION

PROBLEM

A. More than one out of four people 65 years and older fall each year. A review of your home can help prevent falls. Complete a "My Falls-Free" Plan (Table 1) to help you and your caregiver identify problems and the need to have a referral to a specialist.

B. The greatest number of falls occur at night.

C. The most common times falls occur inside the home:

1. Toileting.
2. Walking to the bathroom.
3. Attempting to get out of bed.
4. Rolling out of bed.
5. Transferring from chairs.
6. Walking in the house without using assistive devices (walkers and canes).
7. Walking in hallways.

CAUSES You and your primary care provider should discuss your risk of falling. Most of the time it is a combination of factors that places you at risk for falls.

A. A previous fall makes you at the highest risk for another fall. Tell your healthcare provider if you have ever had any falls.

B. Medical problems that place you at risk for falls include arthritis, dizziness, blood pressure, diabetes, shortness of breath, heart problems, and having to go to the bathroom quickly.

C. Medicines, including over-the-counter medicines and herbals, may interact and cause problems with blood pressure, dizziness, and other side effects, which put you at risk for falls. Ask your health provider or pharmacist to review your medications.

D. Alcohol and use of recreational drugs also increase your risk for falls.

PREVENTION Review of your home to identify items that place you at risk is essential.

A. General home safety review:

1. Walk around each room to see if you have a clear path; move furniture, if needed.
2. Remove clutter.
3. Add side railings on both sides of stairs inside and outside the home.
4. Use nonslip rugs or even remove rugs to prevent tripping. Fix loose and torn carpeting.
5. Increase lighting by using higher watt bulbs and nightlights. Add an overhead light at the top and bottom of the stairway.
6. Wear shoes that fit your feet well and have solid, nonskid soles; wear shoes instead of slippers (and instead of going barefoot).
7. Coil or tape cords and wires next to walls so that you will not trip. Have an electrician put in another outlet.
8. Fix loose or uneven steps.

B. Bathroom safety review:

1. Add grab bars on the inside and outside of the shower and at least one by the toilet.
2. Add a nonslip bath mat in your tub or shower and one right outside the tub or shower.
3. Use a raised toilet seat.
4. Consider the need for a bedside commode.

C. Bedroom safety review:

1. Place a lamp close to the bed so that you do not have to reach it from the edge of the bed.
2. Use a nightlight.
3. Use comforter and blankets instead of long bedspreads that touch the floor.

D. Kitchen safety review:

1. Place bowls, plates, and spices on the lower level of your cabinets in order to get to them safely and easily.
2. Never use a chair instead of a step stool.

E. Other safety tips:

1. Discuss the need for a personal emergency response system.
2. Carry a cell phone in your pocket.
3. Use a shoulder bag, fanny pack, or a backpack purse to leave your hands free.
4. Keep emergency numbers near each phone and posted on the refrigerator.
5. Limit alcohol to one drink a day to help prevent falls.

(*continued*)

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CLIENT TEACHING GUIDE

SAFETY ISSUES: FALL PREVENTION (*CONTINUED*)

TREATMENT PLAN

A. Discuss the need for an eye examination.
B. Discuss the need for assistive devices such as canes and walkers.
C. Discuss the use of a Vial of Life to identify important information in case of an emergency (www.vialoflife.com/how_to_use_the_vial_of_life).
D. Walk two or three times a week.
E. Do balance and strengthening exercises at least three times a week.

TABLE 1 MY FALLS-FREE PLAN

Name: _____ **Date:** _____
As we grow older, gradual health changes and some medications can cause falls, but many falls can be prevented. Use this to learn what to do to stay active, independent, and falls-free.

Check "Yes" if you experience this (even if only sometimes)	No	Yes	What to do if you checked "Yes"
Have you had any falls in the last 6 months?			• Talk with your doctor(s) about your falls and/or concerns. • Show this checklist to your doctor(s) to help understand and treat your risks, and to protect yourself from falls.
Do you take four or more prescription or over-the-counter medications daily?			• Review your medications with your doctor(s) **and** your pharmacist at each visit, and with each new prescription. • Ask which of your medications can cause drowsiness, dizziness, or weakness as a side effect. • Talk with your doctor about anything that could be a medication side effect or interaction.
Do you have any difficulty walking or standing?			• Tell your doctor(s) if you have any pain, aching, soreness, stiffness, weakness, swelling, or numbness in your legs or feet—**don't ignore** these types of health problems. • Tell your doctor(s) about **any** difficulty walking, to discuss treatment. • Ask your doctor(s) if physical therapy or treatment by a medical specialist would be helpful for your problem.
Do you use a cane, walker, or crutches, or have to hold onto things when you walk?			• Ask your doctor for training from a physical therapist to learn what type of device is best for you and how to safely use it.
Do you have to use your arms to be able to stand up from a chair?			• Ask your doctor for a physical therapy referral to learn exercises to strengthen your leg muscles. • Exercise at least two or three times a week for 30 minutes.
Do you ever feel unsteady on your feet, weak, or dizzy?			• Tell your doctor and ask if treatment by a specialist or physical therapist would help improve your condition. • Review all of your medications with your doctor(s) or pharmacist if you notice **any** of these conditions.
Has it been more than 2 years since you had an eye exam?			• Schedule an eye exam every 2 years to protect your eyesight and your balance.
Has your hearing gotten worse with age, or do your family or friends say you have a hearing problem?			• Schedule a hearing test every 2 years. • If hearing aids are recommended, learn **how** to use them to help protect and restore your hearing, which helps improve and protect your balance.
Do you usually exercise less than 2 days a week (for 30 minutes total each of the days you exercise)?			• Ask your doctor(s) what types of exercise would be good for improving your strength and balance. • Find some activities that you enjoy and people to exercise with 2 or 3 days per week for 30 minutes.

(*continued*)

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CLIENT TEACHING GUIDE

SAFETY ISSUES: FALL PREVENTION (*CONTINUED*)

Name: _____ **Date:** _____
As we grow older, gradual health changes and some medications can cause falls, but many falls can be prevented. Use this to learn what to do to stay active, independent, and falls-free.

Check "Yes" if you experience this (even if only sometimes)	**No**	**Yes**	**What to do if you checked "Yes"**
Do you drink any alcohol daily?			• Limit your alcohol to one drink per day to avoid falls.
Do you have more than three chronic health conditions (such as heart or lung problems, diabetes, high blood pressure, arthritis, etc.)? Ask your doctor(s) if you are unsure.			• See your doctor(s) as often as recommended to keep your health in good condition. • Ask your doctor(s) what you should do to stay healthy and active with your health conditions. • Report any health changes that cause weakness or illness as soon as possible.

The more **Yes** answers you have, the greater your chance of having a fall. Be aware of what can cause falls, and take care of yourself to stay independent and falls-free!
Reviewed by: _____
This information is public domain from the Washington State Department of Health, Injury & Violence Prevention Program.

Source: Columbus Regional Hospital. (2017). Avoid the Risk of Falling. Retrieved from https://www.crh.org/healthy-tomorrow/healthy-tomorrow/2017/01/04/avoid-the-risk-of-falling.

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CLIENT TEACHING GUIDE

SAFETY ISSUES: MEDICATION SAFETY

PROBLEM Multiple problems may occur as a result of taking more than two medicines, including drug interactions and side effects. It is very important to ask questions and ask for written instructions to make sure that you understand your medicines.

CAUSES The causes of problems with medications include the following:

A. Not understanding what each prescription is and how to take it.
B. Seeing more than one doctor for different medical conditions.
C. Using more than one pharmacy.
D. Taking some prescription medications with herbal and over-the-counter (OTC) medicines.
E. Mixing other family members' medications with yours.
F. Forgetting if you have taken your medicine and taking it again.
G. Not having a system set up to take medications as prescribed.
H. Taking someone else's medications.
I. Mixing more than one drug in one prescription bottle.
J. Some medicines need to be tapered off instead of just stopping them.

PREVENTION

A. Questions to ask your provider and pharmacist for medicines include the following:
  1. What is the medication for?
  2. What are the side effects of this medicine?
  3. How and when do you take the medication?
  4. What should you do if you miss any medicine?
  5. Can the medicine be taken with food or should it be taken on an empty stomach?
  6. Are there any foods that should not be eaten while on the medicine?
  7. Can the pill be cut in half, crushed, or chewed?
  8. How long should it be taken?
  9. Should this medicine be stored in any special way, like keeping it in the refrigerator?
  10. Is there a generic medicine that costs less?
B. Make a list of all of your medications, including any prescriptions, vitamins, herbal products, and OTC products:
  1. Eye drops, creams, patches, and inhalers are medicines.
  2. OTC products that should be listed include medicines for your bowels, headaches, stomach, and sleep.
  3. Keep your medicine list up to date by adding new medicines, taking off medicines that are no longer taken, and updating your list if the medicine dose changes.
  4. Do not stop medicines just because you feel better.
C. Once or twice a year, take a bag of all of your medications into the office to have your healthcare provider review them.
D. Store medicine in one place instead of multiple medications in the kitchen cabinets/shelves, bedroom, bathroom, and drawers.
E. Lock up your medications in a toolbox to keep them safe and to prevent others from taking your medication. Keep all medication out of reach of children.
F. Do not share medicines with anyone.
G. Keep individual family medicines separated by using a different shelf or basket.
  1. If you need to use inhalers for asthma or other lung problems, use one color basket for fast-acting inhalers and another color basket for the long-acting inhaler medicine.
H. A weekly pill container will prevent you from taking extra doses; have someone help you set up your medicines.
I. Do not mix medicines in one bottle.
J. Medicine that comes in an amber container protects the medicine from light.
K. Before throwing out an old medicine bottle, scratch out or use a marker to make personal information unreadable.
L. Ask the pharmacist how to dispose of unused and old medicines.
M. Throw out outdated medications and those that are no longer taken. The Food and Drug Administration and the White House Office of National Drug Control Policy have developed guidelines for drug disposal.

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SAFETY ISSUES: MEDICATION SAFETY (*CONTINUED*)

1. Do not flush medications down the sink or toilet unless there are specific labels on the medication.
   a. Narcotic pain medicine may have instructions to flush it to reduce the danger of unintentional use or overdose.
   b. Fentanyl patches for pain should be folded in half and flushed down the toilet.

2. Use community drug-takeback programs to bring any unused drugs to a central location for proper drug disposal. Often there are designated disposal containers in police stations.

3. If the drug cannot be put down the drain/flushed or taken to a designated disposal, the following steps should be taken:
   a. Remove drugs from the original container and mix with kitty litter, coffee grounds, or other undesirable substances.
   b. Place the mixture in a sealable bag, empty can, or other container to prevent leaking.

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CHAPTER 4

CAREGIVER AND END-OF-LIFE ISSUES

Ann McQueen Blair

CAREGIVER SUPPORT ISSUES

A. It is estimated that 20% of the population in the United States will be classified as older adults by the year 2030. According to the Centers for Disease Control and Prevention, in 2018, 51.8% (129 million) of civilian, noninstitutionalized adults had been diagnosed with at least 1 of 10 selected chronic conditions. More specifically, 24.6% (61 million) of adults had one chronic condition and 27.2% (68 million) had ≥2 chronic conditions. Trends indicate that individuals living with chronic illness will increase as the population ages, with an estimated 50% of people 65 years of age or older having three or more chronic illnesses. These comorbid conditions are associated with increased utilization of healthcare services and risk of decreased quality of life (QOL), polypharmacy, adverse reactions, and exacerbations of illness, creating a cycle in which health may decline, care becomes more complex, and the individual can no longer live alone. Trends in healthcare, including shorter hospital stays, fewer geriatric primary care providers, and limited community resources, have negatively impacted the ability of the client to maintain living in the community without caregiver support. Therefore, along with the increase in older adults comes an increase in the number of caregivers for older adults. According to the National Alliance for Caregiving (2020), during the past year, 65.7 million Americans (or 29% of the U.S. adult population involving 31% of all U.S. households) served as family caregivers for an ill or disabled relative. The implication for healthcare providers is that the needs of the client and the caregiver should be met in order to provide optimal health services. The purpose of this section is to identify and define formal and informal caregivers (ICs), review the demographics of caregivers in the United States, examine common caregiver concerns, review assessment of caregiver health and well-being, and identify sources of support.

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FORMAL AND INFORMAL CAREGIVERS

A. Caregivers are categorized as formal or informal. Formal caregivers are those who are paid to provide the care to the recipient. Formal caregivers encompass a wide variety of providers and community resources, including primary and specialty medical providers, home health providers, home aides, home companions, social services, and long-term care facilities. It is a myth that most of the care of older adults occurs in long-term care, as most caregiving is provided by informal caregivers (ICs). ICs are family, friends, or volunteers who provide uncompensated care to persons with disease or disability. This care ranges from direct care with activities of daily living (ADL), instrumental activities of daily living (IADL), transportation, household chores, medication administration, and disease management. It is important for APRNs to consider that in the current culture of healthcare the responsibility for disease management has been transferred from healthcare professionals to clients and their ICs. Additionally, one of the most important predictors of successful disease management for elder adults is the well-being of the caregiver; therefore, the needs of the IC must be identified and included in the plan of care.

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INFORMAL CAREGIVER DEMOGRAPHICS

A. Informal caregivers (ICs) are a heterogeneous group of individuals who have a wide range of caregiving experiences, needs, and resources. The trend to remember is that caregivers and care recipients are getting older and that the demographics will change as the nation ages.

1. ICs provide care for an average total length of 4.6 years, although 31% of ICs reported providing care for more than 5 years.

2. The span of caregiving responsibility was 6 months to more than 10 years.

3. The average age of IC is 49.2 years and is increasing.

4. More recent estimates include the mean age of caregivers for clients who have chronic obstructive pulmonary disease (COPD), congestive heart failure (CHF), or cancer as 57 years.

5. The majority of ICs are females, and more than 50% of them are working outside of the home.

6. Almost three-fourths of ICs who worked outside of the home reported that caregiving influenced their ▶

The contributions of Carole K. H. Bartoo and Karla Schroeder to this chapter in prior editions are acknowledged here.

performance at their job, including juggling time at work to meet the demands of caregiving, turning down promotions, losing benefits, and early retirement.

7. Most ICs are married (70%), college-educated to some degree (62%), and one-fourth have trouble paying their bills.

8. The average number of hours per week spent on care provision is 20.4 hours, with a range of less than 8 hours to more than 40 hours.

9. Thirty-two percent of caregivers reported that they experienced a high burden of care, 17% reported a decline in their health, and 31% indicated that they experienced a high level of stress associated with caregiving.

10. ICs are non-Hispanic White (72%), African American (13%), Hispanic (2%), and Asian American (2%).

CARE RECIPIENT DEMOGRAPHICS

A. The average age of the care recipient is 69 and is increasing as the population of elders older than the age of 75 continues to increase.

B. Half of the care recipients live in their own home and need assistance with activities of daily living (ADL).

C. In the survey, over three-fourths of the informal caregivers reported that they desired more information from formal caregivers. The topics of interest include:

1. Home safety.

2. Balancing caregiving demands and personal needs.

3. Communication and working with healthcare professionals.

4. End-of-life (EOL) care.

5. Choosing formal caregiving services.

6. More basic information on how to care for their loved one's needs.

D. Interestingly, 30% of the survey recipients said they would turn to their medical provider for this additional information.

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INFORMAL CAREGIVER CONCERNS

A. Providing care to a loved one has many benefits. The majority of informal caregivers (ICs) report that they find caregiving meaningful and rewarding; in addition, many report low levels of depression and anxiety and high levels of spiritual well-being. However, this heterogeneous group is also diverse in responses to caregiving. Some ICs do not have a positive experience. Some researchers have found that optimal caregiving outcomes are dependent on the amount of physical, emotional, and mental strain experienced by the IC. The need for ICs is great, and ICs have shown a preference for getting information from their medical providers. Medical providers need to be aware of the potential concerns ICs may have. These concerns include work–life balance, financial concerns, medication management, and caregiver health.

WORK–LIFE BALANCE

A. Caregivers face a monumental task as they are often attempting to provide care to a loved one, understand the disease process and trajectory, manage the healthcare system, and maintain their own life roles. Additional caregiver roles may include social, occupational, and personal domains. Caregivers have work, family, and community obligations, self-care needs, and leisure activity preferences. All of these should be considered when assessing caregiver response to the caregiving role. Many caregivers have stated that their own healthcare is of a less priority than that of the care recipient. Caregiving was associated with increased risk of negative health behaviors. Hoffman and Wallace (2018) proposed that a rationale for the decreased participation in preventive health activities is the demand of providing care and decreased opportunity for health-promoting activities. Therefore, it is important that ICs find balance among the multiple demands not only of life but of caregiving responsibilities.

FINANCIAL IMPLICATIONS

A. The financial implications of caregiving affect both caregivers and their employers. Employers of caregivers have reported decreased productivity due to more time missed and interruptions in workflow. Caregivers report increased expenses, decreased resources, and decreased opportunity for advancement in the workplace.

1. Of forty percent of caregivers report having to use their savings to provide care needed by their loved one.

2. Forty percent of caregivers reported cutting back on discretionary spending, while 38% reported cutting back on necessary spending.

3. Nearly one-third of caregivers report that caregiving is too expensive. Expenses incurred by caregivers include lost time at work, decreased income, and cost of direct care, including transportation, daily needs such as food and supplies, and medical expenditures. The implications of such expenditures are an increased burden for the caregiver and fewer resources for caregiving in the community. Lai (2012) found that financial implications were the largest correlating factor for increased caregiving burden.

CAREGIVER HEALTH

A. Caregiver health is another concern that APRNs should consider when assisting ICs. Hoffman and Wallace (2018) found that caregivers engaged in 28% more serious emotional distress and unhealthy behaviors than their peer noncaregivers and participated in fewer health-promoting activities.

1. Negative health behaviors include cigarette smoking, drinking regular sodas, and eating fast foods. These findings suggest that the stress associated with caregiving may lead to negative health behaviors, and the demand of caregiving may decrease the opportunity for caregivers to attend to their own health needs.

2. Common health-related sequelae of caregiving include decreased sleep quality, fatigue, poor eating habits, and less exercise.

3. Caregivers are also at risk for decreased psychological well-being. Stress, depression, and social isolation are reported by many caregivers.

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PROVIDERS' ASSISTANCE WITH THE CAREGIVER

A. APRNs must actively solicit information about the health and well-being of caregivers, the caregiving requirements, and the burden associated with providing care. This conversation should be part of routine client encounters and it can be as easy as asking the question, "Are you a caregiver?"

1. Once the caregiving role has been identified, then APRNs can assess the positive and negative outcomes of caregiving. An integral understanding for the APRN to possess is that the caregiver and care recipient are a unit. The implications are that malfunction of either component in this unit can and likely will lead to poor outcomes.

2. Caregivers often report being inadequately prepared for the caregiving role. A particularly vulnerable time for this preparation is during transitions of care. There is a lack of structure and guidance from the medical community to the caregiver when care is being transitioned from a formal caregiving environment to the informal caregiving environment.

3. Caregivers are often engaged at the point of discharge. Therefore, caregivers are informed of a client's discharge from a facility without planning. Caregivers report frustration and lack of information in providing quality, often technical, care. The high rate of hospital readmission for older adults speaks to this lack of preparation for care provided postdischarge. Caregivers need information, support, and respite. The role of respite for the caregiver or the client needs to be presented when possible.

4. APRNs should seek to engage caregivers by asking about their frustrations, the caregiving demands, and their preparation for providing the needed care. Common caregiver frustrations include:

a. Lack of knowledge on how to access resources.
b. How to provide technical care.
c. How to respond to the care recipient's emotional needs.
d. How to anticipate future needs, including having an "emergency packet" of items to have on hand in case there are transitions of care.

5. The APRN will need to have knowledge of the particular client in order to adequately inform the caregiver. One goal of this assessment is to collaborate with the caregiver in order to respond to care recipient needs, train the caregiver on technical aspects of care, and provide anticipatory guidance for the trajectory of the illness or disease.

6. APRNs should also assess the well-being of the caregiver. There are several tools to assist in assessing the caregiver:

a. Caregivers should be asked about their basic needs, such as sleep, diet, healthcare, and emotional support.
b. Caregivers should be asked about their social support networks and if these social supports are adequate. This assessment can be as simple as asking if the caregiver is receiving adequate support or as complex as administering a measurement tool.
c. Caregivers should be assessed for quality of life (QOL), stress, and depression. QOL and caregiver burden measurement tools are widely available. It is important for APRNs to use this information to plan tailored interventions that will assist caregivers:

i. The five-item Geriatric Depression Scale (GDS) is a quick screening tool to use to evaluate satisfaction with life, boredom, helplessness, wanting to stay in the home instead of social interaction, and feelings of worthlessness. Each answer requires a "Yes/No" response. Further evaluation is recommended for a score of 2 or greater. A copy of the GDS assessment is available for five-item depression screener at sagelink.ca/health-care/hcp-depression/.

ii. An assessment tool such as the Patient Health Questionnaire (PHQ-9) can be used to further evaluate depression. The Likert-scale responses range from no symptoms at all to symptoms nearly every day. A copy of the PHQ-9 is available at www.apa.org/depression-guideline/patient-health-questionnaire.

1) A PHQ-9 total score of 1 to 4 indicates minimal depression.
2) A PHQ-9 total score of 5 to 9 indicates mild depression.
3) A PHQ-9 total score of 10 to 14 indicates moderate depression.
4) A PHQ-9 total score of 15 to 19 indicates moderately severe depression.
5) A PHQ-9 total score of 20 to 27 indicates severe depression.

PROVIDERS' RESPONSE TO CAREGIVER NEEDS

A. One strategy that may prove to be beneficial is for medical providers to be aware of assistive technology that will improve the provision of care.

B. Forty-five percent of informal caregivers (ICs), 45% used technology to facilitate care, including electronic mobility devices, safety alarms, personal electronic health records, and electronic communication with healthcare providers.

C. Another resource that healthcare providers need to identify and promote are healthcare policies and programs that lessen the burdens of providing care. Healthcare policies utilized by ICs included personal tax credit, a voucher program to pay minimum wage for care provided, and respite services. Availability and utilization of these resources vary by states.

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CAREGIVER RESOURCES

A. Caregiver resources are becoming more widely available. As Medicare and other reimbursement agencies continue to strive to reduce readmission rates and reduce the cost of care, more attention will be paid to successful interventions that improve the health and well-being of the caregiver and the recipient.

1. Interventions that have been most successful include efforts to address physical, emotional, spiritual, and social domains.

2. Potential community resources include the primary care location, respite services, home aide services, palliative care (PC), social services, and support groups. An emerging service is geriatric care coordination.

3. The primary care location is an often overlooked resource; however, this location is the ideal site for forming the collaborative connection that will serve the caregiver and care recipient. ▶

4. Respite services may be available through care alliances and organizations. Home aide services may be available to assist the care recipient with activities of daily living; many are utilized through local home health agencies.

5. PC is helpful when learning about how to manage symptoms.

6. Social services may be of assistance to determine if local programs such as Meals on Wheels, as well as financial or housing assistance, are appropriate.

7. Support groups are effective for sharing experiences, problem-solving, and building a community of support. There are also several caregiver informational sites accessed on the internet, including the Family Caregiver Alliance (www.caregiver.org) and the National Alliance for Caregiving (www.caregiving.org).

B. Knowing the impact of family and environment is not new to nursing. Nurses have long acknowledged the significant impact of holistic care that includes not only the client but those people and places important to the client. Research about caregiving has been ongoing for decades. The impact, toll, and benefit of caregiving are well-documented. What is changing is that reimbursement for care is dependent on quality measures, and these quality measures will make their way measuring the effectiveness of caregiving. APRNs are poised to expand their role to empowering caregivers by providing knowledge, facilitating respite, and supporting caregivers.

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ADVANCE DIRECTIVES

A. Decision-making is an integral concept in healthcare and has ethical roots. The ethical principle of client autonomy should be considered when thinking about the role and value of advance directives. Older adults may face a time in which they are unable to make both financial and healthcare decisions. This inability creates an ethical dilemma for family members and healthcare providers. Advance directives should give insight into what the elder would have chosen if they could still make decisions. The APRN will need to be familiar with state laws and facility regulations concerning advance directives. Families often do not understand that the state of a client's residence tethers the content and formalities of execution of advance directives. The National Hospice and Palliative Care Organization CaringInfo provides free advance directives and instructions for each state. The website link to download an individual state advance directive is www.caringinfo.org/i4a/pages/index.cfm?pageid=3289.

B. Advance directives are formal documents that enable older adults to make decisions about their future healthcare when they are no longer capable of making these decisions. The Patient Self-Determination Act of 1990 required that hospitals and other healthcare facilities provide clients with information on the purpose, execution, and documentation of an advance directive for all clients of the facility. Despite this act, many older adults do not have an advance directive. Barriers to completing an advance directive for older adults include lack of knowledge, fear of burdening family members, and desire to have the healthcare provider initiate the conversation about advance care planning. Barriers for healthcare providers include lack of time during an office visit and discomfort in initiating the conversation about advance care planning.

TYPES OF ADVANCE DIRECTIVES

A. Decision-making:

1. Decision-making includes both financial and healthcare determinations. **Adults are deemed capable of making decisions until they are proven to be incompetent. The determination of competency is a legal one and must be made by a judge.** Concerned parties may petition the court for a conservatorship (financial) or guardianship (healthcare) hearing. The court will then investigate; if the older adult is deemed unable to make decisions, then the court will appoint a conservator or a guardian to make decisions regarding finances, healthcare, or both. **It is important to know the difference between advance directives and conservatorship (guardianship); a primary difference is that the client determines who will be making decisions on their behalf when executing advance directives.** If there is no advance directive and the client is no longer capable of making decisions, then the court may have to decide who will make the decisions for the incapacitated adult.

B. Power of attorney:

1. Financial decisions include paying bills, making purchases, and selling, purchasing, or managing assets. **Healthcare decisions include treatment decisions, end-of-life (EOL) care, obtaining medical records, and many others. A competent adult has the option of creating a power of attorney (POA) or a durable POA for financial and healthcare decisions. The same person can be designated POA for both, or two separate designations can be chosen. For both the POA and the durable POA, the competent adult selects an agent who will make decisions.**

a. The POA is a collaboration between the competent adult and the agent.

i. The client retains the rights to make decisions on their own behalf. If the competent adult is subsequently determined to be incompetent, then the POA is often no longer valid and operational.

ii. If the competent adult created a durable POA and is subsequently determined to be incompetent, then the durable POA is still valid and operational. It is important to note that an agent named in a POA, or durable POA, specifically for healthcare or financial decisions and not both may be limited in their ability to make some decisions. For example, an agent of a financial durable POA may not be able to make healthcare decisions based on this document alone. A specific document for healthcare, or financial, decisions must be executed to name an agent for making decisions. These documents may not be interchangeable.

iii. It is also important to remember that the rights granted to an agent by the POA or durable POA end when the incapacitated adult dies. Therefore, an agent cannot make decisions about assets or obtain medical records for a deceased person. These rights are granted to the person, or agent, named as the executor of the person's estate. ▶

C. Living will:

1. A living will is a document created by a competent adult that specifies what treatment or medical decisions are preferred in the event that the adult becomes unable to make decisions.

a. This document is specific and will likely include preferences for resuscitation, intravenous hydration and nutrition, ventilator support, pain management, and organ donation.

b. The benefit of the document is to assist family members and medical providers in making very difficult decisions by making the wishes of the competent adult known to all parties. However, this document is not always recognized as a legally binding decision.

c. A living will does not name an agent to make healthcare decisions. A healthcare POA may be needed in addition to the living will if the living will is not recognized as a legally binding document.

d. The value of a living will should not be understated. The living will acts as a guide to assist decision-makers when making very challenging and emotionally charged decisions on behalf of the loved one.

D. Do-not-resuscitate orders:

1. A do-not-resuscitate (DNR) order is written by a medical provider in observance of the client's wishes for EOL care. A competent adult, or the agent for an incompetent adult, can make this decision and communicate it to the medical provider. The order is written by the medical provider and communicated to staff to alert all that the person does not wish to have CPR started should their heart or breathing stop. **In the absence of a DNR order, healthcare personnel may be required to start resuscitation efforts. A living will, in which a competent adult has expressed wishes against resuscitation, does not take the place of a written DNR order.**

E. MOST and POLST forms:

1. The Medical Orders for Scope of Treatment (MOST) and the Provider Orders for Life-Sustaining Treatment (POLST) forms are written orders that direct medical care based on the client's wishes. These forms are different from a living will because the MOST form is signed by the medical provider and acts as orders to direct client care. Items included on MOST/POLST forms include CPR, antibiotics, fluid, nutrition, comfort measures, and transfer to hospital if indicated. These forms are specific, created between the provider and the client, and signed by both parties. The benefit of the MOST/POLST forms is that the actions and decisions that the client and their agent have agreed upon are formalized as medical orders.

2. The National POLST Paradigm website with state legislative information is located at polst.org/programs-in-your-state.

3. The MOST and POLST forms are intended to supplement an advance directive.

The MOST and POLST forms should be updated at each transition of care.

RESOURCES FOR CREATING AN ADVANCE DIRECTIVE

A. Once a competent adult has made the decision to execute an advance directive, they may need assistance in creating the documents. Advance directives can be created without legal representation. There are many online resources available, including AARP, CaringInfo.org, and state bar associations or agencies for aging. It is important for competent adults to have a conversation with family members or others who may serve as agents to ensure that the agent is willing and able to execute the decisions of the competent adult. The optimal time for such discussions is before a medical condition threatens to incapacitate a person; however, any discussion is better than none. APRNs can assist their clients by initiating the conversation, answering questions, and providing resources.

OVERCOMING BARRIERS

A. Advance directive barriers can be characterized as barriers to creation of an advance directive and barriers to implementation of the advance directive. Spoelhof and Elliott (2012) described completion barriers as client and provider limitations.

1. Clients may have lack of interest, lack of knowledge, fear of burdening family members, and social isolation. These barriers are aptly addressed by initiating conversations at an early stage, before an illness has occurred. Clients can be approached and asked about their wishes for care should they become incapacitated during a wellness visit, or when discussing screenings for illnesses. This nonthreatening time is well-suited for information sharing.

2. Provider barriers include lack of time, lack of reimbursement, and discomfort with the topic. Hoffman et al. (2012) found that clients desire to have this discussion with healthcare providers. In addition, healthcare providers report greater satisfaction when care is congruent with client wishes.

3. Barriers to implementation include nonspecific language in the advance directive, agent issues, and advance directive accessibility:

a. The language in an advance directive should be specific and include the client's wishes for CPR, fluid, and nutrition.

b. An advance directive is also more useful if it is clear in what is meant by incapacitation, as language can make this a difficult determination. For example, it is much clearer to state that the advance directive takes effect in the event of cardiac arrest rather than in the event of no hope for recovery.

c. Agents will need to know that they have been named as healthcare proxy and to know the wishes of the client.

d. Advance directives, once completed, need to be signed by witnesses, and in some states need to be notarized. It should be easily located: the client, agent, and medical providers should have a copy of all documents. The client should keep copies of their advance directives in an easily obtainable location at their residence.

4. There are strategies to help clients and medical providers in completing the process of discussing, creating, and executing advance directives. Interactive, or group, interventions have been more successful than individual encounters for educating on the value and role of advance directives. Having ongoing discussions about the goals of care once a terminal or progressive illness has occurred is another effective means of educating clients and their healthcare agents. Another strategy is the use of advance directive facilitators to introduce and educate ▶

communities. A Values History form may be helpful in assisting the client to consider their own feelings and initiate conversations with potential healthcare agents. The Values History form was created by the Institute of Public Law and is available online. It is not a substitute for a living will or POA; however, it is a complementary document that examines one's personal beliefs in such a way as to help surrogate decision-makers during challenging and emotional EOL decision-making.

SPECIAL CONSIDERATIONS: TIMING THE ADVANCED CARE PLANNING DISCUSSION

A. Billings and Bernacki (2014) reviewed evidence about the well-timed and executed advanced planning discussion. They found the following as important elements:

1. Determining the client's understanding of their medical condition and, when gaps in understanding are found, helping the client to fill gaps and be able to verbalize their understanding of the condition.

2. Helping a client understand prognosis and clinical options, including an awareness of the trade-offs involved in choices to undergo or forego therapies.

3. Determining/discussing a client's values and goals, and using these to make choices about specific methods of life-sustaining care.

4. Involving, when appropriate, a clinician to make a recommendation based on the client's values, goals, and preferences.

5. Allowing adequate time for reflection, discussion with family, and further consultation regarding decisions made in an advanced planning discussion.

6. Creating careful documentation, conveying decisions to the family, and making decisions to other healthcare practitioners.

7. Revisiting choices regularly, especially as the medical situation changes, on hospital admission or institutional transfer.

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PALLIATIVE CARE

A. In the past decade, palliative care (PC) has been in increasing demand. According to a 2010 survey, the estimated gap of available physician palliative or hospice specialists ranged between 6,000 and 18,000 physicians. This creates a need that APRNs will increasing be asked to fulfill.

B. The World Health Organization (WHO) defines PC as an approach that improves the quality of life (QOL) of clients and their families facing the problems associated with life-threatening illness through the prevention and relief of suffering by means of early identification and impeccable assessment and treatment of pain and other problems—physical, psychosocial, and spiritual. PC can be provided along with curative treatment. PC is commonly used in the treatment of cancers, but increasingly it is seen as an important service for other serious, life-limiting medical diagnoses, including renal failure, advanced chronic obstructive pulmonary disease (COPD) and congestive heart failure (CHF), and even dementia. The National Institute on Aging (www.nia.nih.gov/health/what-are-palliative-care-and-hospice-care) encourages care providers to refer older clients for a palliative consult, ideally from the point of diagnosis. If a client's disease has progressed and there are multiple hospitalizations in a year, a PC conversation is warranted and strongly encouraged. Important elements to consider in the discussion include the following:

1. PC may be provided concurrently with curative care.

2. The service can be useful in helping clients and their caregivers understand their choices for medical treatment late in life.

3. Goals of care can be discussed with greater thought and decisions can be disseminated to other key care providers at a time when healthcare needs are not urgent.

4. The topic of end-of-life takes time to be absorbed for both individuals and families and can become more "normalized" in the setting of future planning and discussion.

5. There have been no studies that have shown a decrease in survival from the palliative approach.

6. Medicare, Medicaid, and private insurance policies may cover PC fully or partially. Veterans may be eligible for PC through the Department of Veterans Affairs.

C. Research has shown PC can improve outcomes including:

1. Advance care planning.

2. Greater client and caregiver satisfaction with care.

3. Lower healthcare utilization.

D. Topics that may potentially be improved through quality PC include:

1. Preferred site of death.

2. Client mood.

3. Healthcare expenditures.

4. Caregiver QOL, mood, and burden.

5. Education regarding burdens versus benefits of treatment options.

6. A bridge to facilitate the start of hospice care when the client and their family are ready.

E. Benefits of PC:

1. Anyone with a serious illness or terminal illness can receive PC.

2. No requirement to waive traditional Medicare benefits so the client may pursue continued rehabilitation services.

3. More robust summary of current client status and prognosis.

4. Robust explanation of advance directive creation and updated Medical Orders for Scope of Treatment (MOST)/Provider Orders for Life-Sustaining Treatment (POLST) forms reviewed with family. In addition, the shift of emphasis from length of life to QOL may help in reducing unnecessary testing, reducing polypharmacy, and reducing complexity of care efforts. Coordination of care has the potential to improve through discussion of centralizing care through just one or two providers, reducing the number of specialists involved in care.

F. Timing the PC referral includes the evaluation of the client's risk of mortality. Multiple calculators are available through the ePrognosis website and are divided according to whether the client resides in the community or a nursing home, is hospitalized, or is an outpatient with advanced cancer. Other criteria include age, gender, renal function, ability to perform activities of daily living, and so forth. The ePrognosis website is located at eprognosis.ucsf.edu/calculators.php.

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SPECIAL CONSIDERATIONS: HOSPICE CARE

A. Currently, most hospice clients have their costs covered through the Medicare Hospice Benefits. Ross, Sanchez-Reilly, and Healy at the University of Texas Health Science Center at San Antonio developed the Hospice Criteria Card, which provides a summary of care under Medicare eligibility (Appendix 4.1).

1. Criteria for eligibility for hospice under Medicare include the following:

a. Entitled to Part A of Medicare (Hospital Insurance).

b. Certification must specify that the individual's prognosis is for a life expectancy of 6 months or less if the terminal illness runs its normal course in accordance with the Code of Federal Regulation requirement of §418.22—Certification of terminal illness. Eligibility requires the Medicare recipient to accept palliative care instead of curative care, and the client signs a statement choosing hospice care instead of other Medicare-covered treatment for terminal illness and related conditions.

2. Duration of hospice care coverage:

a. An initial 90-day period.

b. A subsequent 90-day period, followed by if needed.

c. An unlimited number of subsequent 60-day periods.

3. Document of a hospice face-to-face (FTF) encounter by a hospice physician or a hospice nurse practitioner.

4. The Medicare Hospice Benefits has four levels of care:

a. Routine hospice care (RHC) is the most common level of hospice and provides core services of the hospice interdisciplinary team provided at the client's place of residence.

b. Continuous home care (CHC) is provided for between 8 and 24 hours to manage pain and other acute medical symptoms and is intended to maintain the terminally ill client at home during a pain or symptom crisis. Greater than 50% of care must be primarily provided by a licensed practical nurse (LPN) or registered nurse (RN). Hospice aide or homemaker service can also be used to cover the needs.

c. General inpatient care (GIP) provides for pain control and other acute symptom management, such as respiratory distress, and for imminent death when the family cannot cope at home.

d. Inpatient respite care (IRC) provides temporary relief to the client's primary caregiver. Respite care can be provided in a hospital, hospice facility, or a long-term care facility.

5. The Hospice Criteria Card outlines the eligibility criteria for a general, nonspecific, terminal illness, cancer, dementia, heart disease, HIV/AIDS, liver disease, pulmonary disease, neurologic disease, renal failure, and stroke or coma.

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KEY FACTORS OF CARE IN THE ELDERLY CLIENT AT THE END OF LIFE

A. Clear explanation of the plan of care, client and family wishes, and provision of emotional support are all necessary aspects of care in the elderly client. Many times, adult children caring for parents are also taking care of their long-time partners. This adds a level of personal stress to the already emotionally burdensome situation. The following are foundational points to consider when caring for an elderly client and family at the end of life (EOL).

PHYSICAL COMFORT

A. Proper pain and symptom management: Many times clients are afraid to report pain or other symptoms. Clinicians are afraid to treat elderly clients and to overmedicate; thus, clients do not get adequate pain and symptom management.

B. Decreasing mobility, physical discomfort, and pain may cause difficulty with sitting, standing, and walking. The client may be embarrassed to ask for general assistance in completing activities of daily living (ADL). The more physical mobility decreases, the fewer tasks a person is able to complete independently, and the more the person is dependent on others.

DECISION-MAKING

A. Self-determination, also known as autonomy, respects the moral and legal rights of persons with decisional capacity to determine what will be done with their own person.

B. Respect of wishes: Clinicians may have difficulty respecting the wishes of the client when the family has other wishes.

C. Involvement of family/caregiver: Involve the client, family, and caregiver all together in the decision-making process. It helps for the family and caregivers to hear the client's requests, wishes, and perspective. Many times if the family and caregivers understand and know what the client wants, the decision-making process is much easier for all involved to complete and accept.

D. Clear documentation of goals of care: Guide the client and family through the goals of the care conversation. Clearly document and communicate the goals to the interdisciplinary group, especially consistency of care, and ensure the client and their family are supported in the decisions that have been reached.

EDUCATION

A. Discuss progression of the disease.

B. Explain the reasons for medication use, risk versus benefit, and side effects.

C. Give treatment options: Provide an honest discussion of the risk versus benefit for each treatment option.

D. Identify resources available: Utilize the social worker and case manager on the interdisciplinary team (IDT). There are many community resources available for clients. Understand a client or their family may need resources for food, medication, housing, and caregiver assistance.

E. Keep the education, goals, and plan centered on the client and on the family's goals of care. This centering is very challenging but necessary to build a trusting relationship and validate the individual experience.

EMOTIONAL SUPPORT

A. Understanding relationship dynamics.

B. Social isolation due to the effects of their disease process.

C. Caregiver fatigue.

D. Financial burden.

E. Loss of identity.

INTERDISCIPLINARY COLLABORATIVE PRACTICE

A. The palliative care (PC) IDT may include the clinician, nurse, aide, pharmacist, physical therapist, massage therapist, and nutritionist working as a complementary unit. The IDT works together to address the physical, social, spiritual, and psychological symptoms of clients with serious or life-threatening illness and their families' issues. IDT groups work to improve physical symptoms such as pain, nausea/vomiting, decreased mobility, incontinence, weakness, shortness of breath (SOB), change in body image, and other distressful symptoms. The goal is always focused on improving client and family quality of life (QOL).

B. The IDT team's initial and regular reassessment of psychological status is imperative, along with continued evolution of treatment methods. The IDT team works collaboratively with psychological and psychiatric team members to address depression, anxiety, delirium, and cognitive impairment as they directly impact clients with serious or life-threatening illness. Recognition of the need for continued social assessment and support for the PC client and family includes:

1. Initiating support services in a timely manner.
2. Involving case management in assisting with availability of community services, medication issues, financial concerns, or placement concerns.
3. Involving social workers in assisting with availability of community services, counseling, emotional support, spiritual support, and identifying barriers to care.
4. Utilizing volunteers to provide social interaction and caregiver relief.
5. Recognizing the cultural implications of care are important aspects of the client and family IDT assessment. Each individual has different cultural implications related to their specific care. Components of the cultural assessment include:
    a. Birthplace: What is the person's place of birth and what implication might this have on the client's care?
    b. Ethnic identity.
    c. Community: How does the client relate to their community?
    d. Decision-making: Who is the decision-maker for the individual: self, spouse, another person?
    e. Language of communication: Does language create a barrier? Consider things like eye contact, gender, position, and body language.
    f. Religion or spirituality: How do religion and spirituality specifically affect the individual's plan of care?
    g. Food preferences, prohibitions, or allergies.
    h. Economic situation: Can clients afford medications, food, and caregivers? Financial crisis is a major concern at EOL in the elderly population, of whom many have a fixed income.
    i. Health beliefs and practices regarding death, grief, pain, traditional therapies, care of the body, organ donation, and caregiver specifications.
        i. Does the client believe painful suffering is required?
        ii. Does the female client require a female clinician?
        iii. Is the client "refusing" an amputation of a part of the body (e.g., gangrenous toe) because the body has to remain intact in their culture?
6. Realizing spirituality does not necessarily focus strictly on religion or religious practices but rather promotes an individual's sense of well-being. Collaborative assessment for spiritual distress is completed by the IDT team. Symptoms of spiritual distress include:
    a. Feelings of abandonment.
    b. Anger.
    c. Feelings of betrayal or mistrust.
    d. Despair.
    e. Fear.
    f. Guilt.
    g. Regret.
    h. Sorrow or remorse.
    i. Depression.

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7. Enlisting a chaplain as an invaluable resource to help the client focus on spiritual aspects of care, provide support, and allow for a neutral conversation and reminiscence through life's journey, as well as understanding client preference of no chaplain involvement.

PHYSICAL ASPECTS OF CARE

PAIN MANAGEMENT

A. Pain is the most common symptom associated with palliative care (PC). Key concepts of pain management include the subjective nature of pain, an understanding that the symptom may not solely be physical in nature, and knowledge that pain is an experience that greatly affects the whole family unit.

B. The International Association for the Study of Pain (IASP) defines pain as a change to the nervous system and is also reflective of the individual's past pain experiences, making the pain an emotional as well as a physical experience. "Pain is an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage" (IASP, 2021). At the end of life (EOL), many individuals have difficulty or are unable to communicate their pain. This occurs for several different reasons, such as delirium, dementia, aphasia, motor weakness, language barriers, and other factors. Clients who cannot verbally express pain may still be having pain and still require treatment. Often these individuals are either not treated or are inadequately managed for pain.

C. The older adult is at risk for inappropriate pain management. It is estimated that between 25% and 45% of older individuals who live in communities do not receive adequate pain management. Research presents several reasons elderly individuals continue to have untreated, undertreated, or unrecognized pain, including an atypical presentation, failure to report, chronic pain, cognitive impairments, lack of clinician knowledge, and fear.

D. Not all pain is treated the same. In order to treat pain appropriately, one must first understand the different types of pain. What follows are different types of pain, with general definitions, and how clients typically describe them.

1. Acute pain:
   a. Identified event/acute injury.
   b. Clinically described as pain lasting less than 1 month.
2. Chronic pain:
   a. The cause of chronic pain may not be easily identified.
   b. Pain persists longer than 1 month.
3. Acute on chronic:
   a. Some pain syndromes consist of chronic pain with acute exacerbations or pain crisis.
4. Nociceptive:
   a. Usually related to damage to bones, soft tissues, or internal organs.
   b. Somatic pain: aching, throbbing.
   c. Visceral pain: squeezing, cramping.
5. Neuropathic:
   a. Usually related to damage of the nervous system.
   b. Complaints of burning, tingling, shooting pain.

E. Substance abuse issues: 70% to 90% of clients with advanced disease experience pain. The American Pain Society (APS) also reports individuals with a pain score of greater than or equal to a "4" on a standard pain scale. This report has great implications for quality of life (QOL). Assessment of pain is imperative. Pain assessment begins with an in-depth history:

1. History:
   a. Identify which drugs the client has already tried, explain whether these were effective, and state what adverse effect resulted from use.
   b. Identify what medication was prescribed and if it is what dose the client is actually taking. Compare the client's information with the family's information. Discrepancies do not always mean noncompliance; there may be a lack of understanding of the medication, financial reasons, fear, or a problem with memory.
   c. Ask the client about over-the-counter (OTC) medications, recreational drugs, alcohol, and herbal products.
   d. Involve the family in conversations. They may have additional or different views/information than that of the client.
   e. Identify what types of cultural influences on pain exist.
   f. Identify the type of pain.
2. Physical assessment of pain:
   a. Identify specific site of pain if possible.
   b. Characteristics of pain: Ask client to describe pain.
   c. Onset: When did the pain begin?
   d. Duration and frequency: How long does it last; how often does the pain occur?
   e. Exacerbating factors: What makes the pain worse?
   f. Associated factors: What symptoms occur with the pain? Examples are anxiety, fear, nausea.
   g. Alleviating factors: What makes the pain better? Examples are rest, medication, relaxation, massage.
   h. Emotional state: Does the client feel frustrated, anxious, fearful, or depressed?
      i. Existential distress: Does the client have spiritual distress? Does the client need to rectify a relationship with a family member?
   i. Nonverbal pain cues:
      i. Vocal complaints: sighs, gasps, moans, groans, cries.
      ii. Facial: grimaces/winces, furrowed brow, narrowed eyes, clenched teeth, tightened lips, jaw dropped or clenched, distorted expressions.
      iii. Restlessness: constant or intermittent shifting of positions, changes in behavior, combativeness, constant hand motion.
   j. Physiologic cues: tachycardia, tachypnea, hypertension, diaphoresis, insomnia, hyperreflexia, nausea, diarrhea, dilated pupils, anorexia, vasoconstriction, cool extremities.
   k. Therapeutic intervention: Treatment of perceived pain is considered a therapeutic intervention. Treat pain before a procedure is initiated; this could be something as general as turning/repositioning or bathing at EOL. Instruct the family or other caregivers about the importance of proper pain management regarding perceived pain.
3. Pharmacology: **Pharmacologic interventions must be tailored to the individual and use the rule "start low and go slow":**
   a. Acetaminophen:
      i. Safest for long-term use in the elderly.
      ii. Good for mild to moderate pain.

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iii. May be used in conjunction with opioids: caution that some medications already contain acetaminophen.
iv. Avoid in clients with renal or hepatic insufficiency or alcohol dependence.
v. Maximum daily dose 3000 to 4000 mg in 24 hours.

b. Nonsteroidal anti-inflammatory drugs (NSAIDs):
i. Can be initial therapy for mild to moderate pain.
ii. Adjunct for opioids and nonopioids; when used concurrently, lower doses of opioid may be used.
iii. Useful for treatment of nociceptive pain.
iv. Should be taken in conjunction with food only to prevent risk of ulcer formation.
v. Increased risk of gastrointestinal ulceration, renal dysfunction, and impaired platelet aggregation in long-term use.
vi. Use with caution in clients with history of ulcer disease.
vii. NSAIDs are on the American Geriatrics Society (AGS) Beers criteria for potentially inappropriate medications used in the older adult population (refer to Appendix C, Tables C.1, C.2, and C.4).

c. Opioids:
i. Used for moderate to severe pain.
ii. Begin with morphine, hydromorphone, or oxycodone as these medications are easier to titrate appropriately and have fewer side effects.
iii. Morphine:
1) Clients should be carefully monitored for signs and symptoms of sedation and confusion.
2) Labs should be closely monitored for renal function.
3) Morphine should not be used in clients with renal impairment.
4) Signs of morphine metabolite buildup include sedation, confusion, and respiratory depression. These signs will appear after a few days of therapy; they are not immediate signs. If the signs of metabolite buildup appear, the opioid should be changed.
iv. Hydromorphone:
1) Metabolite buildup leads to myoclonus, hyperalgesia, and seizures.
2) Metabolite buildup is most prevalent in clients with renal failure.
3) Hydromorphone is 1:3 times more potent than morphine.
v. Oxycodone:
1) Is a synthetic opioid.
2) Long-acting and immediate-release preparations are available.
3) Medication is expensive and may be cost prohibitive for some clients.
4) Is 1.5 times more potent than morphine.
vi. Fentanyl:
1) Available in transdermal form, which is useful when clients cannot swallow or have issues with intractable nausea/vomiting.
2) Fever, cachexia, obesity, and ascites will significantly affect absorption of the medication.
3) **Fentanyl is approximately 80 times more potent than morphine. Do not use fentanyl on opioid-naïve clients.**
4) Therapy must be monitored closely, and clear education must be provided to clients and their family.
5) Therapeutic effects do not begin until 12 hours after initiation.
6) When using long-acting transdermal fentanyl, ensure the client has adequate medication and plan for breakthrough pain medication.

4. Nonpharmacologic interventions for pain:
a. When determining a plan of care for pain management, both pharmacologic and nonpharmacologic interventions should be incorporated. The plan of care should be tailored to meet the needs of the client and their family. Nonpharmacologic interventions include:
i. Massage: This can be completed by a family member or a caregiver. This can increase the therapeutic relationship and provide a sense of control for the family.
ii. Acupuncture.
iii. Tai chi.
iv. Distraction: This can be anything that distracts: reading, television something that is enjoyed.
v. Relaxation: Teaching deep-breathing techniques can be very useful for both the client and their family and can be completed together.
vi. Music therapy.
vii. Guided imagery/meditation.

5. Diagnostic testing:
a. Always evaluate whether tests should be done based on the client and family goals.
b. Laboratory data, including complete blood count (CBC) with differential, complete metabolic profile (CMP), and urinalysis labs, are useful as a baseline evaluation.
i. With narcotic use, renal function should be evaluated initially and ongoing.
c. Radiologic testing as indicated only.

6. Differential diagnoses for pain:
a. Infection.
b. Depression.
c. Anxiety.
d. Constipation.

7. Plan:
a. The National Comprehensive Cancer Network (NCCN) has outlined the following goals when assessing and managing pain:
i. Pain will be controlled at an acceptable level for both the client and the family.
ii. Distress caused by pain will be decreased for both the client and the family.
iii. The client will have a sense of control.
iv. QOL will be optimized for both the client and the family.
v. An interdisciplinary care team can be utilized.
vi. Assessment is regular and ongoing.
vii. Identify and address barriers to effective pain management.
viii. For controlled substances, implement a risk management plan.
ix. Refer the client to providers with specialized skills (interdisciplinary team):
1) Primary care.
2) Pain management.
3) Pharmacy for medication management.

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4) Physical therapy to promote improved QOL if applicable to goals of care.
5) Occupational therapy to promote improved QOL if applicable to goals of care.
6) Chaplain for existential pain.

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SYMPTOM MANAGEMENT

ANXIETY

A. Anxiety is a common symptom and occurs in approximately 70% of individuals in hospice. It is often a predisposing factor to another symptom, such as pain or shortness of breath (SOB).

1. Causes of anxiety:
a. Fear.
b. Pain: current or unresolved.
c. SOB.
d. Past experiences.
e. Situation.
f. Preexisting anxiety disorder.
g. Delirium.
h. Anticipatory grief.
i. Advanced lung disease and periods of hypoxia.
j. Fear of death.
k. Concerns about religious beliefs, spiritual issues, and existential matters.

2. Clinical characteristics:
a. Pathologic anxiety:
i. A "life of its own."
ii. An intensity that exceeds an individual's ability to cope.
iii. Dread or intense worry.
iv. Tension, jitteriness, or restlessness.
v. Treatment nonadherence, social withdrawal, and avoidance.
vi. Inability to cope or relax.
vii. Symptoms complicated and often exacerbated by insomnia, depression, fatigue, gastrointestinal upset, dyspnea, pain, and dysphagia.

3. Assessment:
a. Begin with a complete history. Identify cause of anxiety if possible:
i. Understand client's disease process and progression. Remember anxiety is often complicated by another symptom. Identify disease processes in which symptoms may be present that may produce anxiety: congestive heart failure (CHF), chronic obstructive pulmonary disease (COPD), cancer, and anemia.
ii. Does the client have a history of psychological issues?
iii. Identify keywords during the conversation, such as fear, concern, scared, and worried.
iv. Utilize tools to help assess anxiety, including the four-item Patient Health Questionnaire for Depression and Anxiety (PHQ-4) available at www.oregonpainguidance.org/app/content/uploads/2016/05/PHQ-4.pdf or the Hospital Anxiety and Depression Scale (HADS) at kytinnitustreatment.com/wp-content/uploads/2015/06/HADS-2014.pdf.
v. Be aware that medications such as albuterol and steroids increase anxiety.
vi. Does the client have new symptoms?
vii. What is the client's knowledge of disease process and progression?
viii. Identify the client's previous healthcare and illness experiences, both positive and negative.
ix. Evaluate and understand if there are any patterns of the client's anxiety to determine if there is a cause and effect.
x. Identify the underlying cause of anxiety:
1) Physical symptoms: inability to sit still, jitteriness, depression, restlessness.
2) Emotional symptoms: edginess, feelings of impending doom, terror.
3) Cognitive difficulties: apprehension, dread, fear, obsession, uncertainty, worry.
4) Behavioral problems: avoidance, compulsions, psychomotor agitation, and potential belief of physical suffering caused from client's past behavior.
5) Autonomic symptoms: diaphoresis, diarrhea, nausea, dizziness, tachycardia, or tachypnea.

4. Diagnostic/laboratory tests:
a. Electrolytes, blood cell counts, relevant hormones, and toxicology as indicated.

5. Always evaluate diagnostic testing per the client's and family plan and goals of care.

6. Treatment is based on foundation of the anxiety.

7. Nonpharmacologic treatment is always the first-line treatment for anxiety:
a. Assess and monitor for anxiety.
b. Offer emotional support.
c. Reassure when appropriate.
d. Assess treatment response and side effects frequently.
e. Provide maximum resolution of anxiety as a goal.
f. Educate the client and their family on anxiety and treatments.
g. Provide cognitive behavioral therapy, relaxation, and distraction.
h. Encourage chaplaincy if there are spiritual and existential concerns.
i. Identify the underlying cause, if possible.

8. Pharmacologic treatment for anxiety: Consider the individual client when treating anxiety with medications using the rule "start low and go slow."
a. Lorazepam (Ativan) 0.25 to 0.5 mg two to three times a day scheduled or as needed. Maximum daily dose is 10 mg/d. May produce paradoxical agitation in clients who are elderly, demented, or brain-injured. Useful to try a small test dose first to see how the client will react to the medication. Ativan is short-acting and produces no active metabolites. It is best absorbed when given parenterally.
b. Haloperidol (Haldol) 0.5 to 1.0 mg two to three times a day scheduled or as needed. Useful if client is anxious and is unable to tolerate benzodiazepines. It may produce extrapyramidal symptoms, including akathisia. Consider dosing carefully for cachectic weight. ▶

c. Alprazolam (Xanax) 0.25 mg BID scheduled or as needed. The maximum daily dose of 6 mg/d. The extended-release alprazolam (Xanax XR) dose is 0.5 to 1.0 mg BID.

9. Plan:
a. Complete a detailed history and physical examination.
b. Discern if the cause of anxiety is psychological or physiological.
c. If fear is the cause, provide education and reassurance. Do not ignore. Validate and address the concern so that the client can directly deal with the fear.
d. Treat the underlying illness or symptom.
e. Control both the underlying symptom and the anxiety.
f. Do not assume that anxiety is inevitable and unavoidable.
g. Educate both the client and the family.
h. Ensure that the goals of the client and the family are discussed, clear, and followed.
i. Assess and reassess.

10. Differential diagnoses:
a. Depression.
b. Confusion.
c. Delirium.
d. Dementia.

11. Potential complications:
a. Difficulty making decisions.
b. Confusion.
c. Inability to intake information.
d. Agitation.
e. Difficulty controlling symptoms.

DELIRIUM

A. Delirium can be classified in two different ways: either as hyperactive or agitated delirium, or hypoactive delirium. Delirium is an acute change in the level of arousal. Accompanying characteristics include altered sleep and wake cycles, mumbling speech, memory and attention disturbances, perceptual disturbances often with delusions, and hallucinations:

1. Clinical characteristics:
a. Clouding of consciousness.
b. Altered ability to maintain attention.
c. Impairment of cognitive function.
d. Daytime sleepiness and nighttime agitation or restlessness.
e. Fluctuation of symptoms by the day or hour.

2. A careful review of the client's drug list should be done to identify potentially reversible causes of psychosis and agitation.

3. Physical examination should be performed to evaluate delirium/differentials that may cause delirium that are treatable. Treatment should be considered comfort-related actions.

4. Delirium assessment tool for end of life (EOL): **CHIMBOP:**
a. **C**: Constipation.
b. **H**: Hypovolemia, hypoglycemia.
c. **I**: Infection.
d. **M**: Medication withdrawal or side effects.
e. **B**: Bladder, catheter, bladder outlet obstruction, urinary retention.
f. **O**: Oxygen deficiency.
g. **P**: Pain.

5. Nonpharmacologic treatments:
a. Identify the possible cause of delirium and treat if indicated and part of the plan of care.
b. Establish safe, soothing, quiet environment.
c. Reduce stimuli, specifically at night; lights; sounds; and interactions. If in a hospital or facility setting, combine treatments as much as possible.
d. Ask relatives/friends/visitors to stay by client for comfort or leave to reduce stimuli.
e. Make frequent reminders of time and place to reorient if appropriate.
f. Educate caregivers about delirium, causes, and plan for treatment.

6. Pharmacologic treatments:
a. The agent of choice for most EOL clients with delirium is haloperidol (Haldol):
i. Haldol has a favorable side effect profile and can be administered safely both orally and parenterally.
ii. Starting doses are .5 to 1.0 mg orally or intravenously (IV). Use with caution and base medications on individual client needs.
iii. **BLACK BOX WARNING, which is a strict warning established by the Food and Drug Administration (FDA): Haloperidol is not approved for dementia-related psychosis because of increased mortality in elderly dementia clients on conventional or atypical antipsychotics. Most deaths were caused by cardiovascular or infectious events.**
b. Benzodiazepines should be avoided unless the source of the delirium has been identified as alcohol-sedative drug withdrawal. Benzodiazepines may also be useful when severe agitation is uncontrolled by a neuroleptic medication. Benzodiazepines can cause paradoxical worsening of confusion. The American Geriatrics Society (AGS) Beers criteria notes that benzodiazepines are associated with an increased risk of adverse effects, including an increased risk of cognitive impairment, delirium, falls, and fractures (refer to Appendix C, Tables C.1 and C.2).
c. Chlorpromazine (Thorazine) has also been used for dying clients with terminal delirium and may also be useful as an antiemetic. However, this may have higher incidence of side effects such as sedation and hypotension.
d. Although no drug has been FDA-approved for treating dementia's neuropsychiatric symptoms, atypical antipsychotics including chlorpromazine (Thorazine), olanzapine (Zyprexa), risperidone (Risperdal), and quetiapine (Seroquel) are used off-label to address agitation at EOL and are newer atypical antipsychotics. These medications are useful in the treatment of confusion and may be a drug of choice for clients with Parkinson.
e. **BLACK BOX WARNING: Atypical antipsychotics should not be used for dementia-related behaviors because of the risk versus the benefit profile caused by increased risk of cerebrovascular adverse events including stroke and a greater rate of cognitive decline and mortality** (refer to Appendix C, Table C.1).

7. Diagnostic tests/evaluation:
a. The degree of workup to seek the cause of delirium is determined by understanding the disease trajectory and overall goals of care. ▶

b. Always evaluate for the reversible cause of delirium.
c. Nonpharmacologic treatments should always be used in delirium management.

8. Plan:
a. The most important initial step is to determine the plan and goal of care.
b. Determine the cause of the delirium.
c. Determine if the cause is reversible.
d. Treat the cause if appropriate to the goals of care.
e. Educate and provide nonpharmacologic treatments.
f. Provide proper individualized medications.
g. Provide education and support for client and family.

DYSPNEA OR SHORTNESS OF BREATH

A. Dyspnea is defined as distressing SOB or abnormal breathing and is frequently referred to as a feeling of "breathlessness." Dyspnea is a subjective experience of breathing and is described as one of the most distressful symptoms. It is common in many EOL clients and serious illnesses such as lung disease, stroke, end-stage renal disease, heart disease, dementia, and cancers. Dyspnea can have severe effects on the quality of life (QOL) for both the client and their family.

1. Clinical characteristics:
a. Dyspnea can have a severe effect on functional status, affecting the person's ability to complete activities of daily living (ADL).
b. Breath sounds vary depending on the disease process.
c. Many factors improve or worsen symptoms, notably, pain, anxiety, movement, and rest.
d. Dyspnea is a subjective report and a different experience for each individual.

2. Physical examination: Evaluate for the following:
a. Jugular vein distension (JVD).
b. Bilateral crackles.
c. Respiratory rate and depth.
d. Use of accessory muscles.
e. Pain with respiratory movement.
f. Pursed-lip breathing.
g. Cyanosis.
h. Inability to speak, or only able to speak a few words, and then has to rest to breathe.
i. Heart rate >90.
j. Respiratory rate >22.
k. Restless or nonpurposeful movements, particularly at EOL or with a change in level of alertness.
l. Abdomen moves in on inspirations; can see how hard a client is working to breathe.
m. Accessory muscle use.
n. Rise in clavicles during inspiration.
o. Grunting at end of expiration.
p. Guttural sound.
q. Nasal flaring.
r. A look of fear.
s. Useful dyspnea scale for assessing SOB:
i. A respiratory distress observation scale for clients unable to self-report dyspnea:
1) pubmed.ncbi.nlm.nih.gov/20078243/.
2) homecareinformation.net/handouts/hen/Respiratory_Distress_Observation_Scale.pdf.

3. Common complaints:
a. Suffocation.
b. Inability to breathe.
c. Fear.
d. Impending doom.

4. Other signs and symptoms:
a. Anxiety.
b. Cachexia.
c. Depression.
d. Tachycardia.
e. Pain.

5. Pharmacologic treatment:
a. Medications:
i. Opioids are the first-line treatment for dyspnea. Opioids alter the client's awareness of SOB, which in turn leads to the lessening of suffering.
ii. Benzodiazepines may be used as an adjunct medication if anxiety is present with dyspnea. May also be used when opioid medication is not sufficient to treat the feelings of breathlessness.
iii. In rare cases, palliative sedation may be necessary to relieve the symptoms of dyspnea at the EOL. This is only used when all other treatment options have been exhausted and the client continues to have dyspnea.

6. Nonpharmacologic treatment:
a. Oxygen.
b. Counseling.
c. Pursed-lip breathing.
d. Energy conservation.
e. Elevated head of bed (HOB).
f. Good oral care.
g. Calm, cool, relaxed environment.
h. Movement of cool air with fans.
i. Muscle relaxation.
j. Activity grouping to conserve energy.
k. Small frequent meals to conserve energy.

7. Potential complications:
a. Uncontrolled symptoms.
b. Cyclical process: SOB produces anxiety which increases SOB, which increases anxiety, which increases SOB, and continues in a cyclical motion unless treated appropriately.
c. Infection.
d. Decreased interaction with family and friends, creating isolation and depression.
e. Increased fear or anxiety.

8. Diagnostic testing:
a. Disease process and progression evaluation.
b. X-ray if indicated.
c. Lab work if indicated.

9. Differential diagnoses:
a. COPD exacerbation.
b. Advancement of disease process.
c. Pneumothorax.
d. Anemia.
e. CHF exacerbation.
f. Anxiety.
g. Unresolved pain.
h. EOL process.

10. Plan:
a. Understand and discuss the client's and the family's goals of care.
b. Evaluate history of illness, disease process, and progression.
c. Evaluate cause.
d. Treat cause if indicated relevant to client and family goals and plan of care.
e. Treat symptoms of SOB based on cause.

▶

f. Assess and reassess effectiveness of treatment frequently.
g. Support and educate client and family.
h. Validate and identify SOB as a very distressful symptom.

ANOREXIA/CACHEXIA

A. Anorexia is defined as loss of appetite and is usually accompanied by a decrease in intake. Cachexia is defined as lack of nutrition and wasting. Both anorexia and cachexia are commonly seen in many serious illnesses and EOL processes. Many families find it difficult when a loved one will not or cannot eat and weight loss and muscle wasting continue "even though I'm trying so hard to eat." There are several causes of anorexia and cachexia, including simply not having the energy to take in the required nutrition, lack of appetite, or fullness if taking numerous medications. Lastly, at EOL, appetite naturally decreases and may also be a cause. Investigate the cause of anorexia and cachexia.

1. Common complaints:
a. Nausea.
b. Fatigue.
c. Lack of appetite.
d. Client may feel like they are taking in adequate nutrition when that is not the case. Or the client truly is taking in adequate nutrition and the body is utilizing all of the nutrition and energy for the disease process.
e. Progressive weight loss and muscle wasting.
f. Cachexia is rarely reversible in advanced illness.
g. Cachexia may be caused by metabolic abnormalities.
h. Increases distress for both the client and family.
i. Negatively impacts body image and causes body image disturbances.
j. Constant reminder of the disease process and progression.

2. Causes:
a. Oral/systemic infections.
b. Pain.
c. SOB.
d. Fatigue.
e. Constipation.
f. Nausea/vomiting/diarrhea.
g. Malabsorption.
h. Depression.
i. Medications.

3. Potential complications:
a. Fatigue.
b. Weakness.
c. Increased risk for skin breakdown.
d. Increased risk for falls.
e. Increased risk for infections.
f. Metabolic disturbances.
g. Frailty.
h. Isolation.
i. Client and family distress related to weight loss and lack of intake.

4. Physical assessment:
a. Temporal wasting.
b. Significant weight loss (5%–10% of body mass every month).
c. Decreased intake.
d. Poor skin turgor.
e. Decreased abdominal mass or fat (helpful to measure abdominal girth and track).
f. Decrease in upper arm mass or fat (helpful to measure upper arm circumference and track).

5. Pharmacologic treatment:
a. There is no single pharmacologic intervention effective for cachexia.
b. May be appropriate to try appetite stimulants:
i. Mirtazapine (Remeron) 7.5 mg may enhance appetite. If there is no effect on appetite in 6 to 8 weeks, it should be discontinued.
ii. In clients with dementia, use of Aricept can enhance the ability of some to feed themselves. Withdrawal of Aricept may lead to reduced ability to self-feed and eat at mealtimes.
iii. Megestrol acetate (MA; Megace) is on the AGS Beers list for minimal effect of weight; increased risk of thrombotic events and possible death (refer to Appendix C, Table C.1).
c. Antioxidant polyphenols from plant foods have been used for cancer-related cachexia. Polyphenols are in fresh and cooked plant foods, particularly those with rich colors, including red, purple, and black. Wine is a polyphenol-rich drink. Wine before meals may be helpful in increasing appetite.
d. Other medications used in the treatment of anorexia and cachexia include dronabinol (Marinol), corticosteroids, thalidomide, adenosine triphosphate (ATP), omega 3 fatty acids, tumor necrosis factor (TNF) inhibitor, or oxandrolone (Oxandrin).
e. In some cases, parenteral or enteral nutrition may be useful in clients who cannot swallow. However, consider risks versus benefits and each individual client, disease process, and progression. Evaluate the client and family goals and plan of care for cachexia and support these decisions.

6. Nonpharmacologic treatments:
a. Educate the client and family concerning causes of cachexia.
b. Eat small frequent meals.
c. Encourage client to eat foods that they enjoy.
d. Eat foods that are easy to chew with sauces.
e. Eliminate dietary restrictions.
f. Encourage high-calorie foods; nutritional supplements may be useful as tolerated.

7. Plan:
a. Overall, when caring for a seriously ill or EOL individual, the problem of anorexia or cachexia may be one of the most distressing to the client or the family. Eating has many emotional ties; provide support and reassurance to both client and family.
b. Constant evaluation and appropriate support and guidance are necessary.
c. Assess and reassess nutritional status.
d. Determine client and family goals and support realistic decisions.
e. Educate client and family on risk versus benefit.
f. Utilize interdisciplinary team (IDT); consult a nutritionist if appropriate.
g. Consider caloric counts.
h. Educate the client and the family about anorexia and cachexia. It is important to listen to their concerns and validate in a respectful manner. Explain that an increase in calories does not always equate with weight gain or client improvement.

▶

i. Consider palliative or hospice referral; anorexia and cachexia in serious illness are associated with decreased survival.

8. Diagnostic tests/evaluation:

a. Weigh the client standing or use a bed scale available for those who are bed-bound.

b. Measure arm/abdominal circumferences; this is particularly helpful for clients who cannot be weighed easily. Track the client's weight measurements.

c. Evaluate electrolytes as indicated.

d. Serum albumin decreases as nutritional status declines and also has a poor prognosis.

9. Special considerations:

a. Aggressive nutritional treatments in advanced illness may cause more harm than benefit due to refeeding syndrome. Refeeding syndrome is the potentially fatal shifts in fluids and electrolytes that may occur in malnourished clients receiving parenteral or enteral refeeding. High-risk clients include those who have been chronically undernourished, especially those who have diminished physiologic reserve. Seriously consider risk versus benefit before initiating aggressive nutritional treatments. The American Society for Parenteral and Enteral Nutrition (ASPEN) has clinical guidelines on their Guidelines and Clinical Resources available at aspenjournals.onlinelibrary.wiley.com/doi/10.1002/jpen.1669.

CONSTIPATION

A. Constipation is defined as the infrequent passage of stool. **The key to constipation in palliative care (PC) is prevention. Clients who are started on opioids should always have a bowel plan in place. If prevention of constipation is not treated seriously, it can lead to more complicated problems such as a preventable bowel obstruction:**

1. Common complaints:

a. Rectal pressure.

b. Straining.

c. Cramps/abdominal distension/low back pain.

d. Nausea.

e. Fullness.

2. Causes:

a. Medications:

i. Opioids.

ii. Antidepressants.

iii. Antacids.

iv. Chemotherapy.

v. Overuse of bowel stimulants.

b. Obstructive tumor.

c. Neurologic disorders.

d. Decreased or no activity.

e. Hypercalcemia/hypokalemia.

f. Spinal cord compression.

g. Surgical adhesions.

h. Dehydration.

i. Weakness.

j. Pain.

k. Depression.

3. Assessment of constipation:

a. History:

i. Characteristics of stool, appearance, and consistency.

ii. Frequency of stool.

iii. History of use of bowel medication.

iv. Fluid intake.

v. Anorexia.

vi. Presence of nausea and/or vomiting.

vii. Review all medications, including over-the-counter (OTC) and herbal products.

b. Physical assessment:

i. Inspect, auscultate, palpate, and percuss the abdomen to evaluate the presence of:

1) Bowel sounds: hyperactive, hypoactive, absent.

2) Bloating.

3) Ascites.

4) Distension.

5) Masses.

6) Pain and tenderness.

ii. Digital rectal examination:

1) Avoid in neutropenic clients.

2) Digital examination may reveal impaction, tumor, or rectocele.

3) Visual examination of the rectal area may reveal hemorrhoids, ulcerations, rectal fissures, or skin breakdown, which all make defecation painful.

4. Diagnostic tests:

a. Abdominal x-ray to rule out obstruction.

5. Differential diagnosis:

a. Obstruction.

6. Plan:

a. Written bowel plans for all clients on opioids are necessary because of constipation.

b. Prophylactic stool softeners should be used as indicated.

c. Bowel elimination should occur no less than every 3 days regardless of intake.

d. Assess and reassess.

e. Check for impaction and obstruction.

f. Educate the client and the family on constipation/bowel plan.

7. Individual/special considerations:

a. May be very embarrassing for client to address bowel problems; consider privacy, and address the issue respectfully.

b. Encourage client and/or family to discuss openly and report problems or issues/patterns in bowel elimination and output.

NAUSEA AND VOMITING

A. Nausea is a feeling and will be a subjective report, whereas vomiting will be visible with objective information. Nausea and vomiting (N&V) occurs in up to 70% of terminally ill individuals. N&V has negative effects on QOL, causing weight loss, electrolyte disturbances, isolation, frustration, anxiety, and many other associated symptoms.

B. It is important to remember that psychological or emotional factors can also cause N&V, stimulating the emetic receptors in the brain. The clinician must understand the type of N&V to treat it correctly:

1. Causes:

a. Physiologic:

i. Gastric irritation, constipation, intestinal obstruction.

ii. Liver failure, ascites.

iii. Autonomic dysfunction.

iv. Anxiety: increases gastric acid production. ▶

v. Specific treatments such as radiation, chemotherapy, or medications.
vi. Infection.
vii. Metabolic abnormalities such as renal failure or hepatic failure.
viii. Increased intracranial pressure.
ix. Vestibular disturbances.

2. Clinical characteristics and pharmacologic treatments:
a. Chemoreceptor trigger zone-based N&V:
i. Stimulated by chemotherapeutic agents.
ii. Bacterial toxins caused by infection or viruses.
iii. Metabolic products such as uremia, hypercalcemia, and infection.
iv. Opioids.
v. Radiation therapy-induced nausea.
vi. Pharmacologic: First-line treatment for chemoreceptor trigger zone N&V is a dopamine antagonist such as Phenergan or Compazine and sometimes Haldol.
b. Cerebral cortex-based N&V:
i. Anxiety.
ii. Taste.
iii. Smell.
iv. Anticipatory nausea.
v. Increased intracranial pressure.
vi. Pharmacologic: The first-line treatment for cerebral cortex N&V is administration of anxiolytics such as lorazepam or hydroxyzine.
c. Vestibular-based N&V:
i. Motion.
ii. Opioids that sensitize the vestibular center.
iii. Pharmacologic: First-line treatment for vestibular N&V involves antihistamines and anticholinergics.
d. Gastrointestinal tract-based N&V:
i. Gastric stasis or obstruction.
ii. Medications.
iii. Metastatic process.
iv. Infection.
v. Treatments.
vi. Pharmacologic treatment for gastrointestinal tract N&V: First-line treatment for gastric stasis is generic metoclopramide. Ondansetron is also useful in the treatment of N&V.

3. Assessment:
a. Obtain a thorough history that includes clear information so that the clinician can identify what type of N&V the client is exhibiting. Much like assessment for pain, the clinician has to ask many questions to properly assess the symptom of N&V.
i. What is the consistency of the emesis?
ii. What is the intensity—wrenching, belching acid?
iii. Does anything make it better or worse?
iv. How often does nausea and/or vomiting occur? Are there any patterns? It may help to have the client or family keep a symptom diary.
v. How much is the client vomiting?
vi. Are there symptoms or factors that accompany the N&V, such as dizziness or low blood sugar? Does it occur when taking medication or eating/smelling certain foods?
vii. Evaluate for constipation, impaction, or obstruction.
viii. Evaluate for uncontrolled pain, infection, or other processes.
ix. Does the client have anxiety or other emotional or existential concerns leading to N&V?

4. Diagnostic tests:
a. Labs to consider:
i. Renal and liver function tests.
ii. Electrolytes: Assess for dehydration.
iii. Urinalysis: Assess for infection.
iv. X-ray of abdomen: Check for bowel obstruction.
v. CT of head: Assess for brain metastasis.

5. Nonpharmacologic treatments for N&V:
a. Educate the client and the family to treat nausea as soon as symptoms start.
b. Teach relaxation techniques.
c. Acupuncture and acupressure techniques may be helpful.
d. Recommend music therapy.
e. Teach families and care providers about meals:
i. Serve meals at room temperature with clear fluids.
ii. Identify and avoid strong smells.
iii. Avoid large high-bulk meals; instead, eat small, frequent meals.
iv. Position the client in an upright position during meals.
v. While preparing meals, the client should avoid close proximity to the food preparation area/kitchen.
f. At times a nasogastric tube may be needed for intractable nausea and/or vomiting or unrelieved abdominal pressure to alleviate symptoms.

6. Plan for N&V control:
a. Identify the cause and type of N&V.
b. Assess and reassess the client as necessary.
c. Identify client and family goals and plan of care.
d. Provide pharmacologic interventions.
e. Educate the client and the family about nonpharmacologic interventions.

XEROSTOMIA

A. Xerostomia is defined as dry mouth, together with difficulty in mastication, swallowing, and speech. Xerostomia occurs in up to 97% of clients at EOL and greatly impairs QOL; it can be distressing to the client and family members. Management is primarily nonpharmacologic and can be completed by educating family and caregivers. This management provides families and caregivers with a means of control to "do" something, often when the individual is feeling helpless at EOL. Oral care can be a method of bonding or of resolution and is a very important aspect of client care.

1. Causes:
a. Medications:
i. Anticholinergics.
ii. Opioids.
iii. Diuretics.
b. Radiation.
c. Systemic disease.
d. Oxygen.

2. Management:
a. Identify and, if possible, correct the underlying cause.
b. Educate the client and the family on providing proper and frequent oral care. ▶

i. Brushing teeth.
ii. Cleaning tongue and lips.
iii. Keeping the mouth and lips moist.

c. Offer frequent sips of liquid, if appropriate, or frequent mouth moistening if the client cannot swallow.
d. If appropriate, soft moist foods can be offered.
e. If client is able, suggest chewing sugarless gum, use of lubricating sprays, and lozenges.
f. Maintain humidity in the room.
g. Humidify oxygen.
h. Oral care should continue even if the client is unresponsive.

3. Plan:
a. Assess for signs and symptoms of xerostomia.
b. Identify cause and correct if possible.
c. Educate client, family, and caregivers regarding proper oral care.
d. Assess and reassess as needed.

FATIGUE

A. Fatigue, or a feeling of weariness, has been reported to occur in up to 90% of cancer clients. Fatigue is also linked to many other serious illnesses such as COPD, CHF, coronary artery disease (CAD), HIV/AIDS, and advanced renal disease. Much of the treatment for fatigue is nonpharmacologic and requires the clinician to educate the client and the family. Proper assessment to determine the cause of fatigue is imperative.

1. Causes:
a. Disease states such as anemia, electrolyte imbalances, malnutrition, infection, hyperglycemia, fever, pain, organ failure, hypoxia.
b. Depression.
c. Inactivity and immobility.
d. Inadequate rest.
e. Unrelieved symptoms.
f. Medications.
g. Radiation/chemotherapy/immunotherapy.
h. Spiritual distress.

2. Assessment:
a. Obtain an adequate history by asking questions to acquire both subjective and objective information.
i. How do you feel? Are you feeling weak, tired, wiped out?
ii. How long does the fatigue last? Can you see a pattern?
iii. Have you noticed anything that makes the fatigue better or worse?
iv. Have you noticed if any of the medications or treatments make the fatigue better or worse?
v. Do you feel anxious or depressed?
vi. Do you find it difficult to concentrate?
vii. Tell me how fatigue is affecting your daily life.
viii. Walk me through a typical day.
ix. Tell me how you sleep and what that is like for you.

b. Monitor vital signs, assessing for fever, increased heart rate, weak pulse, and increased respiratory rate.
c. Assess as the client moves and talks; notice if the individual gets short of breath with very simple tasks (stating a few words, standing, walking a few steps, transferring sitting to standing).
d. Evaluate hydration status.
e. Evaluate muscle status, muscle wasting, muscle strength, and endurance.
f. Evaluate medications: Are there medications that cause sedation and fatigue? Can this be changed; if not, can the time be changed to be taken at bedtime? If not, can the client and family be instructed on how to group tasks when awake?

3. Diagnostic testing:
a. Oxygenation status hemoglobin (Hgb), complete blood count (CBC), and differential.
b. Thyroid function.
c. Electrolytes.

4. Treatments:
a. Provide assistance that will help the individual to maintain independence and functional abilities as long as possible, such as physical therapy and occupational therapy.
b. Initiate therapy as appropriate and in alignment with client goals and plan of care, such as transfusions, oxygen, and IV fluids.

5. Plan:
a. Assess for signs and symptoms of fatigue.
b. Educate client and family regarding frequent rest periods and grouping activities as much as possible in efforts to conserve energy.
c. Work with IDT to provide individualized care in efforts to maintain independence and functional ability.
d. Provide support; suggest home health or hospice aide as appropriate.
e. Assess and reassess for fatigue.

DEPRESSION

A. Depression is noted in many serious illnesses and has been reported in up to 50% of clients in the setting. The National Cancer Institute notes that, although both depression and anxiety have been frequently reported as comorbid factors in chronic medical conditions, depression is often unrecognized, undertreated, or altogether untreated. Symptoms of depression and anxiety are usually responsive to treatment, and early recognition, assessment, and diagnosis is important to improve outcomes. The client, family, and clinician should understand that depression and suicidal ideation are not normal at EOL and that symptoms should be aggressively treated.

1. Clinical characteristics:
a. Persistent feelings of helplessness, hopelessness, or inadequacy.
b. Disease-related causes of depression include:
i. Uncontrolled symptoms such as pain, anorexia, and N&V.
ii. Sleep disturbances.
iii. Sepsis, abnormal metabolic states, delirium, organic mental disorders, medication reactions.
c. Psychological issues that may lead to depression:
i. Existential factors such as fear, loss of independence, loss of control, body image disturbance, impending death.
ii. History of preexisting psychological conditions.

2. Medications:
a. Antihypertensives, analgesics, steroids, hypoglycemics, chemotherapeutics, benzodiazepines, and others.
b. Treatments such as radiation.

3. Individual situations increase the chance of depression: ▶

a. Financial, social, safety, or other issues can increase the chances of depression.
b. Assess and utilize the IDT.

4. Assessment: It may be difficult to assess for depression. Evaluate the client about mood, fear, social withdrawal, and emotional status, including:
a. Complaints of lack of appetite and weight loss with unclear cause.
b. Insomnia.
c. Decreased sexual interest.
d. Decreased interest in activities usually found pleasurable, such as reading, television, and games.
e. Feelings of helplessness and/or hopelessness.
f. Psychomotor agitation.
g. Fatigue and/or a complaint of diminished energy.
h. Ask direct questions when assessing for depression.
i. Are you depressed?
ii. How would you describe your mood?
iii. How are you sleeping?
iv. What is your energy level?
v. What do you see in your future?
1) **Ask: "Have you had thoughts of harming yourself?" The client may need immediate evaluation by a psychiatric professional if they have a lethal, precise suicide plan and the ability to carry out the plan.**

5. Pharmacologic treatment:
a. The first-line medications for treating depression are selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine, paroxetine, sertraline, or citalopram.
i. Geriatric consideration: Citalopram has been best studied for safety in older adults with cardiac problems.
ii. Prozac and other SSRIs can be linked with anorexia and difficulty sleeping.
b. Other medications that may be used to treat depression include:
i. Tricyclic antidepressants such as amitriptyline. However, nortriptyline should not be used if the client has suicidal ideation.

6. Nonpharmacologic treatment measures for depression:
a. Provide mechanisms for the client to remain in control and to maintain autonomy and independence for as long as possible.
b. Encourage reminiscing, promote focusing on life's accomplishments, and help with EOL resolutions and closure with family.
c. Provide anticipatory grief counseling.
d. Provide adequate symptom relief.
e. Use cognitive behavioral techniques.

7. Plan:
a. Assess for signs and symptoms of depression.
b. Understand client diagnosis, prognosis, progression, goals, and plan of care.
c. Have clear conversation with client and family.
d. Identify depression early.
e. Treat depression appropriately, know client's history of major depressive episodes.
f. Incorporate nonpharmacologic methods and utilize IDT.
g. Educate and provide supportive relationship for client and family.
h. Ensure client safety.
i. Assess and reassess frequently.

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SOCIAL ASPECTS OF CARE

A. Access to care:

1. Managing multiple chronic illnesses and disabilities is challenging in the elderly population, who require more extensive and in-depth care from providers.

2. Elderly clients require more assistance from caregivers and/or family members.

3. Care for the elderly is often episodic and unplanned. This pattern for chronic and serious illnesses causes clients to access care such as the ED. Episodic treatment in the ED is inadequate without a discharge plan. The lack of a comprehensive functional assessment during an ED visit places the elder client at risk.

4. Fragmented care puts the elder client at risk for errors caused by polypharmacy, falls, functional decline, untreated symptoms, and increased or undertreated pain and institutionalization. Using an interdisciplinary team (IDT):
a. Coordinates care and assists with follow-up care and consistency of care.
b. Reduces fragmentation of care.
c. Contains cost of care.
d. Reduces risk of polypharmacy as care is coordinated.
e. Clients who receive appropriate follow-up care have improved quality of life, receive proper education, experience fewer hospitalizations, have lower costs, and experience less client and family stress.

5. Other specific considerations when caring for elderly clients in palliative care (PC):
   a. Elderly clients need extensive family and caregiver support and care.
   b. Clients make increased demands on family and caregivers when they lose the ability to drive.
   c. As disease progresses, activities of daily living (ADL) become progressively more difficult to complete and require significant assistance from others.

6. Special considerations for client and family feelings:
   a. Older spouses may be taking care of their ill older spouses.
   b. Both the client and the family may have feelings of helplessness; they need education on tasks that can be completed, giving them a sense of control.
   c. Children and their parents experience emotional difficulty with role reversal, such as bathing their elderly parent when they are no longer able to perform general grooming activities.
   d. Feelings of resentment and/or guilt as well as depression, anxiety, and frustration may be exhibited.

B. Financial concerns:

1. Many older adults are on a fixed income, with medication expenses accounting for a significant portion of their income.

2. Older adults may have limited supplemental insurance and be unable to remain in their own homes as their disease progresses and costs rise.

3. Client and family may have lack of understanding of insurance and supplemental insurance use as situations and level of care change.

4. Placement in residential care in the last days of life may require dissolution of all assets.

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SPIRITUAL, RELIGIOUS, CULTURAL, AND EXISTENTIAL ASPECTS

A. Culture greatly influences how a person and the family respond to an illness or crisis and how decisions are made within the situation. Completion of a cultural assessment is a crucial piece of client and family care. This assessment addresses many facets of a person's life and how it affects the view of life, illness, interactions, quality of life (QOL), and death. Culture includes the whole person and how that individual identifies with ethnicity, race, age, national origin, gender, religion, marital status, family, sexual orientation, professional role, current residence, community role, economic status, and educational status. This assessment shows the client and family that their culture, beliefs, and practices are important and valid.

1. Identify who the major support person or people are.

2. Identify who the decision-makers are.

3. What is the primary or preferred language? Are there language barriers to care?

4. Is it appropriate to make eye contact, touch?

5. Are there gender issues associated with communication or care/providers?

6. Ask about religion or spirituality and how they may affect care.

7. Are there specific nutritional restrictions or specific religious observations?

8. Understand the individual's economic situation. This question can be uncomfortable to ask, however, for it directly affects access to care, medications, safety, living situation, family unit, and other aspects of care.

9. Ask about health and illness beliefs and practices, traditions, routines, and rituals.

10. What types of past experiences has the individual and family had?

11. What do the client/family know about the disease, prognosis, and progression?

12. What are the beliefs and practices about pain and suffering?

13. Ask about traditional therapies, herbal medications, integrative health remedies, and other therapies:
   a. Make sure to address and include traditional therapy when able and appropriate as they are a very important part of client- and family-centered care.
   b. Clients/families may not inform clinicians if they do not believe these practices will be taken seriously or included in the plan of care.
   c. Some herbal treatments can interact with medications.
   d. Traditional therapies are often mistaken, misinterpreted, or translated as noncompliance.

14. To ensure dignity and respect, discuss care of the client's body as death approaches.

15. How do the client and the family view organ donation?

SPIRITUALITY AND RELIGION

A. Spirituality can be of value even to those who do not consider themselves religious. Spirituality provides a means to forgive and to ask for forgiveness. Religion encompasses beliefs and rituals, and includes the importance of prayer, ceremonies, and other traditions.

1. Spiritual assessment: Dr. Christina Puchalski developed the **FICA** spiritual assessment as a tool to evaluate the importance of the client's personal faith and belief system as a guide for discussion (smhs.gwu.edu/spirituality-health/sites/spirituality-health/files/FICA-Tool-PDF-ADA.pdf):
   a. **F**aith and belief.
   b. **I**mportance.
   c. **C**ommunity.
   d. **A**ddress in care.

2. Spiritual symptoms of distress: Spiritual distress often presents as many different symptoms. Often the chaplain from the interdisciplinary team (IDT) can be consulted to assist; other times, education, listening, or just being present is needed to ensure the person can validate their feelings. Common symptoms of spiritual distress include:
   a. Sense of abandonment.
   b. Direct or indirect anger.
   c. Sense of betrayal.
   d. Hopelessness or despair.
   e. Fear.
   f. Guilt.
   g. Need to seek forgiveness.
   h. Regret.
   i. Remorse.
   j. Depression.

3. Plan:
   a. Ensure cultural and spiritual assessment is completed and addressed specific to the whole person's needs.
   b. Utilize the IDT and offer the client and the family appropriate services.

COMMUNICATION

A. Communication is one of the most complicated and fundamental aspects of palliative care. Clinicians are uncomfortable with the discussions surrounding death and dying. Often the clinician believes they have failed the client as the disease progresses to the terminal phases. Communication with the client and the family must meet the needs of the client; be clear and concise, and provide the client and the family with information to make informed decisions.

1. Barriers to effective communication exist for the clinician and the client/family. These barriers include:
- a. Fear.
- b. Lack of experience.
- c. Avoidance of emotion.
- d. Insensitivity.
- e. Guilt.
- f. Disagreement with decisions.
- g. Lack of understanding of the client's culture.
- h. Lack of understanding of goals.
- i. Ethical concerns.

2. Communication tips:
- a. Build a trusting relationship.
- b. Take time to listen, be silent, and present.
- c. Be honest.
- d. Provide consistency in team communication.
- e. Coordinate care among clinicians and provide a unified, clear message.
- f. Validate client and family concerns.
- g. Understand you can give too much information; ask how much they want to know.
- h. Be sensitive.
- i. Sit down when you speak with the client and the family.
- j. Communication is not all that is "said." Body language accounts for over 80% of nonverbal communication.

3. Communication tools:
- a. Use the ask-tell-ask method of communication.
  - i. Ask the client/family to describe their understanding of the subject.
  - ii. Tell the client/family the information in a clear, concise manner without use of medical jargon.
  - iii. Ask the client/family if they understood the information presented and ask them to repeat the content back or summarize the information.
- b. Recognize and respond to emotion.
- c. Assess the client's coping style.
- d. Encourage reminiscing.
- e. Do not change the subject.
- f. Use open-ended questions.
- g. Employ useful phrases to encourage communication.
  - i. "Tell me more."
  - ii. "Tell me what you're hoping for."
  - iii. "Sometimes our hopes change; tell me yours."
  - iv. "Tell me what questions you have."
    1) "I know your family members from a medical standpoint; tell me about them as persons."
    2) "Tell me what this means for you and your life."
    3) "I know what anxiety feels like for me; tell me what it feels like for you."

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ETHICS

A. The American Nurses Association's *The Nursing Code of Ethics* establishes ethical standards for the profession. While working in palliative care (PC), many difficult decisions are made. These decisions often cause clinicians moral or ethical distress. Knowing and utilizing *The Nursing Code of Ethics* allow the nurse to feel confident they are following the ethical obligations of the profession. *The Nursing Code of Ethics* with interpretive statements is available at www.nursingworld.org/coe-view-only.

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APPENDIX 4.1 HOSPICE CRITERIA CARD

Hospice is a program designed to care for the dying and their special needs. All hospice programs should include:
A. **Control of pain and other symptoms** through medication, environmental adjustment, and education.
B. **Psychosocial support** for both the client and family, including all phases from diagnosis through bereavement.
C. **Medical services** commensurate with patient needs.
D. **Interdisciplinary team (IDT)** approach to patient care, patient/family support, and education.
E. Integration into existing facilities where possible.
F. Specially trained personnel with expertise in care of the dying and their families.

HOSPICE ELIGIBILITY CRITERIA
In order to be eligible to elect hospice care under Medicare, an individual must be—(a) Entitled to Part A of Medicare; and (b) certified as being terminally ill in accordance with § 418.22.

Duration of Hospice Care Coverage—Election periods:
A. An initial 90-day period;
B. A subsequent 90-day period; or
C. An unlimited number of subsequent 60-day periods.

Hospice Face-to-Face (FTF) Encounter
Must include documentation that a hospice physician or a hospice nurse practitioner had a FTF encounter with the client. This encounter is used to gather clinical findings to determine continued eligibility for hospice care. The FTF must occur within 30 calendar days prior to the start of the 3rd benefit period and every subsequent recertification period.

HOSPICE LEVELS OF CARE
Routine Home Care (RHC): Core services of hospice interdisciplinary team provided at client's home (place of residence).
Continuous Home Care (CHC): Intended to support patients and their caregivers through brief periods of crisis. CHC provides care for 8–24 hours a day. ≥50% of care must be primarily provided by an LPN or RN. Home health aide or homemaker services can be used to cover the needs.
Inpatient Respite Care (IRC): Short-term care to provide relief to family/primary caregiver. Limited to 5 consecutive days.
General Inpatient Care (GIP): Care provided in acute hospital or other setting with intensive nursing and other support outside of the home. For management of uncontrolled distressing physical symptoms (e.g., uncontrolled pain, respiratory distress) or psychosocial problems (e.g., unsafe home or imminent death when family can't cope at home).

HOSPICE PRINCIPAL DIAGNOSIS
Identify the condition that is the main contributor to the person's terminal prognosis. Non-specific diagnoses such as debility or adult failure to thrive (AFTT) may no longer be listed as a principal terminal diagnosis. Debility and AFTT can and should be listed as secondary (related) conditions to support prognosis if indicated.

TERMINAL ILLNESS: GENERAL (NON-SPECIFIC)
Terminal condition not attributed to a single specific illness.
AND
Rapid decline over past 3–6 months as evidenced by:
Progression of disease evidenced by sex, signs, and test results.
Decline in PPS to ≤50%.
Involuntary weight loss >10% and/or albumin <2.5 (helpful).

Cancer
Client meets ALL of the following:
1. Clinical findings of malignancy with widespread, aggressive, or progressive disease as evidenced by increasing symptoms, worsening lab values, and/or evidence of metastatic disease.
2. Palliative Performance Scale (PPS) ≤70%.
3. Refuses further life-prolonging therapy OR continues to decline in spite of definitive therapy.

Supporting documentation includes:
Hypercalcemia >12.
Cachexia or weight loss of 5% in past 3 months.
Recurrent disease after surgery/radiation/chemotherapy.
Signs and sex of advanced disease (e.g., nausea, requirement for transfusions, malignant ascites, or pleural effusion).

Dementia
The client has both 1 and 2.
1. Stage 7C or beyond according to the FAST Scale.
   AND
2. One or more of the following conditions in the past 12 months:
   Aspiration pneumonia.
   Pyelonephritis.
   Septicemia.
   Multiple pressure ulcers (Stage 3–4).
   Recurrent fever.
   Other significant condition that suggests a limited prognosis.
   Inability to maintain sufficient fluid and calorie intake in the past 6 months (10% weight loss or albumin <2.5 gm/dL).

(*continued*)

APPENDIX 4.1 HOSPICE CRITERIA CARD (*CONTINUED*)

Functional Assessment Scale (FAST) for Alzheimer's Type Dementia

1	No difficulty either subjectively or objectively.
2	Complains of forgetting location of objects. Subjective work difficulties.
3	Decreased job functioning evident to coworkers. Difficulty in traveling to new locations. Decreased organizational capacity.*
4	Decreased ability to perform complex task (e.g., planning dinner for guests, handling personal finances such as forgetting to pay bills).
5	Requires assistance in choosing proper clothing to wear for the day, season, or occasion (e.g., patient may wear the same clothing repeatedly, unless supervised).[a]
6	Occasionally or more frequently over the past weeks*, for the following: A. Improperly putting on clothes without assistance or cueing. B. Unable to bathe properly (not able to choose proper water temp). C. Inability to handle mechanics of toileting (e.g., forget to flush the toilet, does not wipe properly or properly dispose of toilet tissue). D. Urinary incontinence. E. Fecal incontinence.
7	A. Ability to speak limited to approximately ≤6 intelligible different words in the course of an average day or in the course of an intensive interview. B. Speech ability is limited to the use of a single intelligible word in an average day or in the course of an intensive interview. C. Ambulatory ability is lost (cannot walk without personal assistance). D. Cannot sit up without assistance (e.g., the individual will fall over if there are not lateral rests [arms] on the chair). E. Loss of ability to smile. F. Loss of ability to hold up head independently.

[a]Scored primarily on information obtained from a knowledgeable informant.

HEART DISEASE
The client has 1 and either 2 or 3.
1. CHF with **NYHA Class IV* symptoms and both:**
   Significant symptoms at rest.
   Inability to carry out even minimal physical activity without dyspnea or angina.
2. Patient is optimally treated (i.e., diuretics, vasodilators, ACEI, or hydralazine and nitrates).
3. The patient has angina pectoris at rest, resistant to standard nitrate therapy, and is either not a candidate for or has declined invasive procedures.

Supporting documentation includes:
EF ≤20%, treatment resistant symptomatic dysrhythmias, h/o cardiac related syncope, CVA 2/2 cardiac embolism, h/o cardiac resuscitation, concomitant HIV disease.

The client has either 1A or 1B and 2 and 3.
1A. CD4+ <25 cells/mcL **OR 1B.** Viral load >100,000.
AND
2A. At least one (1): CNS lymphoma, untreated or refractory wasting (loss of > 33% lean body mass), (MAC) bacteremia, progressive multifocal leukoencephalopathy, systemic lymphoma, visceral KS, Renal failure no HD, cryptosporidium infection, refractory toxoplasmosis.
AND
3A. PPS of <50%.

LIVER DISEASE
The patient has both 1 and 2.
1. End stage liver disease as demonstrated by
   A or B, and C:
   A. PT >5 sec.
      OR
   B. INR >1.5.
      AND
   C. Serum albumin <2.5 gm/dL.
      AND
2. One or more of the following conditions:

Refractory ascites, h/o spontaneous bacterial peritonitis, hepatorenal syndrome, refractory hepatic encephalopathy, h/o recurrent variceal bleeding.

(*continued*)

APPENDIX 4.1 HOSPICE CRITERIA CARD (*CONTINUED*)

Supporting documentation includes:
Progressive malnutrition, muscle wasting with decreased strength, ongoing alcoholism (>80 g ethanol/day), hepatocellular CA HBsAg positive, hep. C refractory to treatment.

PULMONARY DISEASE
Severe chronic lung disease as documented by 1, 2, and 3.
1. The client has all of the following:
   Disabling dyspnea at rest.
   Little or no response to bronchodilators.
   Decreased functional capacity (e.g., bed to chair existence, fatigue, and cough).
   AND
2. Progression of disease as evidenced by a recent h/o increasing office, home, or ED visits and/or hospitalizations for pulmonary infection and/or respiratory failure.
   AND
3. Documentation within the past 3 months ≥1:

Hypoxemia at rest on room air (p02 <55 mmHg by ABG) or oxygen saturation <88%.
Hypercapnia evidenced by pC02 >50 mmHg.
Supporting documentation includes:
Cor Pulmonale and right heart failure.
Unintentional progressive weight loss.

NEUROLOGIC DISEASE
(Criteria are very similar for chronic degenerative conditions such as ALS, Parkinson's, Muscular Dystrophy, Myasthenia Gravis, or Multiple Sclerosis.)
The client must meet at least one of the following criteria (1 or 2A or 2B):
1. *Critically impaired breathing capacity,* with all:
   Dyspnea at rest, vital capacity <30%, need O2 at rest, patient refuses artificial ventilation.
   OR
2. *Rapid disease progression* with either A or B below:
   Progression from:
   independent ambulation to wheelchair or bed-bound status
   normal to barely intelligible or unintelligible speech
   normal to pureed diet
   independence in most ADLs to needing major assistance in all ADLs.
   AND
   A. *Critical nutritional impairment* demonstrated by all of the following in the preceding 12 months:
      Oral intake of nutrients and fluids insufficient to sustain life.
      Continuing weight loss.
      Dehydration or hypovolemia.
      Absence of artificial feeding methods.
      OR
   B. Life-threatening complications in the past 12 months as demonstrated by ≥1:
      Recurrent aspiration pneumonia, pyelonephritis, sepsis, recurrent fever, Stage 3 or 4 pressure ulcer(s).

RENAL FAILURE
The client has 1, 2, and 3.
1. The client is not seeking dialysis or renal transplant.
   AND
2. Creatinine clearance* is <10 cc/min (<15 for diabetics).
   AND
   1. Serum creatinine >8.0 mg/dL (>6.0 mg/dL for diabetics).

Supporting documentation for chronic renal failure includes:
Uremia, oliguria (urine output <400 cc in 24 hours), intractable hyperkalemia (>7.0), uremic pericarditis, hepatorenal syndrome, intractable fluid overload.
Supporting documentation for acute renal failure includes:
Mechanical ventilation, malignancy (other organ system), chronic lung disease, advanced cardiac disease, advanced liver disease.

STROKE OR COMA
The client has both 1 and 2.
1. Poor functional status PPS* ≤40%.
   AND
2. Poor nutritional status with inability to maintain sufficient fluid and calorie intake with ≥1 of the following:
   ≥10% weight loss in past 6 months.
   ≥7.5% weight loss in past 3 months.
   Serum albumin <2.5 gm/dL.
   Current history of pulmonary aspiration without effective response to speech therapy interventions to improve dysphagia and decrease aspiration events.

(*continued*)

APPENDIX 4.1 HOSPICE CRITERIA CARD (*CONTINUED*)

Supporting documentation includes:
Coma (any etiology) with three of the following on the third day of coma:
Abnormal brainstem response.
Absent verbal responses.
Absent withdrawal response to pain.
Serum creatinine >1.5 gm/dL.

Palliative Performance Scale (PPS)

%	Ambulation	Activity Level Evidence of Disease	Self-Care	Intake	Level of Consciousness	Estimated Median Survival in Days*		
						A	**B**	**C**
100	Full	Normal / *no disease*	Full	Normal	Full	N/A	N/A	108
90	Full	Normal / *some disease*	Full	Normal	Full			
80	Full	Normal with effort/ *some disease*	Full	Normal or reduced	Full			
70	Reduced	Can't do normal job/ work/ *some disease*	Full	Normal or reduced	Full	145		
60	Reduced	Can't do hobbies/ housework / *significant disease*	Occasional assistance needed	Normal or reduced	Full or confusion	29	4	
50	Mainly sit/lie	Can't do any work / *extensive disease*	Considerable assistance needed	Normal or reduced	Full or confusion	30	11	41
40	Mainly in bed	Can't do any work / *extensive disease*	Mainly assistance	Normal or reduced	Full /drowsy/ confusion	18	8	
30	Bed bound	Can't do any work / *extensive disease*	Total care	Reduced	As above	8	5	
20	Bed bound	Can't do any work / *extensive disease*	Total care	Minimal sips	As above	4	2	6
10	Bed bound	Can't do any work / *extensive disease*	Total care	Mouth care only	Drowsy or coma	1	1	
0	Death	–	–	–	–	–	–	–

∗**A** = Survival post-admission to an inpatient palliative unit, all diagnoses (Virik & Glare, 2002).
B = Days until inpatient death following admission to an acute hospice unit, diagnoses not specified (Anderson, Downing, & Hill, 1996).
C = Survival post-admission to an inpatient palliative unit, cancer patients only (Morita, Tsunoda, Inoue, & Chihara, 1999).

DISCLAIMER: The Hospice Criteria Card authors have made every effort to provide information that is accurate and complete. The information contained herein is provided "as is" and without warranty of any kind. The contributors to this card disclaim responsibility for any errors or omissions or for results obtained from the use of information contained herein.

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CHAPTER 5

GERIATRIC SYNDROMES

Wesley Cook

INTRODUCTION

A. Preserving quality of life (QOL) by maintaining function is arguably the chief objective of older adult (OA) primary care. While dynamic and deeply personal, a definition of QOL that is unmoored from the OA's current health status creates unrealistic expectations with costly, cumbersome, and potentially harmful sequelae. Certain findings, referred to as geriatric syndromes, negatively impact QOL and have higher incidence and prevalence in OAs than in younger clients. Thus, the APRN interested in preserving QOL must be able to evaluate and manage these geriatric syndromes.

B. In this section, we explore geriatric syndromes both as a general concept and as a set of specific findings that cannot be pared down to a single disease, providing the APRN a framework for QOL-forward care planning. The list of individual geriatric syndromes is not codified; instead, experts expand or prune the list as they translate new knowledge into evidence-based practice. Because an exhaustive exploration of each geriatric syndrome is beyond the scope of this section, we explore eight specific syndromes, including incontinence, falls, delirium/altered mental status (AMS), weight loss, depression, pain, dementia, and impairment in activity of daily living (ADL) or instrumental activity of daily living (IADL).

DEFINITION

A. Signs and symptoms wherein the etiology is poorly understood or multifactorial.

B. Geriatric syndromes occur widely in the OA population.

C. While there is no codified definition of geriatric syndromes, this working definition highlights the key elements that set syndromes apart from diseases.

D. Approaching any geriatric syndrome simply as a syndrome that happens more frequently in OAs instead of a special codification is helpful both diagnostically and prognostically.

THE RULE OF FOURTHS

A. The Rule of Fourths "clarifies that changes often attributed to normal aging by the general public (and sometimes by medical professionals) are caused by disease, disuse, misuse, and physiology—about one-fourth of the time for each" (Warshaw et al., 2022, p. 4).

1. Incomplete to place the blame for geriatric syndromes solely on expected, physiologic changes of age.

2. Each of the four elements of decline in the OA provides a specific evaluation and management framework that reduces the APRN's decision fatigue (Table 5.1).

DIAGNOSTIC IMPLICATIONS

A. All syndromes are diagnoses of exclusion.

B. Syndromes be both frustrating and advantageous for diagnostician and the client alike:

1. Etiologic mystery and debate about their diagnostic criteria precludes a precision that makes for satisfying and economical identification of disease.

2. Syndromes hold clinicians accountable to a comprehensive workup that leaves no part of the differential unexamined as well as to a surrender of true multicomplexity where palliation is the most appropriate intervention.

C. In functionally independent OAs with low disease burden, any specific geriatric syndrome presented as an acute chief complaint should prompt a workup that assumes its etiology may be found.

D. Presuming that a syndrome is the diagnosis because the client is classified as geriatric, without ruling out a comprehensive differential diagnosis, is dangerous and unethical.

E. To help mitigate the risk of iatrogenic morbidity and mortality due to erroneous medical decision-making, a differential diagnosis is offered for the geriatric syndromes discussed in this section.

DIFFERENTIAL DIAGNOSES

A. In multicomplex or frail OAs, the presentation of geriatric syndromes is more clinically complex and frustrating.

B. Thorough workup is sometimes necessary, especially when the chief complaint is new or acutely worsened concomitantly with a chronic disease exacerbation.

C. Ruling out a reversible or treatable etiology can be paramount to maintaining QOL and reducing morbidity and mortality. As multicomplexity and frailty progress, the burdens of medical investigation can outweigh the benefits of identifying a specific etiology.

D. A palliative, symptom-based approach to interventional planning is most appropriate.

1. Diagnosing a syndrome instead of a disease is both merciful and helpful.

2. Client is comforted where curative measures would likely increase the risk of suffering.

PROGNOSTIC IMPLICATIONS

A. Clinicians remain uncomfortable and unsystematic in approaching prognosis.

B. Several standardized instruments that quantify life span based on health history and status exist; however, they can be cumbersome when used in the context of a busy clinic.

C. Prognostic scales that are useful when limited life span has been established, such as the Palliative Performance Scale, are

TABLE 5.1 THE RULE OF FOURTHS IN PRACTICE

	Disuse	Misuse	Pathology	Physiology
Context	Immobility begets immobility.	Continual injurious use of body causes permanently damage.	Illness and injury have a deleterious effect on the body. Chronic disease creates a persistently deleterious state, if insidious. Medications may cause more harm than good at a certain point in frailty.	Expected changes in the aging body.
Clinical Goal	Regain functional baseline.	Maintain current function.	Treat disease process.	Adapt to change.
Appropriate Intervention	Rehabilitation therapy aimed at regaining functional baseline given fair-to-good rehabilitation prognosis.	Rehabilitation therapy aimed at stopping misuse, maintaining current functional state, and managing client expectations considering guarded rehabilitation prognosis.	Workup and medical intervention.	Environmental adaptation and expectation management.
Questions to Consider	How can functional independence be regained with physical, occupational, or speech therapy?	How can further functional dependence be prevented with physical, occupational, or speech therapy? How can I help this client accept maintenance of current function as a goal since improvement is unlikely?	Can I find a specific cause? If not, is this presentation worsened by a disease or medical intervention present? Is it time to deprescribe rather than add an intervention?	How would a change in physical environment improve the lived experience? Would a personal care aide improve quality of life? Is occupational therapy consultation warranted?

TABLE 5.2 GERIATRIC SYNDROMES AND ASSOCIATED RISKS

Hospitalization Risk	Morbidity Risk	Mortality Risk
Geriatric syndromes increase risk of complications and functional decline of hospitalized OAs with fewer geriatric resources (Meyer et al., 2020) and increase the overall 30-day readmission risk. This increases the overall risk of hospital-associated functional decline that is noted across the OA population despite individual risk factors.	The presence of a geriatric syndrome increases the risk for other geriatric syndromes and general decline of comorbid conditions (Bare et al., 2021). Community-dwelling OAs with geriatric syndromes are less likely to self-report favorable health status (Kwon et al., 2007), placing them at higher risk for motivation loss, disordered mood, or social isolation, all of which are well-documented risks of further morbidity and mortality.	Malnutrition, impaired homeostasis (a symptom of frailty), and impaired cognition influence mortality risk more than any other geriatric syndromes (Kane et al., 2012). The presence of geriatric syndromes in the frail or high-acuity OA increases the risk of mortality compared with OAs of fair-to-good health status (Meyer et al., 2020).

OA, older adult.

often too blunt in clients not currently being considered for hospice referral.

D. An understanding of life expectancy informs the prevention and care plan:

1. New or multiple geriatric syndromes indicate a higher risk for hospitalization, morbidity, and mortality.

2. Identifying new or multiple geriatric syndromes is an indication of poor health status and overall prognosis (Table 5.2).

E. Early identification and intervention of geriatric syndromes—especially frailty, sarcopenia, malnutrition, and impaired cognition—is critical in preventing untimely mortality and worsening morbidity.

F. Clients with frailty, itself a geriatric syndrome, or multicomplexity who present with other concomitant geriatric syndromes likely have fair, guarded, or poor overall prognoses.

1. Clients should be offered an advance care planning encounter to review whether a curative or palliative plan of care best aligns with the goals of care and overall prognosis.

2. Appropriate to establish overall life expectancy using a standardized instrument, such as the *QMortality-2017 Risk Calculator* (qmortality.org/).

PREVENTION

A. Prevention of geriatric syndromes can improve QOL by maintaining functional status and reducing risk of morbidity and mortality (Table 5.3).

TABLE 5.3 GERIATRIC SYNDROME PREVENTION GOALS AND STRATEGIES BY LEVEL

	Level			
	Primary	**Secondary**	**Tertiary**	**Quaternary**
Goal	Create environmental and behavioral changes that reduce harm risk.	Discover treatable diseases and risks and treat early.	Prevent worsening of chronic disease and development of new comorbidities.	Prevent iatrogenic injury and suffering.
Strategies	Implement client education on fall mitigation strategies, expected versus pathologic and physiologic changes in aging, activity/nutrition requirements of the aging body, and community resources.	Screen clients regularly for geriatric syndromes. In addition to specific syndrome screenings discussed later, the Rapid Geriatric Assessment offers unified screening for frailty, sarcopenia, disordered nutrition, and cognitive impairment (Little, 2017).	Counsel clients and caregivers on self-management, deprescribe where possible to avoid side effects from high-risk medications, and implement rehabilitation and skilled services where rehabilitation potential is favorable.	Avoid medical interventions and screenings that offer more risk than benefit or that have low evidence of benefit.

B. Awareness of health status, whether stable or declining, can inform shared decision-making and empower clients to self-manage and self-determine.

FRAILTY AND SARCOPENIA

DEFINITION

A. The frail body is prone to deteriorate more rapidly or to a greater severity than a body that has experienced expected physiologic aging.

B. Likely the permanent effect of chronic inflammation over many years.

1. Chronic inflammation upregulates key inflammatory markers that lead to muscle wasting, hormonal dysregulation, and impaired immunity.

2. Over time, the body simply loses the ability to manage stress.

3. Chronic diseases, especially metabolic diseases, contribute to chronic inflammation exposure.

4. Additionally, sarcopenia, the reduction of lean muscle mass, leads to weakness, exhaustion, and poor reserves, eventually tipping the client into frailty.

SCREENING

A. Identifying frail OAs is an evolving concept dominated by two major models. Both models acknowledge that frail adults are particularly vulnerable to morbidity and mortality because their bodies are no longer able to maintain homeostasis when stressed. While they differ in their diagnostic criteria, each may be uniquely useful based on the specific clinical scenario (Cesari et al., 2014). Fried et al. (2001, 2021) validated a phenotype model that identifies frailty as a threshold of specific progressive signs and symptoms that point to homeostatic dysregulation. Rockland et al. (2005) validated an index model that categorizes clients as frail when they accumulate a threshold of chronic disease and functional dependencies, also framed as *multicomplexity*. Prefrailty is a precursor to frailty and may be more readily reversible. Careful attention to screening and rehabilitation for prefrailty and sarcopenia may improve the chance of the older adult returning to a state of robustness or delaying the onset of frailty (Tables 5.4 and 5.5; Warshaw et al., 2022).

FUNCTIONAL DEPENDENCE

A. Functionally independent older adults (OAs) can maintain activities of daily living (ADLs) and instrumental activities of daily living (IADLs) without assistance. ADLs are self-care tasks that must be completed to maintain control over bodily functions and needs. IADLs are self-care tasks that must be completed to participate in a community or society. Functional dependence, or the loss of ability to self-manage ADLs and IADLs without assistance, can be temporary or permanent, and it may carry significant emotional, financial, and health consequences for the OA and social system. Clients with functional dependence should be assessed for neglect and self-neglect as it may be motivation or social support-related rather than physical (Pavlou & Lachs, 2006; Pickens et al., 2006). Table 5.6 describes ADLs and IADLs, their respective screenings, and suggested management strategies.

FALL

DEFINITION

A. The World Health Organization (2021) considers any "event which results in a person coming to rest inadvertently on the ground or floor or other lower level" to be a fall.

B. Sequelae of falls include fractures—especially hip, wrist, arm, and ankle fractures—traumatic brain injury, hospitalization, fear of falling, functional decline, and death.

C. Falls are preventable and are not an inevitable part of aging. Identifying risk factors is critical to fall prevention because most falls are the result of multiple, concomitant risk factors (CDC, 2021).

RISING ANNUAL INCIDENCE

A. According to the CDC (2021), falls are the leading cause of injury-related morbidity *and* mortality for American adults

TABLE 5.4 SCREENING FOR FRAILTY

Definition (Noncodified)	Loss of ability to maintain or efficiently return to homeostasis
Clinical Significance	Reduced clinical reserve due to chronic homeostatic dysregulation High risk for hospitalization, morbidity, and mortality with minimal stress High risk for postdischarge disability Possibly reversible with appropriate intervention, more likely with prefrailty
Etiology	Phenotype model: chronic inflammation effects over time Index model: accumulation of chronic diseases and functional dependencies (van Kan et al., 2008)
Epidemiology	Frailty: 15% community-dwelling OAs (95% CI, 14%–16%) 45% of community-dwelling OAs (95% CI, 44%–47%) Higher prevalence at older ages, among females, racial and ethnic minorities, those in supportive residential settings, and persons of lower income (Bandeen-Roche et al., 2015)
Screening Tool	Phenotype model: FRAIL Scale (van Kan et al., (2008) • Fatigue: presence of fatigue most of the time over the past month • Resistance: difficulty climbing 10 steps without resting • Ambulation: difficulty walking one block • Illness: 5+ illnesses • Loss of weight: more than 5% of body weight in 1 year • Scoring: robust: 0 points, prefrail 1–2 points, frailty 3+ points Index type: DAI (Rockwood et al., 2005)
Goals of Care	Reduce mortality risk. Reverse frailty or prefrailty. Prevent tertiary admissions. Prevent/slow progression of other geriatric syndromes. Prepare the client and their family for poor prognosis and encourage. anticipatory grief.
Management	Advance care planning. Manage comorbidities. Deprescribe where appropriate. Suspend prevention activities where the benefit requires life expectancy beyond 1 to 5 years per client prognosis. Counsel on early signs of infection in frail clients (behavior changes, etc.). Establish open communication surrounding prognosis. Consult rehabilitation therapy if rehabilitation prognosis remains guarded-to-fair. Encourage comfort protocols and refer to hospice when appropriate.

DAI, deficit accumulation index; OAs, older adults.

TABLE 5.5 SCREENING FOR SARCOPENIA

Definition (Noncodified)	Age-related loss of muscle mass which presents clinically as weakness and reduced physical performance absent a pathologic etiology			
Clinical Significance	Significantly increases risk for frailty, disability, poor quality of life, falls, and mortality; presents in normal, under- or overweight OAs			
Etiology	Leading hypothesis Reduced synthesis of proteins due to chronic inflammation; other hypotheses include:			
	Physiologic	**Pathologic**	**Misuse**	**Disuse**
	Reduced androgen production Reduced motor neurons in the spinal cord	Chronic disease Increased insulin resistance	Protein intake, less than the requirements	Reduced mobility Reduced physical activity
Screening Tool	Dynamometer Grip Strength Test			
Evaluation	Diagnostic confirmations, either: • DEXA scan indicating low muscle mass, or • Bioelectrical impedance, using adjusted appendicular muscle mass for height: 2 *SD* below the mean for healthy adults diagnostic of sarcopenia • Albumin, serum, below normal limits			

(*continued*)

TABLE 5.5 SCREENING FOR SARCOPENIA (*CONTINUED*)

Goals of Care	Rule out disease/iatrogenic cause of weakness and reduced physical performance. Resolve misuse or disuse. Adapt to physiologic changes. Monitor regularly for frailty.
Management	Monitor weight regularly. Rehabilitation therapy for gait training and balance support. Social work for optimization of social support. Counsel on frailty risk and symptoms to report.

DEXA, dual-energy x-ray absorptiometry; OA, older adults.

TABLE 5.6 ADL AND IADL

Type	ADLs	IADLs
Self-care Tasks	Transferring Dressing Bathing Eating Toileting Continence	Housekeeping Food preparation Medication management Laundry Money management Phone use Transportation use Shopping
Screening	Katz Activities of Daily Living Assessment (Shelkey & Wallace, 2000)	Lawton Instrumental Activities of Daily Living Assessment (Graf, 2008)
Risk Factors	Cognitive impairment Mobility impairment Multicomplexity Hospitalization Incontinence Pressure ulcers Frailty	
Goals of Care	Prevent further functional loss. Return to functional independence where possible. Support independent function with adaptations. Maintain.	
Management	Order physical, occupational, or speech therapy consult where rehabilitation potential is not poor.	Consider occupational therapy consult where rehabilitation potential is not poor.
	Order skilled nursing home safety assessment under home health benefit if unable to make a house call. Order social work consult to assess social support system and financial means available for supportive services and to recommend appropriate support programs. Support maintenance of functional independence adaptive equipment/supplies, hiring personal care aides, and modifying the environment to abilities.	

ADL, activity of daily living; IADL, instrumental activity of daily living.

over 65. The incidence of falls has risen by 30% since 2007, contributing to more than 30,000 annual deaths, eight million annual injuries, three million ED visits, and $50 billion in healthcare expenditures. These staggering statistics do not consider the social sequelae of falls for individual older adults (OAs); a traumatic fall can mean forced retirement, loss of home, or separation from family. Given the tremendous burden and risk of falls, prevention is of paramount importance.

SCREENING

A. Encounter-based screenings (Table 5.7).

B. Stopping Elderly Accidents, Deaths, and Injuries (STEADI).

1. The CDC publishes STEADI to healthcare providers at no cost (www.cdc.gov/steadi/index.html).

2. As a comprehensive fall prevention program, STEADI aims to reduce the annual incidence of falls in OAs and by extension lower associated morbidity, mortality, and health system utilization costs. It offers standardized assessments, interventions, and counseling tools that identify and reduce multiple risk factors (Table 5.8).

DEMENTIA, DEPRESSION, AND DELIRIUM

A. While dementia, depression, and delirium may appear as discrete geriatric syndromes, their clinical presentations can often masquerade as one another. Profound depression can result in pseudodementia. Early displays of confusion in delirium might dismissed as a "bad day" in the ebb and flow of dementia manifestations. The self-neglect, social apathy, or

TABLE 5.7 ENCOUNTER-BASED SCREENINGS

Subjective	Objective
Brief Questions Include the following questions in the intake or clinical interview. Positive answers should involve further inquiry into risk factors, injury, urgent or emergent treatment, tertiary admissions, and prevention plan deficits. • Have you fallen since the last visit? • Have you fallen in the past year? • Are you afraid you are going to fall?	**TUG** • TUG ≥ 12 seconds indicates high risk of fall. • This test evaluates functional mobility by measuring the time required to stand from a chair with an armrest, walk 3 m (10 feet) away from, and return to sitting in the same chair. **30-Second Chair Stand Test** • Inability to rise from a chair without using one's arms indicates high risk of fall. • This test evaluates lower extremity strength and balance by timing a client standing from a seated position using only their legs.

TUG, Timed Up & Go.

TABLE 5.8 STOPPING ELDERLY ACCIDENTS, DEATHS, AND INJURIES

Risk Type	Environmental	Pharmacologic	Pathologic	Physiologic
Goal	Modify environment to reduce fall risk	Reduce iatrogenic fall risk	Treat and encourage adaptation to pathologies that increase fall risk	Adapt to expected changes of aging that increase risk of falls.
Strategies	Make a home visit or prescribe a skilled nurse home safety assessment using the home health benefit to assess for: • Tripping hazards: broken stairs, throw rugs, low furniture, clutter, and assistive devices not used. • Slipping hazards: slippery surfaces and improper footwear. • Balancing hazards: lack of grip bars to bathroom, shower, and stairs; and commonly used items in high cabinets requiring use of step stool. • Visibility hazards: poorly lit rooms and stairwells.	Reconcile the medication list for high fall risk medications regularly. Deprescribe or gradually dose-reduce tranquilizers, sedatives, anticholinergics, or antidepressants where possible.	Neuropathy, paresthesia, or pain to feet: Implement regular foot exams, appropriate footwear, and pain management. Generalized weakness to lower extremities causing gait or balance abnormalities: Prescribe rehabilitation therapy and assistive devices. Cognitive impairment: Coach client and their caregivers to anticipate impulsivity and to routinize the day. Delirium: Coach client and caregivers to monitor for early signs of delirium. Substance abuse: Counsel the client to quit and enroll in detoxification program.	Presbyopia: Ensure annual eye examination and appropriate corrective lenses are in place and consider distance-only glasses instead of bifocals for outdoor activities. Osteopenia/Osteoporosis: Implement calcium supplementation, vitamin D monitoring and supplementation, weight-bearing exercises, and DEXA scan. Proprioception changes: Implement weight-bearing exercises and rehabilitation therapy if warranted.

DEXA, dual-energy x-ray absorptiometry.

flattened affect of a person with undiagnosed dementia may appear as depression. Thus, understanding how each may present like the others is critical to timely diagnosis and treatment.

B. The scope of this section is to establish a cognitive framework for dementia, depression, and delirium. Definitions, risk factors, etiologies, and clinical presentations of each are alongside each other so that similarities and differences are plain (Table 5.9). Screening and treatment options specific to older adults will be briefly discussed. It is beyond the scope of this section to explore any of them fully. For a more robust discussion on dementia, see Chapter 23, Neurologic Guidelines. For a more robust discussion on depression, see Chapter 25, Psychiatric Guidelines.

POOR APPETITE AND UNINTENTIONAL WEIGHT LOSS

A. Unintentional weight loss may be attributed to gradually reduced appetite, sarcopenia, poor absorption, malignancy, cognitive decline, functional dependence without support, neglect by self or others, and other factors. Unintentionally losing 5 lbs over 3 years is a risk factor for mortality in community-dwelling (Wannamethee et al., 2005). Any unintentional loss of 5% of total body weight in 3 months or 10% in 6 months should prompt thorough investigation or consideration of hospice referral, depending on health status (Table 5.10).

TABLE 5.9 RISK FACTORS, ETIOLOGIES, AND CLINICAL PRESENTATION OF DEMENTIA, DEPRESSION, AND DELIRIUM

	Dementia	Depression	Delirium
Onset	Gradual (weeks to months)	Gradual (weeks to months)	Sudden (hours to days)
Duration	Progressive	Chronic	Acute
Characteristics	Progressive working and shortterm memory loss that worsens into: • Long-term memory loss • Executive functioning loss • Sensorimotor loss • Functional dependence • Disordered communication (see *DSM-5* for specific criteria by dementia type)	Anhedonia or depressed mood sustained for greater than 2 weeks and with at least four of the following symptoms: • Unintentional weight change • Slowness of thought or mind • Fatigue • Feeling worthless or guilty • Fogging thinking or indecisiveness • Passive death wish, suicidal ideation with or without plan, or suicide attempt (see *DSM-5*)	Acute mental status change caused by a medical condition or an adverse effect of pharmacologic interventions, reversible, manifesting as acutely disordered or disturbed: • Concentration or focus • Thinking patterns • Sensorium, especially tactile or visual hallucinations • Behavior, especially agitation with care and pressured speech (see *DSM-5*)
Aggravating Factors	Inadequate functional and social support, poorly managed comorbidities	Complicated grief, hopelessness, overwhelming stress, isolation/ loneliness, certain medications	UTI or PNA, which are the leading causes of infectious delirium in OAs
Relieving Factors	Symptom-based interventions	Appropriate therapeutic plan	Removal of causative stimulus
Treatment	Medications Lifestyle modifications Environmental modifications Functional support	Medications Psychotherapy Lifestyle modifications	Treatment of causative medical condition or deprescribing causative agent
Screening Instruments	Mini-Cog Mini-Mental State Examination Montreal Cognitive Assessment St. Louis University Mental Status Exam	Patient Health Questionnaire 9 Geriatric Depression Scale	bCAM
Evaluation and Management Pearls	Rule out other. Ensure baseline cognitive status is established and documented to ensure timely diagnosis of any delirium developed. Screen for depression to ensure that concomitant disease is properly treated. Treat dementia and ensure supportive care and a socialization plan are in place to prevent development or worsening of depression. Trend progression using the same instrument used to establish baseline.	Poorly treated or undiagnosed depression can cause pseudodementia of depression. Consider trending mental status exam of choice for improvement through depression treatment in OAs with established or suspected memory loss. Wean serotonin modulators gradually to prevent discontinuation side effects and, rarely, delirium. Distinguish psychosis from delirium by assessing for auditory hallucinations, which are seen more in psychiatric disorders, versus tactile or visual, which are seen more in delirium. Consider screening for and monitoring concomitant anxiety in clients with confirmed or suspected depression. Trend response to treatment using the same instrument used to establish baseline. SSRIs are low risk and often used as first line for OAs with depression.	Delirium increases risk of mortality; timely identification of causative stimulus is critical. Do not delay treatment for cultures if infection is highly suspected and cultures are difficult to obtain because the client could deteriorate rapidly while awaiting collection. Treat empirically per guidelines and adjust as needed. Fever is a late sign of infection in an OA; do not rely on pyrexia before clinical suspicion of infection is raised in an OA whose behavior is "off." Always reconcile the medication list when working up delirium, assessing for new prescriptions, changed doses, and unreported supplements. Terminal delirium is a common phenomenon at the end of life and can be distressing; it is the only time symptoms may be treated without an exhaustive search for causative stimulus.

bCAM, brief confusion assessment method; *DSM-5, Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition;* OA, older adult; PNA, pneumonia; SSRI, selective serotonin reuptake inhibitor; UTI, urinary tract infection.

TABLE 5.10 UNINTENTIONAL WEIGHT LOSS

Definition	Clinically significant weight loss includes: • Loss of 5% total body weight in 3 months • Loss of 10% total body weight in 6 months			
Clinical Significance	Increases risk for geriatric syndromes; may indicate neoplasm or increased risk of mortality			
Etiology	**Physiologic**	**Pathologic**	**Misuse**	**Disuse**
	Reduced taste Reduced smell Reduced elasticity of the stomach fundus Reduced absorption Increased satiety	Impaired cognition Depression CHF-related reduced cardiac output Neoplasm, especially of colon Polypharmacy Dysphagia Chemotherapy or radiation Hyperthyroid	Poor appetite	Functional dependence Inability to access or prepare food
Screening	Monitor weight/BMI annually.			
Evaluation	Monitor weight when clinical complaints or exams prompt concern for unintended weight loss. Inquire about weight changes in the clinical interview at initial, annual wellness, and chronic management encounters, in addition to when appetite and weight loss are the chief complaint(s).			
Goals of Care	Rule out disease/iatrogenic cause. Resolve misuse or disuse. Adapt to physiologic changes. Reduce barriers to adequate intake by mouth.			
Management	Obtain labs: TSH (to rule out pathologic or iatrogenic hyperthyroidism), CMP (low albumin 2/2 poor hepatic absorption or by mouth intake of protein), and fecal occult blood test x 2 to 3 (taken on different days to rule out malignant neoplasm of the colon). Optimize the management of comorbidities. Deprescribe offending or unnecessary medications. Monitor weight monthly. Consult rehabilitation therapy for dysphagia, impaired mobility, and environmental adaption assessment. Consult social work for optimization of social support. Consult a registered dietitian for nutritional recommendations. Encourage pleasure feedings.			

BMI, body mass index; CHF, congestive heart failure; CMP, comprehensive metabolic panel; TSH, thyroid stimulating hormone.

B. Conventional wisdom might lead many clients and their families to inquire about appetite stimulants or nutritional supplementation; clinically significant weight loss can be alarming. The Choosing Wisely campaign recommends against the use of megestrol acetate because it increases risk of thromboembolism, fluid retention, and death. They also caution against the use of high-protein supplements given a lack of enhanced quality of life or function. Instead, it is important to uncover any reversible etiologies and encourage pleasure feedings (Table 5.11).

PAIN

A. Poorly managed pain reduces functional independence and places clients at risk for development of other geriatric syndromes, such as poor appetite, weight loss, sarcopenia, frailty, incontinence, and impaired mobility.

B. Older adults (OAs) may underreport pain for several reasons:

1. Belief that pain is a part of "normal aging."
2. Lack of trust in the efficacy of medical interventions.
3. Fear of pain-relieving pharmaceuticals.
4. Age-related perceptions of pain.
5. Knowledge deficit of available interventions.
6. Cognitive inability to report.

C. Management of pain includes a deft understanding of cultural expectations, mastery of pain types and effective interventions by type, and attention to possible comorbidities such as depression, opiate misuse/dependence/addiction, and pain-related disuse of the affected site.

1. In cognitively intact adults, pain may be assessed using the PQRST (P, Palliative or precipitating factors; Q, quality of pain; R, region or radiation of pain; S, subjective descriptions of pain; T, Temporal nature of pain [time of pain]) mnemonic along with the number or faces scales to trend severity.
2. The Pain Assessment in Advanced Dementia (PAINAD) has been developed and verified for clients who are cognitively impaired or who cannot communicate clearly. It assesses objective findings appropriate to the OA who cannot self-advocate: breathing quality, vocalization quality, facial expression, body language, and consolability (Paulson et al., 2014).

TABLE 5.11 POOR APPETITE

Definition	Reduction in drive for oral intake of food and fluids			
Clinical Significance	Increases risk of malnutrition, involuntary weight loss, sarcopenia, constipation and related incontinence, and falls			
Etiology	**Physiologic**	**Pathologic**	**Misuse**	**Disuse**
	Reduced taste Reduced smell Reduced elasticity of stomach fundus Reduced absorption Satiety due to increased cholecystokinin	Impaired cognition Depression Polypharmacy Multicomplexity Secondary gastroparesis Pain Dysphagia	Poor oral hygiene Delayed gastric emptying 2/2 large meals Satiety due to increased adiposity releasing more leptin	Impaired mobility Reduced social support
Evaluation	Inquire about appetite changes in the clinical interview at initial, annual wellness, and chronic management encounters, in addition to when appetite and weight loss are the chief complaint(s).			
Goals of Care	Rule out disease/iatrogenic cause. Resolve misuse or disuse. Adapt to physiologic changes. Monitor regularly for clinically significant weight loss.			
Management	Consider labs as listed in Unintentional Weight Loss Management in Table 5.10. Optimize the management of comorbidities. Deprescribe offending or unnecessary medications. Monitor weight monthly. Consult rehabilitation therapy for dysphagia, impaired mobility, and environmental adaption assessment. Consult social work for optimization of social support. Consult a registered dietitian for nutritional recommendations. Counsel on behavioral interventions. Encourage pleasure feedings.			

INCONTINENCE

A. In community-dwelling older adults (OAs) up to age 85, urinary incontinence (UI) is more prevalent in females than in males, with prevalence directly correlating to age. After age 85, UI is equally burdensome for both males and females. Childbearing, surgery, obesity rates, and diabetes prevalence may contribute to the difference before age 85, but the onset of clinically significant prostate pathologies causing obstruction-based urgency or weakened sphincter tone incidence brings the male-to-female ratio with UI to nearly 1:1 (Shamliyan et al., 2009). Offermans et al. (2009) found a median prevalence rate of 58% for nursing home residents who were found to be at greater risk than community-dwelling OAs because of age, cognitive function, dementia, functional quadriplegia, and immobility, to name a few of the 45 setting-specific risk factors identified.

DEFINITION

A. UI:

1. Stress incontinence: complaint of involuntary loss of urine on effort or physical exertion, including sporting activities or on sneezing or coughing.

a. Urgency incontinence: complaint of involuntary loss of urine associated with urgency.

b. Mixed incontinence: complaints of both stress and urgency UI, that is, involuntary loss of urine associated with urgency and also with effort or physical exertion, including sporting activities or on sneezing or coughing.

c. Overflow incontinence: complaint of UI in the symptomatic presence of an excessively (over-) full bladder (no cause identified); also called "Overactive Bladder Syndrome."

d. Functional incontinence: UI related to physical barrier to using available toilet (e.g., unable to ambulate to the restroom due to loss of ambulatory or transferring independence).

B. Fecal incontinence:

1. Urgency incontinence: complaint of an involuntary loss of feces (flatus) associated with fecal urgency.

2. Passive incontinence: complaint of involuntary soiling of liquid or solid stool without sensation or warning.

3. Overflow incontinence: complaint of involuntary loss of stool due to an overfull rectum or fecal impaction.

4. Functional incontinence: fecal incontinence related to physical barrier to using the toilet (e.g., unable to ambulate to restroom due to loss of ambulatory or transferring independence).

SCREENING

A. Regular screening is recommended for all OAs to compensate for well-documented underreporting and underscreening (Wenger et al., 2007; Visser et al., 2012). Standardized instruments can be easily incorporated into annual wellness visits and previsit intake workflows completed by the medical assistant or office nurse. Standardizing screening workflows prevents incontinence from being attributed to "normal aging". Specific instruments are discussed in Table 5.12.

B. Many OAs, especially females, incorrectly attribute urinary incontinence to "normal aging" due to community messaging (Kim et al., 2019; Orsal et al., 2020; Visser et al., 2012). Standardized screening and shared decision-making

TABLE 5.12 SCREENING TOOLS FOR URINARY INCONTINENCE

Instrument	Evidence	Scope	Suggested Use
General Screening Questions (Holroyd-Leduc et al., 2008) *Unspecified UI* • "Have you had any problems with bladder or urine control?" • "Do you ever leak urine when you don't want to?" *Stress incontinence* • "Do you ever leak urine coughing, sneezing, lifting, walking, or running?" *Urgency incontinence* • *"Do you experience such a strong and sudden urge to void that you lead before reaching the toilet?"*	*Strong interrater reliability* K = 0.8 (95% CI, 0.3–0.9) *Stress UI: strong LR*[a] +LR 2.2 (95% CI, 1.6–3.2) –LR 0.39 (95% CI, 0.25–0.61) *Urgency: strong LR*[b] +LR 4.2 (95% CI, 2.3–7.6) –LR 0.48 (95% CI, 0.36–0.62)	UI identification	Online check-in, inperson check-in, or preencounter MA/LPN/RN intake New client visits Acute GYN or GU visits Annual wellness visits
ICIQ-UI SF (Hajebrahimi et al., 2012) www.iciq.net	*Strong interrater reliability* K = 0.7 *Stress UI* Sensitivity 0.636 (95% CI, 0.466–0.778) Specificity 0.826 (95% CI, 0.36–0.89) *Urge UI* Sensitivity 0.525 (95% CI, 0.402–0.645) Specificity 0.953 (95% CI, 0.871–0.984) Mixed UI Sensitivity 0.84 (95% CI, 0.653–0.936) Specificity (95% CI, 0.689–0.85)	UI identification UI QOL impact	Online check-in, inperson check-in, or preencounter MA/LPN/RN intake New client visits Acute GYN or GU visits Annual wellness visits HPI Complaint communicated during APRN interview outside of preencounter intake
IIQ (Uebersax et al., 1995) www.ohsu.edu/sites/default/files/2019-06/Female-Urology-Questionnaire-7.pdf	The short form is equally as reliable as the longer 30-item or 19-item forms, and because it is less cumbersome to implement it may be more clinically valuable.	UI QOL impact	Online check-in, in-person check-in, or preencounter MA/LPN/RN intake New client visits with established UI Chronic UI visits Annual wellness visits HPI Complaint communicated during APRN interview outside of preencounter intake Positive UI identification screening

[a]Posttest probability negatively correlates with age. [b]Posttest probability positively correlates with age.

GYN, gynecology; ICIQ-UISF, International Consultation on Incontinence Questionnaire-Urinary Incontinence Short Form; IIQ, Incontinence Impact Questionnaire; K, kappa test; GU, genitourinary; HPI, history of present illness; LPN, licensed practical nurse; LR, likelihood ratio; MA, medical assistant; QOL, quality of life RN, registered nurse; UI, urinary incontinence.

can help disabuse clients of harmful misconceptions of "normal aging."

C. Social anxiety and misconceptions about "normal aging" lead to poor reporting and undertreatment. This underreporting may be worsened by lack of screening in primary care settings (Orsal et al., 2020; Visser et al., 2012).

PLAN

A. Acute presentation: New or worsened UI in the OA without established incontinence or acutely worsened incontinence in the OA with established chronic incontinence should raise clinical suspicion of infection. New or worsened bowel incontinence should prompt a rectal history and examination to evaluate for neoplasm and neurologic alarm signs, as well as sphincter tone loss or fecal impaction (Table 5.13; Warshaw et al., 2022).

B. Chronic presentation: Equipping OAs and their caregivers with medical, behavioral, and environmental controls for incontinence of bladder or bowel improves quality of life and reduces social distress. Poorly managed incontinence increases the risk for pressure ulcers, malnutrition, falls, delirium, and functional decline (Table 5.13; Kim et al., 2019).

CONCLUSION

A. Geriatric syndromes place older adults (OAs) at risk for hospitalization, morbidity, and mortality. Early identification and management of geriatric syndromes produces the best outcomes, so incorporating regular screening into clinical practice is key. APRNs who give attention to the identification of geriatric syndromes while dispelling myths about normal aging can help OAs to self-determine, self-manage, and experience improved quality of life while reducing any caregiver role strain their lay caregivers may be experiencing.

TABLE 5.13 OBJECTIVE-BASED CARE PLANNING FOR INCONTINENCE

Goal	Rationale	Management
Identify appropriate subtype and etiology.	Discovery of precise pathophysiologic mechanism of incontinence allows for planning targeted treatment and clinical endpoint.	Standardized screening instruments Physical exam Targeted diagnostics: UA/UC, PSA, and bladder US for PVRV
Rule out "must-not-misses."	Early identification of alarm signs and symptoms reduces risk for morbidity and mortality.	UI: rule out infection, prostate malignancy, cystocele, nocturia, etc., refer as appropriate FI: rule out cauda equina syndrome, impaction, and rectocele
Treat reversible pathology/identified etiology.	Untreated reversible pathology causes place the client at risk for further morbidity or mortality and preventable social distress.	Urology/GYN referral/procedure Pharmacologic interventions Behavioral modifications: dietary modification, bladder training, toileting schedule, etc. Pelvic floor rehabilitation
Resolve social and functional barriers.	Modifying the environment for an OA with reduced mobility increases functional independence and reduces social anxiety.	Environmental modifications: urinal, bedside commode, grab bars, etc. Incontinence wearables

FI, fecal incontinence; GYN, gynecology; OA, older adult; PSA, prostate-specific antigen; PVRV, postvoid residual volume; UA, urinalysis; UC, urine culture; UI, urinary incontinence; US, ultrasound.

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CHAPTER 6

PAIN MANAGEMENT GUIDELINES

Kathleen Bradbury-Golas

ACUTE PAIN

DEFINITION

A. Acute pain is defined as pain of a short, limited duration usually the result of an injury, surgery, or medical illness (disease/inflammation) that generally arises from tissue injury; however, it may be experienced even with no identifiable cause. Acute pain usually resolves when the tissue injury improves with the healing process. Most acute pain resolves in less than 6 weeks or less than 3 months.

B. General statements about pain:

1. Pain is a distressing sensory and emotional experience often associated with actual or potential tissue damage. It is an actual physical discomfort and the result of many factors including physical, emotional, psychosocial, and spiritual, along with the cultural social contexts that influence how the client expresses/tolerates the pain. Therefore, pain is multidimensional.

2. Pain is what the client says it is, "totally subjective." Clients do not always verbalize their pain but may express it in nonverbal ways. Therefore, a provider must assess the client using various methods.

3. The initial assessment of pain is to characterize the pathophysiology of the pain, identify its cause, determine its intensity, and evaluate its impact on the client's ability to function.

INCIDENCE

A. Acute pain is the most common reason for self-medication and presentation for treatment in the healthcare system. Acute pain is very individual, and if not treated properly it can have devastating physiologic and psychological effects. Because everyone experiences some type of acute pain, it is very difficult to determine the incidence and prevalence of pain. Because pain is very subjective, the client care plan needs to be individualized to meet the client's needs. Proper treatment of acute pain could prevent the development of some types of chronic pain syndromes.

PATHOGENESIS

A. Acute pain is usually the result of stimulation of the sympathetic nervous system.

B. Types of pain:

1. Nociceptive pain: normal response to peripheral tissue injury or damage (sprains, fractures).

2. Neuropathic pain: originates from injury or dysfunction of the peripheral nervous system or central nervous system structures or both (postherpetic neuralgia, diabetic neuropathy).

3. Inflammatory pain: nociceptive pain with a localized immune response due to proinflammatory mediators (arthritis).

4. Centralized/functional pain: peripheral and central sensitization with no detectable origin (fibromyalgia, headaches).

COMMON COMPLAINTS

A. Pain at the specific site.
B. Increased heart rate.
C. Increased respiratory rate.
D. Elevated blood pressure.
E. Sweating.
F. Nausea.

OTHER SIGNS AND SYMPTOMS

A. Urinary retention.
B. Dilated pupils.
C. Pallor.
D. Anxiety/depression.

SUBJECTIVE DATA

A. Elicit location of pain.
B. Note effects of pain on activities of daily living (ADLs).
C. Note intensity of pain at rest and during activity.
D. List precipitating factors.
E. Identify alleviating factors.
F. Note the quality of pain.
G. Is there radiation of pain?
H. Rate pain on a pain scale (usually on the 0–10 scale, with 0 being no pain and 10 being the worst pain).

PHYSICAL EXAMINATION

A. Check temperature, pulse, respiration, and blood pressure.
B. Inspect:
1. Observe overall appearance.
2. Note affect and ability to express self and pain.
3. Note facial grimaces with movement.
4. Note gait, stance, and movements.
5. Inspect area at pain site.
C. Auscultate:
1. Auscultate heart and lungs.
2. Auscultate neck and abdomen.
D. Palpate:
1. Palpate the affected area of pain. ▶

The contributions of Moya Cook to this chapter in prior editions are acknowledged here.

E. Percuss:
  1. Percuss chest.
  2. Percuss abdomen.

F. Perform musculoskeletal exam:
  1. Perform complete musculoskeletal exam, concentrating on the area of pain.
  2. Assess deep tendon reflexes (DTRs).

When performing a musculoskeletal exam, identify the location of pain, presence of trigger points, evidence of injury or trauma, edema, erythema, warmth, heat, lesions, petechiae, tenderness, decreased range of motion, pain with movement, crepitus, laxity of ligaments or cords, spasms, or guarding.

G. Perform neurologic exam:
  1. Perform complete neurologic exam.
  2. Identify change in sensory function, skin tenderness, weakness, muscle atrophy, and/or loss of DTRs.

DIAGNOSTIC TESTS

A. No diagnostic testing is required unless clearly indicated to rule out organic cause of pain.

B. If organic disease is suspected, diagnostic testing may include the following:
  1. CT imaging.
  2. MRI.
  3. Blood chemistries.
  4. Radiographic x-ray.
  5. Lumbar puncture.
  6. Ultrasound.
  7. Echocardiogram.

DIFFERENTIAL DIAGNOSES

A. The differential diagnoses depend on the location of the acute pain.
  1. Head:
    a. Migraine headache.
    b. Cluster headache.
    c. Temporal arteritis.
    d. Intracranial bleeding or stroke.
    e. Sinusitis.
    f. Dental abscess/infection/caries.
  2. Neck:
    a. Meningitis.
    b. Muscle strain/sprain.
    c. Whiplash injury.
    d. Thyroiditis.
  3. Chest:
    a. Pulmonary emboli.
    b. Myocardial infarction.
    c. Pneumonia.
    d. Costochondritis.
    e. Angina.
    f. Gastroesophageal reflux disease/esophagitis.
  4. Abdomen:
    a. Peritonitis.
    b. Appendicitis.
    c. Ectopic pregnancy/uterine pregnancy.
    d. Endometriosis.
    e. Pelvic inflammatory disease.
    f. Peptic ulcer.
    g. Cholelithiasis.
    h. Colitis/diverticulitis.
    i. Constipation.
    j. Gastroenteritis.
    k. Irritable bowel syndrome.
    l. Urinary tract infection, kidney stone, pyelonephritis.
    m. Prostatitis.
  5. Musculoskeletal:
    a. Muscle sprain/strain/tear.
    b. Skeletal fracture.
    c. Viral infection.
    d. Gout.
    e. Vitamin D deficiency.

PLAN

A. General interventions:
  1. Acute pain is a symptom, not a diagnosis. Try to identify the cause or source of the acute pain, depending on the location. If the pain is organic in nature, make the appropriate referral. The overall goal is to treat the acute pain appropriately.

B. Client teaching:
  1. The pain management plan must include client and family/significant other education regarding preventing and controlling pain, potential medication side effects, and preventing side effects.
  2. Discussion must include addiction concerns and a possible addiction risk assessment. Explain addiction is low when the medication is used as directed for a short duration. Explain that complete pain relief may not be achievable initially, but the overall goal is to reduce the pain, thus allowing some daily activities at home to begin recovery.

C. Pharmaceutical therapy:
  1. Source of acute pain:
    a. Visceral pain: The treatment of choice is corticosteroids, intraspinal local anesthetic, nonsteroidal anti-inflammatory drugs (NSAIDs), and opioids.
    b. Somatic pain: Treatment includes acetaminophen, cold packs, corticosteroids, localized anesthetics, NSAIDs, opioids, and tactile stimulation.
    c. Neuropathic pain: Tricyclic antidepressants (TCAs) and serotonin norepinephrine reuptake inhibitors (SNRIs) are useful in reducing pain in clients with/without depression. TCAs such as amitriptyline or imipramine can be used as first-line treatment for neuropathic pain. Anticonvulsants such as carbamazepine (Tegretol), phenytoin (Dilantin), gabapentin (Neurontin), and valproic acid (Depakene) can be useful in treating neuropathic pain but should be reserved for second-line treatment. Carbamazepine and oxcarbazepine can be used for first-line treatment of trigeminal neuralgia. Other treatments include local anesthetics, tramadol (Ultram), and glucocorticoids.

Know each medication's mechanism of action, potential adverse side effects, half-life, and drug–drug interaction potential. Always document that you have advised on potential for sedation, no driving/machinery use, or alcohol while taking medication with these potential adverse side effects.

FOLLOW-UP

A. Once the organic cause of pain has been ruled out, initial follow up is 48 to 72 hours after onset.

B. Ensure the client has access to care on a regular schedule.

CONSULTATION/REFERRAL

A. If the acute pain is organic, make the appropriate referral to a specialist.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Failure to treat pain in the geriatric population is inhumane and increases the risk of delirium in this population.

2. Normal changes associated with the aging process require the clinician to be on high alert when using pain medications in the geriatric population. Age-associated decreases in hepatic and renal function allow for increasing serum drug concentrations and associated adverse effects.

3. Due to age-associated decreases in hepatic and renal function, it is appropriate to initiate opioid medications anywhere from 30% to 50% of the normal dose and titrate up to the desired effect.

4. Because of the risk of inciting renal failure and gastrointestinal bleeding in the geriatric population, NSAIDs should be avoided or used with extreme caution and frequent monitoring.

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CHRONIC PAIN

DEFINITION

A. See the "Acute Pain" section for general statements about pain.

B. Chronic pain is defined as alteration in comfort that persists longer than 3 months (or longer than the anticipated healing time).

C. The pain may be continuous or recurrent and of sufficient duration and intensity. Legitimate chronic pain interferes with a client's ability to function with normal daily activities and decreases quality of life.

D. Chronic pain can be chronic cancer pain, where there is an expected end (either with cure or death), or chronic nonmalignant pain, where there is no predictable ending or cure.

INCIDENCE

A. Pain syndromes are commonly seen in clinical practice and are the third most widespread health problem in the United States. As of 2012, chronic pain cost the American people about $635 billion a year in healthcare expenses, disability costs, and lost productivity. Approximately 50 million or 20% to 30% of adults in the United States experience chronic pain. As the U.S. population continues to age, more people develop pain-associated medical conditions (obesity, diabetes mellitus [DM]), and the average life expectancy increases, the primary care provider will be providing care for more chronic diseases and handling more chronic pain clients.

1. Females are affected more than males by 2 to 1.

2. Onset is usually in the fourth, fifth, or sixth decade and is often associated with marked functional disability.

PATHOGENESIS

A. *Skeletal muscle pain* is pain in soft tissue involving the neck, shoulders, trunk, arms, low back, hips, and lower extremities. *Myofascial pain syndrome* relates to the fascia surrounding the muscle tissue.

B. *Inflammatory pain* is caused by chemicals such as prostaglandins, leading to stimulation of the pain receptors. Examples include arthritis, infection, tissue injury, and postoperative pain.

C. *Mechanical/compressive pain* is the direct result of the muscle, ligament, and tendon causing strain, leading to stimulation of the pain receptors. Diagnosis may be based on diagnostic imaging results which may include fracture, obstruction, dislocation, or compression of tissue by tumor, cyst, or bony structure.

D. *Neuropathic pain* involves dysfunction of the somatosensory system. The most common types are diabetic neuropathy, sciatica from nerve root compression, trigeminal neuralgia, and postherpetic neuralgia.

E. *Nociceptive pain* is caused by nociceptors, a type of sensory neuron that receives the pain signal. Mechanical/compressive and inflammatory pain are examples of this type of pain. They both respond well to opioids, with the exception of arthritis. However, opioids should not be the initial management as other modalities may be just as effective.

PREDISPOSING FACTORS

A. Age 30 to 50 years.

B. Female sex.

C. History of having seen many physicians.

D. Frequent use of several nonspecific medications.

E. Depression: Major depressive disorder is the single most important prevalent chronic pain morbidity.

F. Personality, including moods, fears, expectations, coping efforts, and resources.

COMMON COMPLAINTS

A. Specific to site of pain.

B. Emotional distress related to fear, maladaptive or inadequate support systems, and other coping resources.

C. Treatment-induced complications.

D. Overuse of drugs.

E. Inability to work.

F. Financial complications.

G. Disruption of usual activities.

H. Sleep disturbances.

I. Pain becomes primary life focus.

OTHER SIGNS AND SYMPTOMS

A. Pain lasts longer than 3 months.

B. There may be anger and loss of faith or trust in the healthcare system. This type of client frequently takes too many medications, stays in bed a great deal, has seen many physicians, has lost skills, and experiences little joy in either work or play.

C. Chronic pain interferes with daily life activities. This impacts a person's self-esteem, mood, and energy level. The link between pain and psychological factors is cyclic. The more the client is in pain, the more depressed they become; this in turn leads to more pain.

SUBJECTIVE DATA

A. Elicit a clear description of the onset, location, quality, intensity, and time course of pain and any factors that aggravate or relieve it. Use the acronym OLD CARTS-U: O = onset, L = location, D = duration, C = characteristics, A = aggravating triggers, R = relieving triggers, T = timing, S = severity, U = YOU, What do YOU think is going on? What have YOU done to relieve it? ▶

B. *Self-reporting pain assessment tools* should be used early in the process of client evaluation. Use the tool at each office visit to see progression or regression. Lack of pain assessment is a barrier to good pain control. Consider the age of the client; their physical, emotional, and cognitive status; and their preference when choosing the self-reporting pain assessment tool:

1. The Verbal Rating Scale rates pain as mild, moderate, or severe.

2. The Numeric Rating Scale rates pain intensity from 0 to 10. The tool is client-friendly and quick to complete.

3. The Faces Scale is useful in pediatric and cognitively impaired clients. Multicultural translations may be downloaded at wongbakerfaces.org.

4. There are also several scales that combine different dimensions of the pain experience. Examples include the McGill Pain Questionnaire (long and short versions).

C. Determine the extent to which the client is suffering, disabled, and unable to enjoy usual activity. It is important to inquire about activities of daily living (ADLs) and functional limitations. Disability refers to the inability to do one's usual activities as a result of impairment. There is no gold standard for assessing disability as each organization has different requirements for evaluation and documentation.

D. Obtain a complete review of systems, including nausea, numbness, weakness, insomnia, loss of appetite, dysphoria, malaise, fatigue, or depression signs and symptoms.

E. Obtain a complete family and social history. Address spiritual and cultural issues. Evaluation of the risk of opioid dependence/abuse in this population is essential. There are several instruments that can be used, such as The Screening Instrument for Substance Abuse Potential.

F. Obtain the client's medical history relevant to the pain, including diagnosis, testing, treatments, and outcomes.

G. Obtain a pain history to identify the client's attitudes, beliefs, level of knowledge, and previous experiences with pain. Are previously used methods for pain control helpful? What is the client's attitude toward the use of certain pain medications? Often, the client will discuss certain adverse side effects or allergies from undesired pain medication.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.

B. Inspect:

1. Observe overall appearance.

2. Note affect and ability to express self and pain.

3. Note facial grimaces with movement.

4. Note gait, stance, and movements.

5. Inspect area at pain site.

C. Auscultate:

1. Auscultate heart and lungs.

2. Auscultate neck and abdomen.

D. Palpate:

1. Palpate the affected area of pain.

E. Percuss:

1. Percuss chest.

2. Percuss abdomen.

F. Perform musculoskeletal exam:

1. Perform a complete musculoskeletal exam, concentrating on the area of pain.

2. Note limitations in range of motion.

When performing a musculoskeletal exam, identify the location of pain, presence of trigger points, evidence of injury or trauma, edema, erythema, warmth, heat, lesions, petechiae, tenderness, decreased range of motion, pain with movement, crepitus, laxity of ligaments or cords, spasms, or guarding.

G. Perform neurologic exam:

1. Perform complete neurologic exam.

2. Note the client's affect and mood. Is client cooperative during exam?

3. Identify change in sensory function, skin tenderness, weakness, muscle atrophy, and/or loss of deep tendon reflexes (DTRs).

H. Perform functional assessment:

1. The baseline functional assessment provides objective measurable data on a client's physical abilities and limitations. It can be used to determine if the client's efforts are valid and complaints are reliable.

2. The information may be used to identify areas of impairment, establish specific functional goals, and measure the effectiveness of treatment interventions.

3. These objective data may be used in worker compensation cases, returning to work status, federal disability, and motor vehicle accident lawsuits.

4. Know the resources in your area that are trained to perform functional assessments. Physical therapists and occupational therapists are the best qualified to perform the assessments.

DIAGNOSTIC TESTS

A. None required unless clearly indicated to rule out the organic cause of pain.

1. Remember that pain previously diagnosed as chronic pain syndrome can be organic and vice versa. Organic causes must always be evaluated and excluded.

2. Health insurance usually requires plain radiography ordered first for muscle, inflammatory, or skeletal pain. Plain radiography will diagnose a fracture. Additional studies may be recommended by the radiologist if a lesion/abnormality is seen on plain radiography.

3. MRI and CT are ordered if the plain radiography is negative and the client continues to complain of pain.

4. Electromyography and nerve conduction studies are used to evaluate neuropathic pain. Numerous serum and urine studies should also be considered if the neuropathic pain is undiagnosed.

B. Consider using a depression assessment tool, such as the Beck Depression Inventory or the Patient Health Questionnaire 9 (PHQ-9). These tools can be administered at a subsequent appointment to follow the client's symptoms.

DIFFERENTIAL DIAGNOSES

A. Pain disorder.

B. Pain related to a disease with no cure.

C. Somatization disorder.

D. Conversion disorder.

E. Hypochondriasis.

F. Depression.

G. Chemical dependency.

H. Fibromyalgia.

PLAN

A. General interventions:

1. Treatment is multidimensional and should not be focused on pharmacologic treatment alone.

2. Offer hope and potential for improvement of pain control and of function but NOT always cure. ▶

3. The pain is real to the client, and acceptance of the problem must occur before a mutually agreed-on treatment plan can be initiated.

4. Depression is a common emotional disturbance in chronic pain clients and is treatable. Consider oral therapy. See the "Depression" section in Chapter 25.

5. Identify specific and realistic goals for therapy, such as having a good night's sleep, going shopping, or returning to work. Client discussion needs to include the idea that the goal may be reducing pain intensity, not eliminating pain.

6. Carefully assess the level of pain using available tools, such as a daily pain diary or other pain assessment scales.

7. Avoid pain reinforcement such as sympathy and attention to pain. Provide positive response to productive activities. Improving activity tolerance assists in desensitizing the client to pain.

8. Shift the focus from the pain to accomplishing daily assigned self-help tasks. The accomplishment of these tasks functions as a positive reinforcement.

B. *See Client Teaching Guide for this chapter, "Chronic Pain."*

C. Pharmaceutical therapy:

1. See Figure 6.1 for summary of medications used in pain. Medications should be based on the World Health Organization's (WHO) scale for mild to severe pain. Although this was developed for use in cancer pain, it is helpful for noncancerous chronic pain as well.

a. Skeletal muscle pain: Treatment should focus on physical rehabilitation and behavioral management. Trichloroacetic acids (TCAs) and muscle relaxants (cyclobenzaprine) may be used. There is little evidence indicating the need for opioids in the treatment of skeletal muscle pain.

b. Inflammatory pain: Nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroids are first-line pharmaceutical interventions. Topical creams (Voltaren Gel) and solutions have been used in treating arthritis pain.

c. Mechanical/compressive pain: Opioids may be used to manage these symptoms while other measures are being taken. Referral to specialist(s) is usually indicated.

d. Neuropathic pain:

i. Gabapentin (Neurontin) and pregabalin (Lyrica) have become first-choice treatments in recent years for diabetic neuropathy and postherpetic neuralgia.

ii. TCAs and serotonin norepinephrine reuptake inhibitors (SNRIs) are extremely useful. Clients who are not depressed obtain excellent pain relief with TCAs such as amitriptyline and doxepin. Examples of antidepressants that may help with pain include but are not limited to venlafaxine (Effexor XR) and duloxetine (Cymbalta). ▶

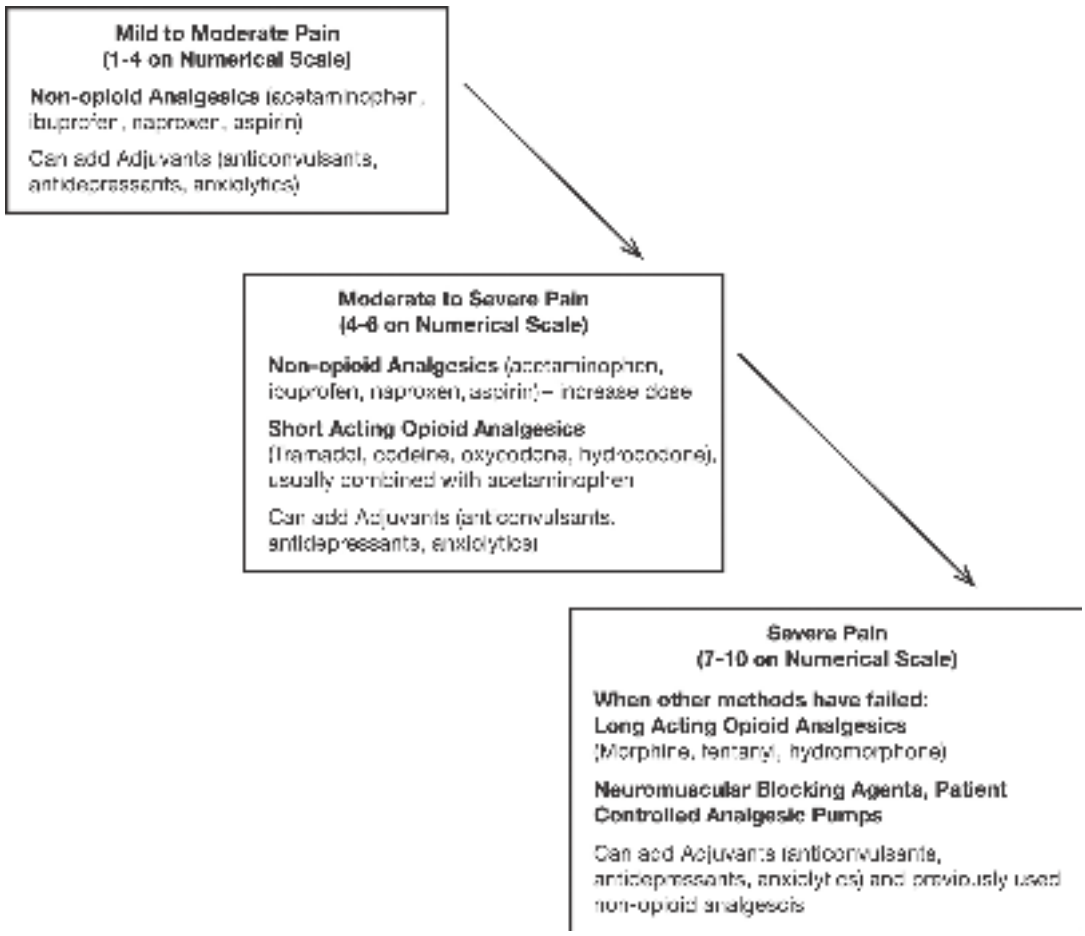


FIGURE 6.1 World Health Organization pain analgesic ladder.

Source: Adapted from World Health Organization from Anekar, A., & Casselar, M. (2021). *WHO analgesic ladder.* https://www.ncbi.nlm.nih.gov/books/NBK554435/.

iii. Anticonvulsants are useful in controlling some neuropathic pain: carbamazepine (Tegretol), phenytoin (Dilantin), and valproic acid (Depakene). **Clients need to be monitored monthly for hepatic dysfunction and hematopoietic suppression.** Blood work should be ordered on a regular basis for assessment of liver function, blood studies, and medication toxicity levels.

iv. Topical agents: Capsaicin applied three to four times per day can be used to reduce pain without significant systemic effects. Topical lidocaine 5% patches are approved for postherpetic neuralgia.

v. Carbamazepine is used as first-line treatment for trigeminal neuralgia.

vi. Opioids: Tramadol is considered a good first-line choice if an opioid is indicated. In addition to pain control, tramadol also causes serotonin reuptake inhibition similar to that seen with TCAs. **Precautions should be taken to avoid the risk of seizure disorder and serotonin syndrome when using tramadol with other selective serotonin reuptake inhibitors (SSRIs).**

e. All therapies need a 2- to 3-week trial period to adequately evaluate therapy. Some medications take longer to evaluate.

f. NSAIDs, especially cyclooxygenase (COX-2) inhibitors such as celecoxib (Celebrex), should be used for flare-ups of mild to moderate inflammatory or nonneuropathic pain. They may be used along with opioids as long as medication dosing remains within the recommended guidelines.

g. Safe opioid prescribing:

i. Opioids are often prescribed by potency, with the pure *mu* opioid the most powerful (hydromorphone, oxycodone, fentanyl, morphine).

ii. Opioids require careful client selection, titration, and monitoring.

iii. Avoid long-term, daily treatment with short-acting opioids (oxycodone, hydrocodone). For as-needed use, prescribe minimal quantities (no more than 3–7 days).

iv. After completing a full biopsychosocial assessment, start with the lowest effective dose and combine it with other pain-relieving modalities (see "Alternative interventions").

v. Check your state's prescription drug monitoring program (PDMP) prior to prescribing controlled substances, as needed and at least annually. PDMPs are state-run electronic databases that track dispensing of controlled substances. PDMPs provide clinicians with critical information about clients' narcotic prescription history and identify seeking behavior patterns.

vi. Contact the client's pharmacy for a list of current medications. The PDMP is not real time, and all current client prescriptions are available from the pharmacy.

vii. Perform urine drug screen prior to prescribing controlled substances initially, as needed, and annually. National guidelines recommend enzyme immunolinked assay (EIA) and gas chromatography/mass spectroscopy urine screen. Depending on results of urine drug screening, the provider may seek additional consultation, change medication therapy, refer for substance abuse, or discharge the client for noncompliance with the established drug contract between the prescriber and the client.

viii. A written controlled substance treatment agreement between client, provider, and clinic is recommended (informed consent). This should include full diagnosis, nature/purpose of the opioid treatment plan, complete listing of potential risk/benefits of therapy, and alternative and risks/ benefits of not receiving treatment. In addition, include the expectations of the client.

1) No other controlled substances will be prescribed by any other provider.

2) Only one pharmacy should be used when filling the prescription of the controlled substance.

3) Medication must be taken as prescribed.

4) There are no early refills on controlled substances.

5) Refills require a clinical visit for evaluation.

6) Lost/stolen prescriptions will not be refilled.

7) The client must agree to random drug screens and may be called to report to the clinic for random drug screens and/or pill counts. An example of this informed consent can be found at www.painmed.org/files/consent-for-chronic-opioid-therapy.pdf.

ix. Utilize tools such as the Addiction Behaviors Checklist, DIRE (Diagnosis, Intractability, Risk, Efficacy) score, or CAGE assessment (Have you ever tried to **C**ut down on your alcohol/drug use? Do you get **A**nnoyed if someone mentions your use is a problem? Do you ever feel **G**uilty about your use? Do you ever have an **E**ye-opener first thing in the morning after you've been drinking or using the night before?).

x. Red flags for misuse, abuse, addiction, and diversion with opioids include the following:

1) Psychiatric illness.

2) Personal history of alcohol or drug abuse or overdose.

3) Family history of alcohol or drug abuse.

4) Concurrent benzodiazepine use.

h. Ambulatory infusion pumps can be programmed to administer pain management medication continuously, intermittently, tapered, or client-controlled. Infusion pumps prescribed for clients should be overseen by a pain medicine specialist.

i. Benzodiazepines and barbiturates are not advised for treatment of chronic pain due to the high risk of substance abuse.

D. Alternative interventions:

1. Cognitive behavioral training: Examples of cognitive behavioral training include problem-solving, guided imagery, hypnosis, controlled breathing exercises, attention diversion, meditation, and yoga exercises; progressive muscle relaxation (PMR) is recommended to help relax major muscle groups.

2. Exercise: Examples of exercise include yoga exercises and PMR. PMR is recommended to help relax major muscle groups. Research indicates that yoga reduces bothersome pain after 12 weeks of regular exercise. The benefits of yoga exercise include improved strength, balance, coordination, range of motion, and reduced anxiety. Yoga ▶

instruction by a qualified teacher is a low-cost intervention. Yoga is an effective form of self-care and is an affordable way to alleviate pain. Always advise the client to start slowly and be prepared for an approach to pain management that may take several weeks of therapy.

3. Alternative therapies: Randomized controlled trials showed significant reduction in pain with alternative interventions such as music, relaxation, distraction, acupuncture, myofascial release treatments, and massage use.

4. Occupational therapy.

5. Vocational therapy.

6. Physical therapy such as noninvasive techniques, transcutaneous electrical nerve stimulation (TENS), hot or cold therapy, hydrotherapy, traction, massage, bracing, and exercise.

7. Individual and family therapy or counseling.

8. Aesthetic or neurosurgical procedures.

9. Cold laser therapy: Low light therapy stimulates healing. This therapy can be used for inflammation and has been used to stimulate healing in sprains/strains, dental ulcerations, and muscle spasms, as well as skin rejuvenation and wound healing.

10. Clients will inquire about the use of herbal products to treat chronic pain. Advise clients that these products are not regulated by the Food and Drug Administration (FDA) and may interact with current medications and cause complications. Advise clients to research all herbal products on reputable medically based websites, not blogs or chat rooms. Caution clients regarding devil's claw, feverfew, willow bark, glucosamine, and chondroitin. Discourage any use of dimethyl sulfoxide.

FOLLOW-UP

A. See clients every 4 to 6 weeks for evaluation. Reassess using the 5As: Analgesia, Activity, Adverse effects, Aberrant behaviors, and Affect.

B. Ensure the client has access to care on a regular schedule.

C. These brief visits should be regular so that care is not perceived to be dependent on escalation of symptoms.

D. Monitor for possible side effects, including sedation, pruritus, opioid-induced constipation/bowel dysfunction, nausea, and vomiting and treat as indicated.

CONSULTATION/REFERRAL

A. Consider client referral to a pain management clinic/specialist if pain control is not adequate. Interventions commonly performed at the specialty clinic include facet joint injections, percutaneous radiofrequency neurotomy, epidural corticosteroid injections, transforaminal epidural injections, and sacroiliac (SI) joint injections.

B. Consult with a physician/specialist if referral is needed for psychological counseling or if substance abuse is suspected.

C. Refer to a certified pain specialist provider if the client is taking high doses of opioids and detoxification is indicated. Buprenorphine (Subutex) is the most common medication prescribed by a certified pain specialist physician.

D. Consider rheumatology consult if a rheumatic condition is suspected.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. When prescribing pharmacologic agents, consider drug–drug interactions. When administering tramadol with use of any SSRI, educate the client regarding the possible interactions between the two medications and teach the client the signs and symptoms of serotonin syndrome and to report these signs if they occur. Avoid using this combination of medications if the client is known to have problems with metabolizing CYP2D6.

B. Pregnancy:

1. Careful consideration should be used when prescribing pain medication to clients who are pregnant. Tylenol is safe during pregnancy. NSAIDs are not recommended during pregnancy. When prescribing medications, refer to the prescribing classification of all medications for safety clarification during pregnancy.

C. Geriatrics:

1. Guidelines for assessment of pain in the geriatric population suggest the following:

a. Client self-report is the most accurate and reliable method—even for clients with cognitive impairment.

b. Consider using a narrative approach, allowing the client an opportunity to relay meaningful stories describing pain and their conclusions as to the source of their pain.

c. If clients deny "pain," try other descriptives such as "soreness" or "stings" or "aching" or "uncomfortable." Reword questions. For example, "Is there any place on your body that hurts? Please show me." "Is there anywhere inside that doesn't feel right?" "Is there any symptom that is stopping you from doing what you want to do?"

d. Educate and train clinical staff and collaborative professional teams how to recognize and engage in a conversation that will help older adults express their pain symptoms.

e. For geriatric clients who have dementia, the recommended tools are Pain Assessment in Advanced Dementia (PAINAD) and Doloplus-2.

2. Prior to prescribing medications to the geriatric client, review their current medication list for drug–drug interactions. Also, check the new medication against the Beers criteria for medications that should be avoided in the geriatric population (www.elderconsult.com/wp-content/uploads/PrintableBeersPocketCard.pdf).

3. Initiate new medications at the lowest dose and titrate slowly for the desired effect. In some cases with profound renal and hepatic insufficiency, it is not unreasonable to initiate medications at 30% to 50% lower doses than even the minimal suggested dose. Avoid extended-release products in renal and hepatically impaired clients.

4. Concerns with opioids in older adults are neurotoxicity, delirium, cognitive impairments, and increased risk of falls leading to orthopedic injury.

5. Tramadol decreases the seizure threshold and may place clients at risk of seizure-related injury. Use with caution in geriatric clients (although it may be used in clients with well-controlled seizure disorders).

6. A concern with tramadol in older adults is fall risks (fractures/injuries). Use with caution in geriatrics treated for seizures because it lowers the seizure threshold. It may be used if seizures are well controlled. Avoid extended-release product in renal-impaired geriatric clients. Instead, reduce the dose of an immediate-release product.

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PAIN ASSESSMENT AND MANAGEMENT IN THE AGING POPULATION

DEFINITION

A. Persistent pain occurs quite frequently in the elderly population. However, pain is not a normal part of aging. Chronic pain is defined as having sensory/emotional changes with experience of tissue change/damage for longer than 3 months. Uncontrolled chronic pain may lead to a downward spiral for the client, progressing to alterations in physical and mental health status. Inadequate assessment and management of chronic pain may lead to a decrease in the quality of life for all clients.

COMMON PAIN SYNDROMES

A. There are many common pain syndrome that are present in older adults. Assessing for these unidentified pain syndromes may enhance the discovery of the source of pain for the client. Common pain syndromes experienced by elderly clients include fibromyalgia, gout, osteoarthritis, unidentified fractures, neuropathies, and vitamin deficiencies such as vitamin B_{12} deficiency.

INCIDENCE

A. Older adults are the fastest growing population. It is estimated that by the year 2040 there will be more than 1.3 billion people worldwide older than the age of 65. In the United States alone, there will be over 80 million people living in this age group.

PATHOGENESIS

A. Studies indicate that pain sensitivity changes and pain threshold increases with aging. Therefore, the absence of reportable pain does not mean that pain does not exist in the elderly population. As one ages, the number of neurotransmitter cell receptors in the brain diminishes as brain tissue begins to atrophy. This loss of neurons has a direct impact on pain transmission, which ultimately increases pain tolerance and disguises the presence of pain in the elderly.

PREDISPOSING FACTORS

A. Aging adult.
B. Barriers to effective pain assessment:
1. Cognitive and language impairments.
2. Comorbid chronic condition (e.g., depression, anxiety, agitation, illness, dementia, psychological disorders, and memory disorders).
3. Medications taken by the client.
4. Ineffective communication to the provider.
5. Ineffective assessment by the provider.
6. Noncompliance with medication regimen.

SUBJECTIVE DATA

A. On the initial exam, a comprehensive history is imperative when assessing for pain. Communication is a key ingredient when assessing for pain. Hearing loss, vision loss, and the inability to fill out documents (pain scales) are a few components of the assessment that can interfere with a successful interview. Adjustments should be made to ensure that communication among the client, family/significant other, and provider is clear. Clients who are not able to provide verbal information should be assessed for behaviors and physical signs of pain. Physical signs of pain may include grimacing, moaning, silence, poor eye contact, and so forth. Behaviors that may be an indicator of pain may include anxiety, agitation, confusion, depression, and isolation. Other factors to consider when assessing a client for pain include their age, medical history, surgical history, current medications, current cognitive status, and functional status. A focused history will assist in establishing a baseline status for the client.
1. Assess for communication/cognitive impairments that may interfere with the assessment. Consider communication factors, comorbid conditions/illnesses, and current medications.
2. Assess the current mood and physical state of the client (e.g., depression, anxiety, anger, illness).
3. Have the client describe the duration of pain and what time of day symptoms begin.
4. Ask the client to describe the pain, for example, crushing, stabbing, or burning.
5. Ask the client where the sensation begins and in what direction it radiates. Does pain come and go, or is it constant? What makes pain worse? What makes pain better?
6. Have the client rate pain on a scale of 0 to 10, with 0 being no pain.
7. Ask the client to complete a functional status form to assess functional abilities. Identify impairments of activities of daily living (ADLs) and note limitations.
8. Ask what medications/treatments have been used in the past for pain. Was pain improved?
9. Inquire regarding family/social support, financial resources, and concerns the client may have regarding social complexities.

PHYSICAL EXAMINATION

A. Start with vital signs: Check temperature (if applicable), pulse, respirations, and blood pressure.
B. Inspect:
1. Inspect general appearance, noting facial grimaces, frowning, body movement, and response to assisted movement.
2. Inspect site of pain for swelling and erythema.
C. Palpate:
1. Palpate specific sites of noted discomfort/pain.
2. Palpate adjacent joints/areas to site of discomfort. (Pain may also be caused from referred pain. A thorough exam of the surrounding joints/tissues should be assessed.) Note inflammation, change in skin temperature (warm/cool), and pain with palpation.
D. Perform musculoskeletal exam:
1. Perform a complete musculoskeletal exam, concentrating on the site of pain.
2. Assess range of motion, gait, and mobility. Evaluate strength, noting symmetry of strength, and/or weakness.
E. Perform neurologic exam:
1. Perform a neurologic exam on sites involved.
F. Auscultate:
1. Conduct a complete heart exam, checking for dysrhythmias. ▶

2. Conduct a complete lung exam.
3. Auscultate abdomen, noting bruits if present.

G. Mental status:
1. Assess for confusion, agitation, change in personality, anxiety, and so forth.

DIAGNOSTIC TESTS

A. Perform a Mini-Mental State Exam (MMSE) if indicated. This form is available at www.ihpa.gov.au/sites/g/files/net636/f/publications/smmse-tool-v2.pdf.

B. Perform a functional status assessment. There are many instruments that can be used; examples include the Instrumental Activities of Daily Living Scale, the 10-Minute Geriatric Screen, and the Katz Independence in Activities of Daily Living. A provider resource for assessing functional status can be found at hmsa.com/portal/provider/PRC_Guide_COA_Functional_Status_Assessment.pdf.

C. Perform the STEADI test (Stopping Elderly Accidents, Deaths & Injuries) to assess mobility and predict fall risk. Video and form information for provider and client is available at www.cdc.gov/steadi/materials.html.

D. Assess for pain using a valid method of testing for elderly clients. The Verbal Descriptor Scale (VDS) is recommended for assessment of the elderly client. Other available tools include the Faces Pain Scale and the Numeric Rating Scale. A listing of validated clinically tested tools can be found at www.practicalpainmanagement.com/resource-centers/opioid-prescribing-monitoring/list-clinically-tested-validated-pain-scales.

PLAN

A. General interventions:
1. The primary goal is to identify the source of pain. Once the source is noted, measures to improve pain should be performed.

B. Client teaching:
1. Educate the client regarding the source of pain.
2. Discuss possible sources of improving pain according to the diagnosis.
3. Depending on the source of pain, consider nonpharmacologic treatments to improve pain.

C. Special elderly considerations for pain management:
1. Nonpharmacologic treatments: Such treatments may include physical therapy, aquatic therapy, exercise, chiropractic treatments, massage, acupressure, acupuncture, transcutaneous electrical nerve stimulation (TENS), guided imagery, relaxation, hot/cold therapy, and so forth.

D. Pharmaceutical therapy:
1. Body fat, total amount of water/fluid in the body, poor nutritional status, and muscle mass all impact drug metabolism. There is a heightened sensitivity to adverse effects that results from physiologic changes, drug interactions, and drug–disease interactions.
2. Medications, such as diuretics, may influence the impact of drug distribution in the body. These factors should be considered when prescribing medications in the elderly:
a. Some changes affected by these components include onset of action, availability of the drug, and excretion of the drug.
b. Use caution when prescribing medications and repeating the dose if no response is perceived. Repeating the drug dose to obtain therapeutic results can cause adverse events and/or overdose.
c. If the drug has a slow onset, it may build up in the body and cause an overdose of the medication.
d. Aging is associated with higher steady-state concentrations of water-soluble drugs and increased half-life of fat-soluble drugs. Therefore, when prescribing medications to elderly clients, all medications should be started at the **lowest** dose possible and titrated up slowly as tolerated. Lower doses are better tolerated and potentially will lead to fewer side effects.
3. Refer to the "Chronic Pain" section for modalities and assessment for pharmacologic agents.
4. For special considerations with specific pain medications, please refer to Table 6.1.
a. Nonopioids: Acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs) should be discussed and considered for treatment of mild pain. Assess liver and kidney function prior to administering these medications:
i. Acetaminophen: Contraindications to acetaminophen include liver failure, hepatic insufficiency, and chronic alcohol use. The maximum recommended dose of acetaminophen is 2 g per 24-hour period, which includes all Tylenol products and combination medications that include Tylenol.
ii. NSAIDs: Contraindications to NSAIDs include peptic ulcer disease, chronic kidney disease, and heart failure. Monitor closely if the client is taking other medications that may interact with the NSAID, such as corticosteroids and selective serotonin reuptake inhibitors (SSRIs). Elderly clients should only use one NSAID at a time. Advise the client to avoid using over-the-counter NSAIDs, along with prescribed NSAIDs. Elderly clients should also be advised to use a proton pump inhibitor along with the NSAID to protect the gastrointestinal system. Clients taking aspirin should not use ibuprofen while also taking aspirin. Clients prescribed these medications should be closely monitored for gastrointestinal problems (ulcers, gastroesophageal reflux), prolonged bleeding times, and adverse change in kidney function. Monitor the current use of other medications (e.g., aspirin, NSAIDs, proton pump inhibitors, Coumadin) that would interact with these medications.
b. Opioids: Opioids may be considered for treatment of moderate to severe pain when nonopioids are not effective. Proper education regarding the use of these medications is necessary to avoid misconceptions regarding the use of these drugs. Educate the client that opioids are safe medications when used properly as prescribed and that with proper use addiction can be prevented.
i. Opioids that should not be used in the elderly client include meperidine and propoxyphene. These agents may cause central nervous system toxicity if poorly metabolized by the liver.
ii. Opiates have a higher risk of falls and the client should be educated on this possibility.
c. Other medications:
i. Anticonvulsants, such as gabapentin, have been used successfully to treat neuropathic pain. Starting doses should be low and gradual titration upward can eliminate side effects and can be used successfully to treat pain. Studies have shown that doses from 2,400 to 3,600 mg/d reduce pain and improve mood and sleep disturbance. ▶

TABLE 6.1 PAIN MEDICATIONS

Classification	Examples	Special Administration/Dosing Considerations[a]
Acetaminophen	Acetaminophen	Contraindications to acetaminophen include liver failure, hepatic insufficiency, and chronic alcohol use (active/present). Do not exceed 4 g in healthy adults. Do not exceed 2 g in healthy elderly clients.
NSAIDs	Ibuprofen Naproxen Aspirin Diclofenac Meloxicam Indomethacin	Contraindications to NSAIDs include peptic ulcer disease, chronic kidney disease, and heart failure. May prolong PT. Note potential interactions with corticosteroids and SSRIs. Take only one NSAID at a time; do not use with aspirin. Consider a PPI.
NSAIDs, COX-2	Celecoxib	Use with caution due to cardiac risks (stroke and MI). Do not exceed 400 mg/d. Celecoxib is contraindicated in clients with sulfa allergy.
Tramadol (considered weak opioid)	Tramadol	Be aware of potential acetaminophen or NSAID toxicity with concurrent use of opioids. Be aware of the interactions with the use of SSRIs and risk of serotonin syndrome and seizures.
Topical agents	NSAIDs (diclofenac sodium) Lidocaine Ketamine Capsaicin	Begin with low dose and increase as tolerated. Lidocaine patches: postherpetic neuralgia. NSAIDs: localized nonneuropathic pain. Capsaicin: regional pain syndromes.
Corticosteroids	Prednisone Methylprednisolone Cortisone	Tapering for short-term use as indicated. Used for neuropathic pain and pain associated with inflammatory changes or metastatic bone pain. Not for long-term use. Avoid use in the elderly due to high side effect risk.
Muscle relaxants	Cyclobenzaprine Carisoprodol Methocarbamol Baclofen Metaxalone	Use in conjunction with NSAIDs or separately if NSAIDs are ineffective. High fall risk as muscle relaxants cause drowsiness and sedation.
Stronger opioids (short-acting)	Codeine Hydrocodone Oxycodone Morphine	Begin low with short-acting only. Increase over time. Avoid morphine in clients with renal insufficiency. Sustained release formulation only for constant pain and should be according to the recommendation of pain specialist. Be aware of potential acetaminophen or NSAID toxicity with concurrent use of opioids.
Neuropathic Pain		
Tricyclic antidepressants	Amitriptyline Imipramine Doxepin Nortriptyline Desipramine	Begin low; reevaluate and increase every 4–7 days. Order an EKG prior to starting therapy. Use with caution in clients with cardiac issues, especially conduction abnormalities. Avoid amitriptyline, imipramine, and doxepin in the elderly due to higher side effect risk (anticholinergic effects).
SNRI depressants	Duloxetine Venlafaxine Milnacipran	Duloxetine is a P450 2D6 isoenzyme inhibitor but the only one approved by the FDA as an analgesic.
Anticonvulsants	Gabapentin Pregabalin Carbamazepine	Gabapentin and pregabalin are usually well tolerated.

[a]All medications should be checked for use during pregnancy and lactation prior to prescribing.
COX-2, cyclooxygenase; FDA, Food and Drug Administration; MI, myocardial infarction; NSAID, nonsteroidal anti-inflammatory drug; PPI, proton pump inhibitor; PT, prothrombin time; SNRI, serotonin norepinephrine reuptake inhibitor; SSRI, selective serotonin reuptake inhibitor.

ii. Tricyclic antidepressants (TCAs): Nortriptyline has been shown to be successful in treating pain in elderly clients. Use with caution in clients with known cardiac conditions. Consider ordering a baseline EKG prior to beginning therapy. Avoid use if cardiac/conduction abnormalities are present. The starting dose should begin low, and a gradual increase in dose every 4 to 7 days should be prescribed as tolerated. Amitriptyline, imipramine, and doxepin should be avoided in the elderly due to a higher risk of side effects.

iii. Local anesthetics: Pain patches may be used with discretion. Lidocaine patches are useful for postherpetic neuropathy. Other pain patches may also be trialed, beginning at a low dose and titrated as tolerated. Topical NSAIDs may be used for localized nonneuropathic pain. Capsaicin and menthol topical agents have been shown to improve regional pain syndromes. Epidural injections have also been found useful in alleviating pain in older adults.

iv. Corticosteroids: Steroids may be used for neuropathic pain and pain associated with inflammatory changes or metastatic bone pain. However, great caution should be used in treating clients with steroids. Long-term use is not recommended. Significant side effects with use of steroids can occur in the elderly population in a very short period of time (such as muscle wasting, hyperglycemia). Consider other alternatives for pain control instead of steroids for elderly clients.

d. Cancer pain: The World Health Organization (WHO) recommends around-the-clock pain treatment for clients diagnosed with cancer. Cancer treatment strategies include the following:

i. Acetaminophen is the first-line treatment for mild pain.

ii. NSAIDs are recommended as second-line therapy for mild to moderate pain.

iii. Mild opioids (codeine, hydrocodone, propoxyphene, and oxycodone) are recommended for moderate pain and pain not responding to NSAIDs.

iv. Morphine may be used for severe pain and pain not responding to mild opioids. Other alternatives to morphine include hydromorphone, fentanyl, and methadone.

v. Consider adding additional medications for severe pain complicated by anxiety/mood disorders and not controlled on a single agent. Common medications that enhance the effects of analgesics include antidepressants, anticonvulsants (gabapentin), muscle relaxants, antihistamines, corticosteroids, topical creams/gels, antiarrhythmics (beta-blockers), and alpha-2 adrenergic agonists (clonidine).

CONSULTATION/REFERRAL

A. Refer all clients to a pain management specialist if considering long-term pain management.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. As many as 53% of older adults report acute pain in the preceding 3 months and 75% of these adults identify pain in more than two locations. Pain is a significant burden to the geriatric population and must be treated.

2. Asking geriatric clients about pain at night and difficulty performing ADLs is an efficient way of identifying older adults who are vulnerable to developing depression illness.

3. Older adults taking opioids for pain experience only small amounts of relief and improved function and adverse effects were three times more likely to occur. Common opioid-induced symptoms include constipation, drowsiness, nausea, headache, and confusion. Most of these effects placed the elderly client at greater risk of falls, which could lead to serious injuries. The risk-to-benefit ratio of the use of opioid medications in this population must be thoughtfully considered.

4. Integrative approaches such as yoga, massage, or chiropractic manipulation might be too strenuous for geriatrics. Tailor interventions to the individual and assess the client for osteoporosis before recommending high-impact exercises or aggressive alternative physical activities.

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LOW BACK PAIN

DEFINITION

A. Painful conditions of the lower back may be categorized as follows:

1. Potentially serious disorders: acute fractures, tumor, progressive neurologic deficit, nerve root compression, and cauda equina syndrome.

2. Degenerative disorders: aging or repetitive use, degenerative disease, and osteoarthritis.

3. Nonspecific disorders: benign and self-limiting with unclear etiology.

INCIDENCE

A. Lower back pain (LBP) is commonly seen in clients from ages 20 to 29 years, with increasing incidence until age 65 years. It may be acute or chronic in nature. Chronic LBP lasts more than 3 months.

B. Approximately 70% to 80% of people experience back pain at one point in their lifetime.

C. The long-term outcome of LBP is highly favorable. However, the cost of treating LBP can be upward of $134 billion or more per year, with increasing prevalence.

PATHOGENESIS

A. Pain arises from fracture, tumor, nerve root compression, degenerative disc, osteoarthritis, and strain of the ligaments and musculature of the lumbosacral area.

PREDISPOSING FACTORS

A. Trauma causing ligament tearing; stretching of the vertebra, muscles, tendons, ligaments, or fascia.

B. Repetitive mechanical stress.

C. Tumor.

D. Exaggerated lumbar lordosis.

E. Abnormal, forward-tipped pelvis.

F. Uneven leg length.

G. Chronic poor posture due to inadequate conditioning of muscle strength and flexibility, improper lifting techniques causing excessive strain, and poor body mechanics.
H. Inadequate rest.
I. Emotional depression.

COMMON COMPLAINTS

A. Pain in the lower back area may range from discomfort to severe back pain, with or without radiation down either leg or bilaterally.

OTHER SIGNS AND SYMPTOMS

A. Ambulating with a limp.
B. Limited range of motion.
C. Posture normal to guarded.

SUBJECTIVE DATA

A. Ask the client to discuss the origin of pain. How has the pain progressed or changed since the initial injury or stress?
B. Ask the client to point to an area where pain is felt.
C. Have the client describe the pain. Is it radiating, with sharp, shooting pain down to the lower leg and feet?
D. Ask: What makes the pain worse or better? Have the client list current medications or therapies used for pain, noting the results of treatment.
E. Investigate occurrence of systemic symptoms such as fever and weight loss.
F. Explore client's medical history. Note previous trauma or overuse, tuberculosis, arthritis, cancer, and osteoporosis.
G. Inquire about symptoms such as dysuria, bowel or bladder incontinence, muscle weakness, paresthesia, and loss of sensation. Bowel or bladder dysfunction, bilateral sciatica, and saddle compression may be symptoms of severe compression of the cauda equina and necessitates an urgent workup and referral.
H. Ask the client about precipitating factors such as athletics, heavy lifting, driving, yard work, occupation, sleep habits, or systemic disease.
I. Use a pain scale to describe the worst pain and the best pain levels.

PHYSICAL EXAMINATION

A. Check temperature, pulse, blood pressure, and respirations.
B. Inspect:
  1. Observe general appearance; note discomfort and grimacing on movement and/or examination.
  2. Distraction may distinguish pain behavior from actual pathology.
  3. Note evidence of trauma with bruises, cuts, and fractures.
  4. Note posture and gait.
C. Palpate:
  1. Palpate spine and paravertebral structures, noting point tenderness and muscle spasm. Palpation elicits paravertebral tenderness and generalized tenderness over lower back to upper buttocks.
  2. Examine abdomen for masses.
  3. Palpate peripheral pulses of extremities.
D. Perform neurologic exam:
  1. Identify sensation and pain distribution.
  2. Determine motor strength and evaluate whether muscle strength is symmetrical: Upper extremity resistance is equal bilaterally.
  3. Test deep tendon reflexes (DTRs) and dorsiflexion of big toe.
E. Evaluate using the FABER test (flexion, abduction, and external rotation) or the Stork test (stand on one leg; lift the other leg and extend backward; palpate the sacroiliac joint on both sides while performing test). Back extension requires that you lie on your stomach, bring your legs upward, press your abdomen downward, and clasp your hands behind your shoulder blades (upper part of body should not be on the ground).
F. Check sensation of the perineum to rule out cauda equina syndrome.
G. Perform traction tests: straight leg raises, crossed leg raises, Yeoman Guying, and Patrick test. Musculoskeletal findings include the following:
  1. Straight leg raising and dorsiflexion of foot on affected side may reduce lower back discomfort.
  2. Elevate each leg passively with flexion at hip and extension of knee. Positive straight leg raise is radicular pain when the leg is raised 30° to 60°.
  3. Crossed leg raises: Test is positive when pain occurs in leg not being raised.
  4. Yeoman Guying: Unilateral hyperextension in prone position identifies lumbosacral mechanical disorder.
  5. Patrick test: Place heel on opposite knee and apply lateral force; check for hip or sacroiliac disease.
  6. Range of motion: Increased pain with extension often indicates osteoarthritis. Increased pain with flexion often indicates strain or injured disc.
H. Perform pelvic exam:
  1. Consider pelvic and rectal exam, if indicated. If the client has fallen on the coccyx, a rectal exam is needed to check for stability.

DIAGNOSTIC TESTS

A. Laboratory (usually unnecessary on initial exam): complete blood count, erythrocyte sedimentation rate, C-reactive protein (CRP), serum calcium, alkaline phosphatase, urinalysis, and serum immunoelectrophoresis when inflammatory conditions, neoplastic conditions, diffuse bone disease, or renal disease is suspected.
B. Radiography of spine (imaging studies not usually necessary during the first 6 weeks unless there are red flag signs/symptoms).
C. Consider the following tests (after radiography is completed):
  1. MRI to rule out disc disease and tumors.
  2. Bone scan to rule out cancer.

DIFFERENTIAL DIAGNOSES

A. Back pain secondary to musculoskeletal pain.
B. Herniated intervertebral disease.
C. Sciatica.
D. Fracture.
E. Tumor.
F. Abdominal aneurysm.
G. Pyelonephritis.
H. Metabolic bone disease.
I. Gynecologic disease.
J. Peripheral neuropathy.
K. Depression.
L. Prostatitis.
M. Spinal stenosis.
N. Osteoarthritis.
O. Osteoporosis.
P. Ankylosing spondylitis.

PLAN

A. General interventions:

1. The client should continue physical activity as tolerated. Guidelines indicate that bedrest should be avoided.

2. For acute muscle strain, have the client apply local cold packs for 20 to 30 minutes several times a day for the first 24 hours. Heat packs are recommended after the initial 24 hours of injury.

3. Chronic or recurrent pain may be treated with either ice or heat applications, whichever gives relief. Other chronic LBP treatment methods, other than pharmacology, include physical therapy, cognitive behavioral therapy, acupuncture, radiofrequency ablation, facet injections, spinal cord stimulation, and surgical intervention. Referral to the appropriate specialty provider is indicated.

B. Client teaching:

1. Give accurate information on the prognosis for quick recovery, such as continuing light physical activity, performing back-strengthening exercises, and avoiding overuse of medications.

2. Improvement occurs in most cases in a few weeks, although mild symptoms may persist.

3. Joint guidelines by the American College of Physicians and the American Pain Society recommend rehabilitative therapies for clients who do not improve after medications and self-care recommendations. Rehabilitative therapies include exercise therapy, acupuncture, massage therapy, spinal manipulation, cognitive behavioral therapy, and yoga.

4. Provide educational handouts on back exercises; *see Client Teaching Guide for* Chapter 21, *"Back Stretches."*

5. After intense pain abates, the client may perform low back exercises for range of motion and strengthening, and isometric tightening exercises of abdominal and gluteal muscles.

6. Teach client knee–chest exercises. Recommend to the client to place their back against the wall and contract abdominal and gluteal muscles with 5 to 10 repetitions four to six times per day.

7. Research indicates that yoga is beneficial for many types of back pain. Types of back pain benefited by yoga include musculoskeletal injury, herniated disc, spinal stenosis, spondylolisthesis, piriformis syndrome, arthritis, and sacroiliac joint derangement.

8. Encourage the client to perform walking exercise daily.

9. Teach relaxation techniques.

10. Encourage the client to modify work hours and job tasks.

11. Refer the client for therapeutic massage or physical therapy as needed.

12. Obesity is often related to decreased exercise and poor physical fitness with reduced trunk muscle strength and endurance. Obese clients may experience back pain with normal activity.

C. Pharmaceutical therapy:

1. Analgesics: Analgesics include acetaminophen 325 to 650 mg every 4 to 6 hours. Maximum dose is 4,000 mg/d for the average adult. Inquire of any other current medications and/or over-the-counter preparations containing acetaminophen.

2. Nonsteroidal anti-inflammatory drugs (NSAIDs), unless contraindicated due to gastrointestinal symptoms or cardiovascular disease:

a. Aspirin: 325 to 650 mg every 4 to 6 hours.

b. Ibuprofen: 400 to 800 mg every 6 to 8 hours. Maximum dose is 3.2 g/d under the care of the provider; otherwise 1.2 g/d.

c. Naproxen: 250 to 500 mg Q12H (maximum dose of 1,500 mg/d).

d. Piroxicam (Feldene): 10 to 20 mg every day (progressive response to steady state over 7 to 10 days).

e. Meloxicam (Mobic): 7.5 to 15 mg daily (maximum dose of 15 mg/d; dialysis clients only 7.5 mg/d).

f. Celecoxib (Celebrex): 100 to 200 mg BID (maximum dose of 400 mg/d).

g. Diclofenac: 50 mg two to three times per day (maximum dose of 150 mg/d).

3. Muscle relaxants:

a. Cyclobenzaprine HCl (Flexeril): 5 to 10 mg oral up to three times daily as needed.

b. Carisoprodol (Soma): 350 mg QID.

c. Methocarbamol (Robaxin): 1.5 g every day initially, then 750 to 1,000 mg every day.

d. Orphenadrine citrate (Norflex): 100 mg BID.

e. Metaxalone (Skelaxin): 800 mg three to four times a day.

FOLLOW-UP

A. If pain is severe or unimproved, follow up in 24 hours.

B. If pain is moderate, reevaluate client in 7 to 10 days.

C. See client in 2 to 4 weeks to reevaluate condition and behavioral changes.

D. Recurrences are not uncommon but do not indicate a chronic or worsening case.

CONSULTATION/REFERRAL

A. Consult with a physician/specialist when considering red flag diagnoses such as cauda equina syndrome, herniated disc, widespread neurologic involvement, carcinoma, or significant trauma.

B. Referral to a physician/specialist is recommended for clients who have symptoms indicating other conditions such as inflammatory disorders (e.g., ankylosing spondylitis, rheumatoid arthritis, iritis). Symptoms may include morning stiffness with gradual onset prior to age of 40 years, synovitis (inflammation of peripheral joints) skin rashes, and so forth.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Pregnancy is often associated with low back discomfort. This is due to the redistribution of body weight. As weight increases in the abdominal area with the growing fetus, clients tend to compensate by changing posture and tilting their spine back.

B. Adults:

1. Refer to the abovementioned material on chronic pain in the elderly. For clients older than 50 years of age presenting with no history of backache, consider a differential diagnosis of neoplasm.

2. The most common metastasis seen is secondary to the primary site of breast cancer, prostate cancer, or multiple myeloma. Pain most prominent in a recumbent position rarely radiates into the buttock or leg.

C. Geriatrics:

1. LBP is the leading cause of disability worldwide. Geriatric clients who are at the highest risk of LBP are those who had physically demanding jobs as well as those who smoke, are obese, and/or have physical/ ▶

mental comorbidities. Disabilities and costs are expected to increase in the coming decade secondary to the rising older adult population within a burdened healthcare system.

2. Studies indicate that LBP is a common reason for geriatrics to visit the ED. The prevalence of LBP is high in older adults primarily because it is underdiagnosed and/or undertreated. Approximately 60% to 80% of geriatric clients experience LBP through their lifetime, with 69% stating pain was in multiple sites, not just one. Acetaminophen is recommended as first-line treatment and management of persistent LBP.

3. Although low-dosed opioids may be considered for geriatrics with spinal pain and LBP, clinical guidelines recommend trying nonpharmacologic treatments or physical therapy/exercise adjuvant with acetaminophen first, then antidepressants/gabapentin, and lastly low-dosed opioids.

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CLIENT TEACHING GUIDE

CHRONIC PAIN

You have been diagnosed with a chronic pain syndrome. This type of pain lasts longer than 3 months. This pain may be constant or it may come and go.

[ ] Apply ice (apply an ice pack to the affected area for 15 to 20 minutes) _____ times per day.

[ ] Apply heat (warm compress or heating pad 20 to 30 minutes) _____ times per day.

[ ] Physical therapy as directed _____ .

[ ] Complete the following exercises [ ] daily _____ [ ] at least three times per week [ ] as directed.

_____

[ ] Avoid caffeine in the afternoon and evening.

[ ] Rest at the beginning of the pain sequence.

[ ] You have been prescribed:

_____. Please take only as directed.

[ ] You have been prescribed a narcotic. Please follow these instructions:
A. You may need to increase fiber or take a stool softener to prevent constipation.
B. Do not drink alcohol.
C. This medication may make you drowsy or dizzy. It may impair your ability to drive or work and it may increase your risk of falling.
D. Never share this medication; keep out of reach of children and store in a safe location.

[ ] You have been referred to a pain management specialist for further evaluation and treatment.

[ ] Call your provider or go to the ED if any of the following occur:
A. Your heart rate is too slow or is fluttering too fast.
B. You develop side effects of the pain medication, such as itching, severe nausea, vomiting, or rash.
C. Your pain gets worse even though you are taking the medication correctly and as prescribed.
D. You cannot think clearly, become extremely dizzy, or begin falling more frequently.

RESOURCES Many resources on pain are available at your local library, bookstores, and on the internet. Look for a local support group in your area to join and learn how other people are coping with the condition same as yours.

There are many pain organizations available to assist clients. Clients may wish to visit the following websites for further information:
A. American Academy of Pain Management: www.aapainmanage.org.
B. American Chronic Pain Association: www.theacpa.org.
C. Arthritis Foundation: www.arthritis.org.
D. National Chronic Pain Outreach Association: www.healthcentral.com/chronic-pain/h/national-chronic-pain-outreach-association.html.
E. National Fibromyalgia Association: www.fmaware.org.
F. A nice resource available with many of the different types of logs and tools to use can be found at www.painmed.org/patientcenter/tools/.

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CHAPTER 7

DERMATOLOGY GUIDELINES

Jill C. Cash and Amy C. Bruggemann

ACNE ROSACEA

DEFINITION

A. A multivariate skin disease with phenotype manifest, acne rosacea is characterized by chronic and relapsing inflammatory processes in which flushing and dilation of the blood vessels occur on the face.

INCIDENCE

A. Acne rosacea affects approximately 13 million people in the United States.

PATHOGENESIS

A. The origination and development of rosacea are poorly understood. Abnormalities in the innate immune system, inflammatory reactions to cutaneous organisms, UV exposure, genetic factors, and vascular hyperreactivity have all been identified as possible causative factors.

PREDISPOSING FACTORS

A. Tendency to flush frequently.
B. Exposure to heat, cold, or sunlight.
C. Consumption of hot or spicy foods.
D. Some topical medications, astringents, or toners.
E. Acute psychological stressors (e.g., anger, rage, embarrassment).
F. Menopausal hot flashes.
G. Hormonal changes of pregnancy.
H. Alcoholic beverages and nicotine.
I. Certain drugs, such as nicotinic acid and vasodilators.

COMMON COMPLAINTS

A. Diagnostic phenotypes:
  1. Persistent centrofacial redness that may repeatedly accentuate phymatous changes.
B. Major phenotypes:
  1. Papules.
  2. Pustules.
  3. Flushing.
  4. Telangiectasia.
  5. Ocular manifestations.
C. Secondary phenotypes:
  1. Burning.
  2. Stinging.
  3. Facial edema.

SUBJECTIVE DATA

A. Ask the client to describe the location and the onset. Was the onset sudden or gradual? How have the symptoms continued to develop?
B. Assess if the skin is itchy or painful.
C. Assess for any associated discharge (blood or pus).
D. Complete a drug history. Has the client recently taken any antibiotics or other medications?
E. Determine whether the client has used any topical medications, astringents, toners, or new skin care products.
F. Rule out any possible exposure to industrial or domestic toxins, insect bites, and possible contact with venereal disease or HIV.
G. Ask the client about close contact with others with skin disorders.
H. Identify whether exposure to heat, cold, or sunlight provokes the symptoms.
I. Ask whether eating or drinking hot or spicy foods or consumption of alcoholic beverages provokes the symptoms.
J. Identify current or smoking history.

PHYSICAL EXAMINATION

A. Check temperature, pulse, and blood pressure.
B. Inspect:
  1. Inspect skin, focusing on face and scalp.
  2. Inspect nose and paranasal structures.
  3. Inspect eyes, eyelids, conjunctiva, and cornea. **An ocular manifestation, rosacea keratitis, may cause corneal ulcers to develop.**

DIAGNOSTIC TESTS

A. None.
B. Consider skin biopsy to rule out granulomatous rosacea, if suspected.

DIFFERENTIAL DIAGNOSES

A. Clinical findings shared with centrofacial erythema:
  1. Acne rosacea.
  2. Sun-damaged skin.
  3. Seborrheic dermatitis.
  4. Acute cutaneous lupus erythematosus.
  5. Dermatomyositis.
B. Clinical findings shared with papules and pustules:
  1. Acne rosacea.
  2. Acne vulgaris.
  3. Steroid-induced acne. ▶

The contributions of Cheryl Glass to this chapter in prior editions are acknowledged here.

4. Perioral dermatitis.
5. Keratosis pilaris rubra faceii.
6. Demodicosis.
C. Granulomatous rosacea.
D. Pyoderma faciale.

PLAN

A. General interventions:
1. Identify any causative or provocative factors: heat, cold, hot or spicy foods, alcoholic beverages, nicotine, sunlight.
2. Advise washing the face with mild soap, such as Cetaphil soap, daily.
3. Avoid direct sunlight exposure; wear protective clothing/hats when outdoors. Suggest using a sunscreen with sun protection factor (SPF) 30 when exposed to sunlight.
B. Client teaching: *See Client Teaching Guide for this chapter, "Acne Rosacea."*
C. Pharmaceutical therapy:
1. Rosacea with persistent facial erythema, **no** papules or pustules:
a. Topical brimonidine 0.33% gel.
b. Oxymetazoline 1% cream.
2. Rosacea with persistent facial erythema **and** papules or pustules:
a. Mild to moderate disease:
i. Topical metronidazole gel 0.75% apply BID or 1.0% apply daily.
ii. Azelaic acid 20% cream or 15% foam apply BID.
iii. Ivermectin 1% cream apply daily.
b. Moderate to severe disease:
i. Initial treatment options:
1) Tetracycline 250 to 500 mg BID for 4 to 12 weeks.
2) Doxycycline 50 to 100 mg BID for 4 to 12 weeks.
3) Minocycline 50 to 100 mg BID for 4 to 12 weeks.
ii. Maintenance treatment:
1) Topical metronidazole gel 0.75% BID.
2) Azelaic acid cream 15% apply BID.
3. Other:
a. Do not use topical steroids. Topical steroids may worsen irritation.
b. Refractory cases may respond to isotretinoin (Accutane).

FOLLOW-UP

A. Follow up in 2 weeks to evaluate therapy.
B. See clients monthly for evaluation until maintenance is reached.
C. Relapses are common following discontinuance of antibiotics; repeat treatment.

CONSULTATION/REFERRAL

A. Consult or refer the client to a dermatologist if there is no improvement or if the client is unable to reach maintenance.
B. Provide an immediate referral to an ophthalmologist for treatment and follow-up if the eye is involved.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Precautions should be used when prescribing medications for treatment during pregnancy.
2. Hormonal change can cause exacerbation. Topical preparation is first-line treatment. Metronidazole topical is thought to be safe in pregnancy.
3. The benefits of the use of steroids during pregnancy must outweigh the risks to be used. Risks of use include hypertension, intrauterine growth restriction, and diabetes.
B. Geriatrics:
1. Recent studies indicated rosacea is a systemic disorder associated with dyslipidemia, HTN metabolic disorders, cardiovascular disease, and alcohol/tobacco use, which are all prevalent in chronic kidney disease (CKD). Geriatric populations with a high rosacea severity have an increased risk of CKD and need ongoing evaluation of renal function labs: creatinine, blood urea nitrogen (BUN), and glomerular filtration rate (GFR).
2. Research suggested that rosacea is an independent risk factor for Parkinson disease through association with pathogenic mechanisms of elevated metalloproteinase activity. There was a twofold risk of Parkinson disease in clients with ocular rosacea and treatment of tetracycline appeared to reduce the risk of developing Parkinson disease. Practitioners must keep consistent skin assessments with clients who suffer from Parkinson disease and discuss treatment options with families and caregivers.
3. Monitor geriatrics for trigger medications that can worsen rosacea: vasodilators, niacin, or topical steroids.
4. Encourage elderly clients to use sunblock with titanium and/or zinc oxide on areas of the scalp with alopecia. Remind them that stress, hot/humid environments, alcohol, nicotine, spicy foods, and hot drinks can trigger a rosacea outbreak.
5. Avoidance of oil-based cosmetics and use of mild, nondrying soap is recommended.

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ACNE VULGARIS

DEFINITION

A. Acne vulgaris is a disorder of the sebaceous glands and hair follicles of the skin, which are most numerous on the face, back, and chest. The sebaceous glands become inflamed and form papules, pustules, cysts, open or closed comedones, and/or nodules on an erythemic base. In severe cases, scarring can result.

INCIDENCE

A. Nearly 80% to 90% of all adults experience acne during their lifetime. Acne vulgaris, commonly seen in adolescence, may even extend into the third or fourth decade of life.

PATHOGENESIS

A. Sebum is overproduced and collects in the sebaceous gland. Sebum, keratinized cells, and hair collect in the follicle. ▶

With *Propionibacterium acnes* (also known as *Cutibacterium acnes*) present, the duct becomes clogged and lesions (noninflammatory and/or inflammatory) evolve.

PREDISPOSING FACTORS

A. Age (adolescence).
B. External irritants to skin (makeup, oils, equipment contact on skin).
C. Hormones (oral contraceptives with high progestin content).
D. Medications (lithium, halides, hydantoin derivatives, rifampin).
E. Hot, humid weather.
F. Mask use.

COMMON COMPLAINTS

A. Outbreak of pimples on face, chest, shoulders, and back that does not resolve with over-the-counter (OTC) treatment.
B. Acne rosacea: telangiectasia, flushing, and rhinophyma present.

OTHER SIGNS AND SYMPTOMS

A. Mild: comedones open (blackhead) and closed (whitehead).
B. Moderate: comedones with papules and pustules.
C. Severe: nodules, cysts, and scars.

SUBJECTIVE DATA

A. Elicit the age of onset of outbreak, duration, and course of symptoms.
B. Determine what makes the lesions worse or better.
C. Ask whether there are certain times of the month of year when lesions are better or worse.
D. Identify the client's current method of cleanser or moisturizer treatment.
E. Ask if the client has ever been treated by a provider for this problem. If so, determine the treatment and results of the treatment.
F. Assess whether other family members have this same problem.
G. Ask the client for a description of the their environment and occupation.
H. Explore with the client any current stress factors in their life.
I. Inquire about the extent of mask use and frequency of washing and/or replacement.

PHYSICAL EXAMINATION

A. Inspect:
  1. Observe skin for location and severity of lesions.
  2. Rate the severity of the lesions as mild, moderate, or severe:
    a. Mild: few papules/pustules, no nodules.
    b. Moderate: several papules/pustules, rare nodules.
    c. Severe: many papules/pustules with many nodules.
  3. Take a picture of areas of affected skin for chart and document date. Use this for future appointments as a reference to compare results for follow-up visits.

DIAGNOSTIC TESTS

A. No tests are generally required.
B. Culture lesions to rule out gram-negative folliculitis with clients on antibiotics.
C. Consider hormone testing if other primary causes of acne are considered (follicle-stimulating hormone, luteinizing hormone, testosterone levels).

DIFFERENTIAL DIAGNOSES

A. Acne vulgaris.
B. Acne rosacea.
C. Steroid rosacea.
D. Folliculitis.
E. Perioral acne.
F. Drug-induced acne.
G. Sebaceous hyperplasia.
H. Keratosis pilaris.
I. Hidradenitis suppurativa.
J. Acne cosmetica.
K. Tropical acne.

PLAN

A. General interventions:
  1. Document location and severity of lesions. Assess the quality of improvement at each office visit.
  2. The primary goal of treatment is prevention of scarring. Good control of lesions during puberty and early adulthood is required for best results. Anticipate ups and downs during the normal course and treatment.
B. Client teaching: *See Client Teaching Guide for this chapter, "Acne Vulgaris."*
  1. Instruct the client on the proper cleansing routine. The client should wash affected areas with mild soap (Purpose, Cetaphil) twice a day and apply medications as directed.
  2. Warn the client that washing the face more than two to three times a day can decrease oil production and cause drying.
  3. Discuss current stressors in the client's life and discuss treatment options.
  4. Recommend exercise routine 3 to 5 days a week.
  5. Recommend oil-free sunscreens. UV light is beneficial; however, there is a need to use with caution when retinoids and tetracycline have been prescribed.
C. Pharmaceutical therapy: **It may take 2 to 3 months before results are visible when using medications:**
  1. Mild: The treatment of choice is combination topical:
    a. Benzoyl peroxide, 2.5%, 5%, and 10%; start with 2.5% at bedtime. May graduate to 5% or 10% BID if needed, as tolerated. When ordering with Retin-A, apply the benzoyl peroxide in the morning and Retin-A at night. Combination therapy of Retin-A and/or topical antibiotic is more effective.
    b. Topical tretinoin 0.1% (Retin-A Micro); use at bedtime. Apply 20 to 30 minutes after washing skin:
      i. With Retin-A use, the client may see rapid turnover of keratin plugs.
      ii. Instruct the client to avoid abrasive soaps.
      iii. Warn the client regarding photosensitivity.
    c. Clindamycin gel 1% applied once daily.
    d. Erythromycin gel 3% applied once daily.
  2. Moderate: Use the previous topical retinoid and benzoyl peroxide medications in addition to one of the following oral medications:
    a. Doxycycline 50 to 100 mg BID or 100 mg once daily treatment of choice.
    b. Tetracycline 500 mg BID:
      i. Instruct the client to take tetracycline on an empty stomach and to avoid dairy products, antacids, and iron.
      ii. Warn the client about photosensitivity. This medication may be used as a maintenance dose at ▶

250 mg daily or every other day for clients who break out after discontinuing antibiotic therapy. No drug resistance is seen with tetracycline.

c. Minocycline 50 mg taken one to three times daily:

i. Have the client drink plenty of fluids.

ii. Central nervous system (CNS) side effects (headaches) have been seen.

d. Oral contraceptives with higher doses of estrogen have also been effective in females.

3. Severe: Medications as prescribed per dermatologist.

FOLLOW-UP

A. See clients every 6 to 8 weeks for evaluation.

1. Mild: Adjust dose depending on local irritation.

2. Moderate (oral and topical medications):

a. Adjust dose according to irritation.

b. Taper oral antibiotics with discretion and/or continue topical medications.

c. Oral antibiotics may be tapered and discontinued when inflammatory lesions have resolved.

CONSULTATION/REFERRAL

A. Consult with a physician if treatment is unsuccessful after 10 to 12 weeks of therapy or if acne is severe.

B. The client may need dermatology consultation.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Acne may flare up or improve during pregnancy, may improve in the first trimester, and worsen in the third trimester.

2. Medications preferred during pregnancy are topical agents.

3. Teratogens include tretinoin, tetracycline, and minocycline.

a. When using teratogenic medications, contraception must be practiced to avoid pregnancy to prevent severe fetal malformations.

b. Begin contraception 1 month prior to starting the medication and 1 month after finishing the medication.

B. Geriatrics:

1. Favre-Racouchot syndrome may present as acne look-alike and needs to be evaluated. The causes of this are sun exposure with aging, environmental exposures, nutrition, and skin type.

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ATOPIC DERMATITIS

DEFINITION

A. This pattern of skin inflammation has clinical features of erythema, itching, scaling, lichenification, papules, and vesicles in various combinations. Currently, the term *eczema* is used interchangeably with *dermatitis.* Most common variants are atopic dermatitis and atopic eczema. Classification is by cause, either endogenous or exogenous.

INCIDENCE

A. Overall prevalence of all forms of eczema is about 18 in 1,000 in the United States.

B. With atopic dermatitis, 60% of those affected become afflicted between infancy and 12 years of age. It is more common in boys.

PATHOGENESIS

A. Eczema is characterized by a lymphohistiocytic infiltration around the upper dermal vessels. Epidermal spongiosis or intercellular epidermal edema and inflammation is seen.

PREDISPOSING FACTORS

A. Family history of atopic triad: dermatitis, asthma, and allergic rhinitis.

B. Exposure to allergen:

1. Common foods: cow's milk, nuts, wheat, soy, and fish.

2. Common environmental allergens: dust, mold, cat dander, and low humidity (dry air).

C. Exposure to topical medications, most commonly neomycin, lanolin, and topical anesthetics like benzocaine.

D. Skin irritants: harsh soaps, skin-care products with perfumes, chemicals and alcohol, fabrics containing wool, tight clothing.

E. Stress.

COMMON COMPLAINTS

A. Skin changes:

1. Itching, impossible to relieve.

2. Dryness.

3. Discoloration, lichenification, and scaling.

4. Skin thickening.

5. Associated bleeding and oozing skin.

OTHER SIGNS AND SYMPTOMS

A. Primary lesions, papules, and pustules that may lead to excoriation.

B. Adults will have lesions on face, trunk, neck, and genital area.

C. Other common features include infraorbital fold (Dennie sign), increased palmar creases, facial erythema, and scaling.

SUBJECTIVE DATA

A. Determine whether the onset was sudden or gradual.

B. Ask the client if the skin is itchy or painful.

C. Assess if there is any associated discharge (blood or pus).

D. Ask if the client has recently taken any antibiotics, other oral drugs, or topical medications.

E. Ask the client about use of soaps, creams, or lotions.

F. Assess for any preceding systemic symptoms (fever, sore throat, anorexia, vaginal discharge).

G. Ask the client about recent travel abroad.

H. Rule out insect bites.

I. Rule out any possible exposure to industrial or domestic toxins.

J. Elicit what precipitates itching.

K. Evaluate for increased stress level at home, work, relationships, and so forth.

PHYSICAL EXAMINATION

A. Check temperature (if indicated).

B. Inspect.

1. Inspect skin for lesions.

2. Recognize bacteria-infected eczema; *Staphylococcus aureus* is the most common pathogen. It appears with acute weeping dermatitis; crusted and small, superficial pustules.

DIAGNOSTIC TESTS

A. Culture skin lesions to determine viral, bacterial, or fungal etiology.

B. Blood work: Serum immunoglobulin E (IgE) is elevated with atopic dermatitis.

DIFFERENTIAL DIAGNOSES

A. Atopic dermatitis, acute or chronic.
B. Allergic or irritant contact dermatitis, acute or chronic.
C. Seborrheic dermatitis.
D. Ichthyosis vulgaris.
E. Bacterial/fungal infections.
F. Neoplastic disease.
G. Immunologic and metabolic disorders.
H. Psoriasis.
I. Scabies.

PLAN

A. General interventions:

1. Frequently treat the dry skin with emollients (Aquaphor, Eucerin).

2. Pat, do not rub skin.

3. Avoid wool products and lanolin preparations.

4. Keep fingernails cut short to prevent scratching/scarring skin.

5. Treatment of secondary bacterial infections may be needed as appropriate.

6. Eliminate trigger foods one at a time for 1 month to see improvement. Begin with eliminating cow's milk products. Consider soy-based foods instead.

7. Allergy testing may be considered if symptoms continue.

8. Ointments are usually recommended over creams for moisturizing.

B. Client teaching: *See Client Teaching Guide for this chapter, "Eczema."*

C. Pharmaceutical therapy:

1. Atopic: acute, adult:

a. Wet dressings with Burow solution and changed every 2 to 3 hours.

b. Topical corticosteroid:

i. Mild (low potency): desonide 0.05% or hydrocortisone 2.5% one to two times per day for 2 to 4 weeks.

ii. Moderate (medium to high potency): fluocinolone 0.025%, triamcinolone 0.1%, betamethasone dipropionate 0.05% one to two times per day up to 2 weeks.

c. Antihistamine of choice: Cetirizine HCl (Zyrtec) or diphenhydramine HCl (Benadryl).

d. Severe cases: oral steroid: prednisone 1 mg/kg (40–60 mg/d) tapered over 2 to 3 weeks.

2. Atopic: chronic, adult:

a. Short course of potent topical corticosteroid betamethasone dipropionate (Diprolene) or clobetasol propionate (Temovate) BID for 7 days.

3. Secondary bacterial infections:

a. Antibacterial treatments for secondary bacterial infections: Treat per culture and sensitivity results:

i. *S. aureus:*

1) Augmentin 875 mg PO BID for 10 to 14 days.

2) Keflex 500 mg PO QID for 10 to 14 days.

3) Erythromycin 500 mg PO QID for 10 to 14 days. OR

4) Dicloxacillin 250 mg Q6H for 10 days.

FOLLOW-UP

A. See client in office in 1 to 2 weeks and then every month until condition is stabilized.

B. Monitor the client for superimposed staphylococcal infection; may use oral erythromycin or dicloxacillin.

C. Client may be seen every 3 to 6 months thereafter for client education updates.

CONSULTATION/REFERRAL

A. Eczema herpeticum (herpes simplex type 1) may progress rapidly. Refer the client to a dermatologist.

B. Refer the client to a dermatologist if:

1. Diagnosis is uncertain.

2. Skin eruptions are severe or fail to respond to conservative treatment.

3. Second-line therapies may be required, such as phototherapy, cyclosporine, or methotrexate.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Avoid oral steroids; if needed, can use mild to moderate strength steroid cream.

2. Hormonal change can flare eczema; keep the skin moisturized.

B. Young adults and older adults:

1. Nummular eczema is commonly seen, characterized by coin-shaped vesicles and papules seen on extremities and/or trunk.

D. Geriatrics:

1. Avoid first-generation anticholinergics due to risk of confusion, dry mouth, constipation, and other anticholinergic effects or toxicity.

2. Research indicates that atopic dermatitis in older adult clients shows high rates of positivity for IgE antibodies against house dust mites and is associated with IgE allergic asthmatic exacerbations. In moderate to severe cases with geriatric clients suffering from atopic dermatitis, oral corticosteroids combined with standard treatments may be needed; however, use sparingly.

3. Avoid higher potency steroid topicals to face and skin folds of older adults and use no more than 5 to 7 days. Skin atrophy could result from overuse.

4. Avoid heat and prolonged hot showers and baths.

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BENIGN SKIN LESIONS

DEFINITION

A. A benign skin lesion is a cutaneous growth with no harmful effects to the body. Benign lesions must be distinguished from the following:

1. Basal cell carcinoma (BCC): nodular tumor with pearly surface, telangiectasia on surface, and depressed center or rolled edge.

2. Squamous cell carcinoma (SCC): irregular papule, with scaly, friable, bleeding surface.

3. Malignant melanoma: asymmetric papule, with irregular border, of two or more colors, and greater than 6 mm in diameter.

INCIDENCE

A. Benign lesions are common to all races and are seen primarily in the adult and older adult populations.

PATHOGENESIS

A. The course varies depending on the specific type of lesion.

PREDISPOSING FACTORS

A. Sun exposure in the adult and older adult populations.

B. Dermatosis papulosa nigra: common in African Americans and Asians.

COMMON COMPLAINT

A. New lesion on the skin.

OTHER SIGNS AND SYMPTOMS

A. Seborrheic keratosis: Waxy papule with a stuck-on appearance is seen in adults on sun-exposed areas; they appear symmetric, 0.2 to 3.0 cm in size, with a well-demarcated border and a variety of colors (tan, black, and brown).

B. Dermatosis papulosa nigra: Hyperpigmented mole is located on face or neck; a pedunculated papule is symmetric, 1 to 3 mm in diameter.

C. Cherry angioma: Vascular papule, red to purple, is located on trunk in adults; begins in early adulthood; 1- to 3-mm diameter papules that do not blanch.

D. Solar lentigines (liver spots): Tan maculae appear on sun-exposed areas in the elderly, especially on face and hands; border is irregular and the size varies.

E. Senile sebaceous hyperplasia: Enlarged sebaceous glands appear as yellow papules on sun-exposed areas, especially on face in the elderly; papules have central umbilication and their size varies.

F. Keratoacanthoma: Sun-exposed area lesion is smooth, skin-colored or reddish in appearance, dome-shaped papule at first, then may turn and grows to 1 to 2 cm in a few weeks, with crusted interior.

SUBJECTIVE DATA

A. Identify when the client first discovered the lesion.

B. Determine whether the lesion has changed in size, shape, or color.

C. Ask if the client has discovered more lesions.

D. Elicit information regarding a family history of skin lesions or cancer.

PHYSICAL EXAMINATION

A. Inspect:

1. Observe skin; note all lesions and evaluate each for asymmetry, border, color, diameter, evolving changes, and/or elevation change.

2. Note the client's skin type.

DIAGNOSTIC TESTS

A. Benign lesions do not require any tests.

B. If unsure regarding possible malignancy, a biopsy is recommended.

DIFFERENTIAL DIAGNOSES

A. Benign skin lesion:

1. Seborrheic keratosis.

2. Dermatosis papulosa nigra.

3. Cherry angioma.

4. Solar lentigines.

5. Senile sebaceous hyperplasia.

6. Keratoacanthoma.

B. Malignant skin lesion.

PLAN

A. General interventions:

1. Reassure the client that lesions are benign. No treatment is required unless the client chooses to have the lesion removed for cosmetic purposes.

2. Lesions may be removed using cryotherapy if they are bothersome for the client.

B. Client teaching: *See Client Teaching Guide for this chapter, "Skin Care Assessment."*

C. Pharmaceutical therapy:

1. Topical 5-fluorouracil, 5% imiquimod cream (Aldara), or topical diclofenac gel may be used for benign lesions.

FOLLOW-UP

A. Routine skin exams should be performed yearly.

CONSULTATION/REFERRAL

A. Immediately refer the client to a dermatologist if malignancy is suspected or confirmed by biopsy.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. Skin lesions begin to appear in early adulthood. Encourage clients to monitor lesions over time.

B. Geriatrics:

1. Benign lesions are commonly seen in the older adult population.

a. Encourage annual/biannual skin screenings in older adult populations and the continual use of sunscreen with UVA and UVB coverage. Protective clothing and avoidance of sun exposure are highly recommended for geriatrics with a history of basal cell/benign lesions. Encourage wearing hat to cover bald spots and scalp from sun exposure as well.

b. Treatment of benign lesions for geriatrics must be balanced with client's preferences, quality of life, and life expectancy. Mohs micrographic surgery, cryosurgery, excision, photodynamic, and topical creams/solutions are acceptable treatments for geriatric clients if they choose. Monitor closely for allergic reactions or toxicity with topical medications: imiquimod (Aldara), 5-fluorouracil, and vismodegib (Erivedge).

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BITES

ANIMAL BITES, MAMMALIAN

DEFINITION

A. A bite from any mammalian animal to a human can be potentially dangerous. Bites from human to human are included.

INCIDENCE

A. Bites account for 2 to 5 million ED visits each year.
B. About 80% to 90% of bites are dog bites.
C. About 6% of bites are cat bites.
D. From 2% to 3% are human bites.
E. Older adults are especially prone to bites.

PATHOGENESIS

A. Mechanical trauma and break to skin and/or underlying structures.
B. Infection from transmission of bacteria:
1. *Pasteurella multocida* is primarily associated with cat bites but may also be associated with dog bites.
2. *Staphylococcus aureus, S. epidermidis*, and *Enterobacter* species can be transmitted with dog and cat bites.
3. *Streptobacillus moniliformis* can be transmitted with rat and mice bites.
4. *Streptococcus, Staphylococcus*, and *Eikenella* can be transmitted with human bites.
5. Human bites can transmit diseases such as actinomycosis, syphilis, tuberculosis, hepatitis B, and potentially HIV.
C. Rabies, an acute viral infection, may be transmitted by means of infected saliva or by an infected animal licking mucosa from an open wound. It is rarely contracted by means of airborne transmission, but this has been reported to occur in bat-infested caves.

PREDISPOSING FACTORS

A. Older adults.
B. Entering an animal's territorial space and/or surprising an animal.

COMMON COMPLAINTS

A. Bitten by an animal.
B. Pain.
C. Redness.
D. Swelling.

SUBJECTIVE DATA

A. What person or type of animal bit the client?
B. Was this a provoked or an unprovoked attack?
C. Did the client identify and contact the owner of the animal?
D. What was the behavior of the animal? Unusual, strange, or ill-appearing?
E. How much time has elapsed from being bitten to seeking treatment?
F. Did the client start any self-treatment?
G. What is the client's tetanus immunization status?
H. Review history for any prior rabies immunizations.
I. Does the client know if the animal was a domestic animal? Is the animal's vaccination status known?
J. If the bite is of human origin, determine if it is a closed-fist injury (a human bite/injury that occurs when the closed fist is injured, with teeth penetrating the skin, joint, and possibly bone, over the metacarpal heads) or plain bite.

PHYSICAL EXAMINATION

A. Check blood pressure, pulse, and respirations; observe overall respiratory status.
B. See Table 7.1.

DIAGNOSTIC TESTS

A. Refer to Table 7.1.

DIFFERENTIAL DIAGNOSES

A. Animal bite: dog, cat, human, and so forth.
1. Cat bites more frequently become infected.
2. Bites on the hand have the highest infection rates. Bites on the face have the lowest infection rates.
B. Cellulitis and abscesses.
C. High-risk potential for rabies from bites from the following: ▶

TABLE 7.1 BITES

Animal	Signs and Symptoms	Physical Exam[a]	Diagnostic Tests
Dog	Crush injury, lacerations, and abrasions	Inspect site, underlying structures, and distal neurovascular, motor, and sensory function. Palpate area. If wound is over 24 hours old, check any signs of cellulitis, lymphangitis.	If infected: laboratory—CBC, culture and sensitivity for anaerobes and aerobes
Cat	Puncture wounds, may be deep	Determine depth and extent of wound. Check for foreign bodies. If wound is over 24 hours old, check any signs of cellulitis, lymphangitis.	If sepsis suspected: laboratory—CBC, culture and sensitivity of abscess/tissue site
Rat and squirrel	Laceration, abrasions; more superficial in nature	Check for signs of infection. If wound is over 24 hours old, check any signs of cellulitis, lymphangitis.	If infected: laboratory—CBC, culture, and sensitivity
Human	Crush injury, laceration; wound in the hands (closed-fist wound)	Check for signs of infection. If the wound is over 24 hours old, check any signs of cellulitis and lymphangitis. Also, examine for fractures, air in the joint, subchondral bone defects, and osteomyelitis. Examine for full range of interphalangeal and metacarpophalangeal joints.	As above Also, take x-ray of structures underlying bite

[a] For all animal bites, check temperature.

CBC, complete blood count.

1. Skunks, foxes, raccoons, and bats are primary carriers.
2. Rabbits, squirrels, chipmunks, rats, and mice are seldom infective for rabies.
3. Properly vaccinated animals are seldom infective.

PLAN

A. General interventions:
1. Control bleeding.
2. Wound care:
   a. Immediately wash wound copiously with soap and water.
   b. Irrigate wound with saline, benzalkonium chloride (Zephiran), or 1% povidone-iodine (if the client is not allergic to iodine).
   c. May use Waterpik.
   d. Use 150 to 1,000 mL of saline solution.
   e. Direct stream on the entire wound surface.
   f. Scrub the entire surrounding area.
   g. Debride all wounds.
   h. Trim any jagged edges to prevent cosmetic and/or functional complications.
   i. Cover with dry dressing.
3. Do not suture wounds at high risk of infection:
   a. Hand bites, closed-fist injuries.
   b. Bites older than 12 hours.
   c. Deep or puncture wounds.
   d. Bites with extensive injury of surface or underlying structures.
   e. Immunocompromised clients.
   f. Lower extremity wounds in clients with venous or arterial compromise.
4. Rabies control measures:
   a. Consult with the local health department regarding risk of rabies in the area.
   b. The domestic animal should be identified, caught, and confined for 10 days of observation. If the animal develops any signs of rabies, it should be destroyed and its brain tissue analyzed. No treatment is necessary if results are negative.
   c. The wild animal should be caught and destroyed for brain tissue analysis. No treatment is necessary if the results are negative.
   d. If the bat or wild carnivore cannot be found, rabies prophylaxis is instituted.

B. Client teaching:
1. Instruct the client how to keep the site free from infection.

C. Pharmaceutical therapy:
1. Antibiotic prophylaxis:
   a. Antibiotic prophylaxis is controversial, but it is generally recommended for wounds involving subcutaneous tissues and deeper structures.
      i. Agent of choice: amoxicillin-clavulanate acid (Augmentin) 875/125 mg Q12H.
      ii. Alternatively you can prescribe (aerobic and anaerobic agents together to cover):
         1) Doxycycline 100 mg BID.
         2) Trimethoprim-sulfamethoxazole (TMP-SMX) double strength (DS) BID.
         3) Ciprofloxacin 500 to 750 mg BID.
         4) Levofloxacin 750 mg once daily.

         PLUS an anaerobic agent:
         5) Metronidazole 500 mg TID.
         6) Clindamycin 450 mg TID.
      iii. Alternatively, prescribe erythromycin (E-Mycin) 250 mg QID for 7 days.
2. Tetanus prophylaxis:
   a. Tetanus immune globulin and tetanus toxoid should be given to all clients who have had fewer than three primary immunizations.
   b. Tetanus toxoid alone can be given to those who previously have had the primary immunization, but have received a booster 5 or more years ago.
3. Rabies prophylaxis:
   a. Active immunization: Human diploid cell vaccine (HDCV), 1 mL, is given intramuscularly on the first day of treatment, and repeat doses are administered on days 3, 7, and 14. For persons with immunosuppression (corticosteroid use, immunosuppressive drugs, and immunocompromising illnesses), they will receive an additional dose on day 28.
   b. Passive immunization: Rabies immunoglobulin (RIG; human) should be used simultaneously with first dose of HDCV; the recommended dose of RIG is 20 IU/kg. Approximately one-half of RIG is infiltrated into wound and the remainder is given intramuscularly.

FOLLOW-UP

A. Evaluate the wound and change dressing in 24 to 48 hours.
B. Reevaluate as indicated. If the client is on immunoprophylaxis and has no signs of infection, see in 1 week.
C. Instruct the client to return immediately for any signs of infection.

CONSULTATION/REFERRAL

A. Refer all clients with bites in the ears, face, genitalia, hands, and feet to plastic surgery.
B. Consult with a doctor if there is suspicion of rabies.
C. Contact the local health department.
D. Wounds involving the tendon, joint, or bone require hospitalization and surgical consultation.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Use appropriate antibiotic management that is safe in pregnancy.

B. Geriatrics:
1. Consider chronic conditions (chronic kidney disease, diabetes, etc.) when prescribing the client antibiotics.
2. Pet therapy has been allowed in long-term care facilities, and studies suggested that 75% did not report health and safety concerns or had policies in place for animals. Be sure to discuss pet precautions (e.g., bite prevention, how to treat bites if they occur, and safety issues) with geriatric clients, whether they live at home, assisted living, or are residents of a long-term care facility.
3. Healing time for bite wound may be delayed due to age and chronic disease state.

INSECT BITES AND STINGS

DEFINITION

A. Bites and/or stings on the skin come from commonly encountered insects, bees, hornets, wasps, mosquitoes, chiggers, ticks, fleas, and fire ants.

INCIDENCE

A. Bites are seen in all age groups, more commonly in summer months.

PATHOGENESIS

A. Some bites elicit local tissue inflammation and destruction due to proteins and enzymes in the poison or venom of the insect.

B. Serum immunoglobulin E (IgE)-mediated allergic reactions (immediate or delayed) may occur.

C. Serum-sickness reaction may appear 10 to 14 days after a sting with venom. Toxic reactions can also occur from multiple stings, yielding large inoculation of poison or venom.

D. With tick bites, exposure to Rocky Mountain spotted fever, Lyme disease, alpha-gal allergy from Lone Star tick, ehrlichiosis, and babesiosis disease may occur.

PREDISPOSING FACTORS

A. Exposure to areas of heavy insect infestations.

B. Warm weather months.

C. Outdoor exposure with barefoot and bright clothes.

D. Use of perfumes and/or colognes.

E. Previous sensitization.

COMMON COMPLAINTS

A. Local reaction: pain, swelling, and redness at site after insect bite.

B. Toxic reaction: local reaction plus headache, vertigo, gastrointestinal symptoms (nausea, vomiting, diarrhea), syncope, convulsions, and/or fever.

SUBJECTIVE DATA

A. Did the client see what bit or stung them?

B. If the client felt the bite or sting, was they bitten or stung once or multiple times?

C. How long ago did it occur?

D. Where was the client when the injury occurred (environment)?

E. Has the client ever been bitten or stung before? If so, did they have any reaction then? If so, what was the treatment?

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure. Observe overall respiratory status.

B. Inspect:

1. Inspect site of injury for local reaction; note erythema, rash, or edema.

2. Perform ears, nose, and throat exam.

C. Auscultate:

1. Assess the heart and lungs.

E. Palpate:

1. Palpate injured site.

2. Assess nodes for lymphadenopathy.

3. Perform abdominal exam, if appropriate.

DIAGNOSTIC TESTS

A. None required.

B. Consider taking skin scrapings to evaluate under microscope.

C. Consider culture if infection is suspected.

DIFFERENTIAL DIAGNOSES

A. Insect bite:

1. Bees, hornets, wasps: Local pain, redness, pruritus, and swelling occur at site. Red papules and wheals appear, enlarge, and then subside within hours. Delayed hypersensitivity occurs within 7 days with enlarged, local reaction with fever, malaise, headache, arthralgias, and lymphadenopathy. Toxicity can occur. Anaphylaxis may be seen with generalized warmth and urticaria, erythema, angioedema, intestinal cramping, bronchospasm, laryngospasm, shock, and collapse.

2. Ticks: Local redness, swelling, itching, and an enlarged area of redness and swelling may occur.

3. Mosquitoes and chiggers: Local redness, swelling, and itching occur. Delayed reaction can include edema and burning sensation.

4. Fleas: Local redness, swelling, and itching occur. Usually, papules are noted in a zigzag pattern, especially on legs and waist. Note hemorrhagic puncta surrounded by erythematous and urticarial patches.

5. Body lice: Small noninflammatory red spots, intensely pruritic, are found on waist, shoulders, axilla, and neck. Note linear scratch marks. Note secondary bacterial infection.

6. Scabies: Pruritus is the dominant symptom. Note inflammation and burrows in skin with papules and vesicles, especially in the webs of the hands and feet.

7. Fire ants: Papules appear and turn to pustules within 6 to 24 hours after bite. Watch for localized necrosis with scarring. Urticaria and angioedema can occur.

B. Allergic reaction.

C. Folliculitis.

PLAN

A. General interventions:

1. Anaphylaxis: Activate emergency medical services (EMS) immediately. With all bites and stings, treat anaphylaxis first.

2. Local reactions: Treat with analgesic of choice. Apply ice packs to site for approximately 10 minutes. Elevate affected extremities.

3. Delayed reactions: Administer antihistamines as needed. Consider corticosteroid use.

4. Routine wound care: Cleanse wound. Remove the stinger. If it is a painful sting, apply a cotton ball soaked in meat tenderizer or sodium bicarbonate paste.

5. Debride as necessary.

6. For embedded insects, apply petroleum jelly, nail polish, or alcohol over the site for 30 minutes and wait for insect or the tick to withdraw.

7. Referral to allergist–immunologist is recommended for clients with a severe systemic reaction for skin testing and to evaluate for candidacy of venom immunotherapy treatment.

8. Hospitalize the client with severe reactions.

B. Client teaching: *See Client Teaching Guide for this chapter, "Insect Bites and Stings."*

C. Pharmaceutical therapy:

1. Mild anaphylaxis:

a. Epinephrine 1:1,000 aqueous solution: 0.3 to 0.5 mL intramuscular (IM). Repeat dosing may be necessary.

b. Oral antihistamines for the next 24 hours:

i. Cetirizine 10 g once daily or loratadine 10 mg once daily.

ii. Hydroxyzine hydrochloride (Atarax) 10 to 25 mg QID.

2. Severe anaphylaxis:

a. Epinephrine 1:1,000 aqueous solution: 0.3 to 0.5 mL IM. Repeat dosing may be necessary.

b. Oxygen 2 to 4 L as needed.

c. Albuterol (Ventolin) 5 mg/mL per dose by nebulizer:

i. Adults: 2.5 mg (0.5 mL of 0.5% solution) in 2-mL saline.

3. Self-treatment for anaphylaxis (emergency treatment kits):

a. Ana-Kit (epinephrine, chlorpheniramine) contains a preloaded syringe. This will relieve respiratory distress due to bronchospasms during the allergic reaction.

b. EpiPen (epinephrine) autoinjectors are spring-loaded automatic injectors.

FOLLOW-UP

A. Follow-up in 2 weeks to evaluate effectiveness of treatment. If symptoms worsen prior to this, evaluation is needed.

REFERRAL/CONSULTATION

A. Consult with a physician when anaphylaxis occurs.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Older adults, clients who are immobilized, those with preexisting heart disease or mast cell disorder, those being treated with certain medications (such as beta-adrenergic or angiotensin-converting enzyme [ACE] inhibitors), and those who have had a previous reaction to an insect bite are at the greatest risk of complications when an insect bite occurs.

2. Bites obtained from fire ants, bees, hornets, and wasps may result in severe stings that may even result in death. The honeybee sting is known to present the greatest risk of complications and/or anaphylaxis.

3. Occasionally, the geriatric population misinterprets symptoms of neuropathy or psychocutaneous sensations as sting/bites. Carefully assess the history of comorbidities and actively listen to the client's description. If necessary, discuss the client's descriptive symptoms with family/surrogates. If physical exam is stable and neuropathy has been ruled out or effectively treated, acknowledge the client's experience and consider psychiatric referral.

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BULLOUS PEMPHIGOID

DEFINITION

A. Bullous pemphigoid is an autoimmune skin condition that occurs primarily in older adults and is characterized by blisters and/or erosive lesions of the skin or mucosa.

INCIDENCE

A. Most cases occur in adults older than 60 years of age. Studies show that during 2000 to 2005, the incidence of bullous pemphigoid was approximately 22 cases per million people, which is three times greater than in previous years.

PATHOGENESIS

A. The mechanism of the physiologic changes that occur in the body to cause bullous pemphigoid is not fully understood. It is believed to be an autoimmune disorder driven by autoantibody-mediated effects that cause damage to the epithelial basement membrane. Antibodies attach to the antigens on the basement membrane, causing inflammatory changes, resulting in separation of the epidermis from the dermis in the skin and separation of the epithelium from the subepithelial tissue in the mucous membranes, producing blister lesions on the affected tissues.

PREDISPOSING FACTORS

A. Age (adults older than 60 years).

B. Exposure to certain medications (penicillin, etanercept [Enbrel], sulfasalazine [Azulfidine], and furosemide [Lasix]).

C. UV light and radiation therapy.

COMMON COMPLAINTS

A. Large, tense blisters commonly located on the arms, legs, or trunk. Commonly seen along skin folds/creases.

B. Blisters may be asymptomatic or may be erythemic and pruritic.

OTHER SIGNS AND SYMPTOMS

A. Irritation to the oral mucosa, mouth sores, and/or bleeding gums.

B. Rash in addition to skin blisters.

C. Changes in mucous membranes of the eyes, noting redness and discomfort.

POTENTIAL COMPLICATIONS

A. Bacterial skin infection.

B. Sepsis.

C. Scarring of the mucous membranes in the mouth or eye.

SUBJECTIVE DATA

A. Ask client if there was any precipitating event prior to the outbreak of the blisters.

B. Have client describe when the blisters began and the duration of the blisters.

C. Ask client to describe the onset of the presenting lesions and the progression of the lesions.

D. Ask if the blisters are painful. If so, describe pain, such as stabbing, burning, throbbing, and so forth.

E. Rate pain on a scale of 0 to 10, with 0 being no pain.

F. List all medications currently being taken, particularly substances not prescribed. Did the blisters begin after the initiation of any new medications?

PHYSICAL EXAMINATION

A. Assess vital signs: Check temperature, pulse, respirations, and blood pressure.

B. Inspect:

1. Inspect general appearance of the skin, noting types of blisters/lesions. Note the location of the blisters, along with erythema, inflammation, or weeping. Inspect skin folds carefully.

2. Inspect the blisters for staging.

3. Inspect oral cavity for oral lesions.

4. Perform ophthalmologic exam: Inspect eyes for erythema, edema, and ulcers.

DIAGNOSTIC TESTS

A. Biopsy: skin lesion biopsy for staining and for direct immunofluorescence (DIF) studies; the gold standard for diagnosis is a positive DIF.

B. Blood tests: peripheral blood smear; elevated eosinophils and increased immunoglobulin G (IgG) levels may be present.

DIFFERENTIAL DIAGNOSES

A. Bullous pemphigoid.
B. Cellulitis.
C. Dermatitis.
D. Shingles.

PLAN

A. General interventions:
  1. Promote healing of blisters and prevent secondary bacterial infection.
  2. The goal of treatment is to control blister formation.

B. Client teaching:
  1. Inform client regarding the course of bullous pemphigoid. This skin condition may last for months or years. Therefore, treatment options are variable for all clients.
  2. Advise the client to avoid sun exposure directly on the skin.
  3. For skin care, educate the client regarding taking care of the fragile skin, avoiding injury to all sites. Tense blisters should be cleansed well with alcohol and carefully ruptured with a sterile needle. The overlying tissue from the blister should remain intact to act as a natural wound cover. Open sores should be covered with a nonadherent bandage to reduce the risk of infection.
  4. Oral blisters should be protected by avoiding spicy or hard foods, which would cause more injury to the oral ulcers.

C. Pharmaceutical therapy:
  1. First-line therapy suggested is topical steroids. Topical steroids, such as clobetasol propionate 0.05% cream, are applied to the affected area BID until inflammation, new blister formation, and pruritus have resolved for at least 2 weeks. Then gradually taper the steroid cream over the next few weeks to months.
  2. If the topical steroids are not successful, oral steroids may be used. Prednisone 0.5 to 0.75 mg/kg/d may be used as a starting dose. Oral prednisone is used until inflammation, new blister formation, and pruritus have resolved for at least 2 weeks. Then gradually taper the prednisone over the next several weeks to months.
  3. In severe cases, immunosuppressants may be prescribed to help control the lesions. These medications may include methotrexate, azathioprine (Imuran), and mycophenolate mofetil (CellCept). Using these medications may also allow weaning of the use of corticosteroids.
  4. Medications with anti-inflammatory effects may also be used, such as tetracycline and dapsone.
  5. Antibiotics and niacin may be prescribed for signs of bacterial infection. Tetracycline antibiotics may be used for mild cases. Niacin may also be prescribed, along with tetracycline, to help control gastrointestinal side effects:
    a. **Clients who do not respond should be referred to a specialist.** Clients who do not respond to steroid treatments may be prescribed biologic therapy such as intravenous (IV) rituximab by the specialist.

FOLLOW-UP

A. For clients on oral or topical treatments, recommend a follow-up appointment in 1 to 2 weeks, depending on the presentation. If signs of bacterial infection are present, then follow-up would need to be sooner.

CONSULTATION/REFERRAL

A. For all clients who present with signs of bullous pemphigoid, consult with the physician or the dermatologist for treatment options.
B. Consult with the physician if signs of bacterial infection are present. In severe cases, sepsis can develop and become life-threatening for compromised clients.
C. The physician or the dermatologist should prescribe potent medications, such as immunosuppressants.
D. Refer any client who presents with any eye involvement to the ophthalmologist.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
  1. Rarely seen in this population.

C. Geriatrics:
  1. Most commonly seen in adults older than 60 years of age. Clients who are compromised or in poor health may develop a secondary bacterial infection, which can be life-threatening.
  2. Studies suggested that eczematous dermatitis in the older adult population is possibly an early indication of a bullous pemphigoid outbreak. Because the disease is closely associated with neurodegenerative diseases, consider collaboration with a neurologist and a dermatologist for treatment management.
  3. Two categories of medications should be screened for possibly causing the development of callous pemphigoid. diabetes medications in the dipeptidyl peptidase-4 inhibitor class such as Tradjenta and psychotropic medications such as Risperidone.
  4. Recommendations for treating older adults with bullous pemphigoid:
    a. First-line clobetasol propionate 0.025% cream topically (except the face).
    b. Second-line tetracycline and nicotinamide combination therapy.
    c. Third-line PO dapsone or PO/subcutaneous methotrexate weekly.
    d. Discontinue therapy if clinical remissions last a minimum of 6 weeks and client has a negative immunofluorescence test.

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CANDIDIASIS

DEFINITION

A. A fungal infection of the mucous membranes and/or skin, candidiasis is caused by the *Candida albicans* fungus and other fungal strains as well.

INCIDENCE

A. It occurs frequently in females, children, and older adults.

PATHOGENESIS

A. An overgrowth of *C. albicans* occurs when mucous membranes and/or skin are exposed to moisture, warmth, and an alteration in the membrane barrier.

PREDISPOSING FACTORS

A. Immunosuppression.
B. Use of antibiotics.
C. Hyperglycemia.
D. Chronic use of steroids.
E. Frequent douching by females.
F. Adults who wear dentures.
G. Urinary/fecal incontinence.

COMMON COMPLAINTS

A. Oral: persistent white patch on the tongue or roof of mouth, which may be slightly reddened with or without crevices on the tongue.
B. Vaginal: Thick, white, "cottage-cheese-like" vaginal discharge with or without vaginal itching.
C. Genital: bright, red rash with well-demarcated satellite lesions advancing to pustules or erosions in genital area.
D. Males: erythemic rash that may advance to erosions on the genitalia/scrotal area.

SUBJECTIVE DATA

A. Question the client about onset, duration, and location of lesions.
B. Determine whether the client has a history of infections.
C. Inquire into medical history and current medications.
D. Rule out the presence of any other current medical conditions.

PHYSICAL EXAMINATION

A. Inspect:
  1. Assess skin and mucous membranes for discharge and lesions.
  2. Observe location and severity of lesions. Document size, diameter, and characteristics of lesions.
B. Palpate:
  1. Palpate the lymph nodes in neck and groin.

DIAGNOSTIC TESTS

A. Vaginal and genital infections need to be evaluated for sexually transmitted infections (STIs), especially if the client is sexually active with multiple partners. Vaginal/genital culture specimen should be sent for gonorrhea/chlamydia testing.
B. Other specimens to consider include wet prep/potassium hydroxide (KOH) 10% solution, Gram stain vaginal culture for *Candida*.

DIFFERENTIAL DIAGNOSES

A. Oral candidiasis:
  1. Leukoplakia.
  2. Stomatitis.
B. Genital area:
  1. Candidiasis.
  2. Bacterial infection.
  3. Bacterial vaginosis.
  4. Chlamydia.
  5. Gonorrhea.
  6. Trichomoniasis.

PLAN

A. Client teaching:
  1. Instruct the client on how to apply medication to the skin.
  2. Advise the client to avoid scratching the site and to keep their fingernails short.
  3. Affected skin tissue should be kept cool and dry; avoid wearing tight and irritating clothing.
B. Pharmaceutical therapy: Choose *one* of the following pharmaceutical therapies:
  1. Oral:
    a. Nystatin (Mycostatin) oral suspension 100,000 U/mL QID for 7 to 10 days.
    b. Gentian violet aqueous solution, 2% for adults, one to two times per day.
    c. Lotrimin buccal troches, five times per day for 2 weeks.
  2. Topical:
    a. Nystatin cream, three to four times per day for 7 to 10 days.
    b. Mycolog-II: Apply sparingly to skin BID until resolved.
  3. Vaginal:
    a. Clotrimazole 1% cream, 5 g intravaginally for 4 to 7 days.
    b. Miconazole 2% cream, 5 g intravaginally for 7 days (over-the-counter [OTC]).
    c. Terconazole 0.8% cream, 5 g intravaginally for 3 days.
    d. Terconazole 80 mg vaginal suppository at bedtime for 3 days.
    e. Butoconazole 2% cream 100 mg intravaginal, for use one applicator, one time.
    f. Fluconazole (Diflucan) 150 mg, oral tablet one time.

FOLLOW-UP

A. None indicated unless not resolved or complications arise.

CONSULTATION/REFERRAL

A. Consult a physician if not resolved within 2 weeks.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
  1. Most effective medications for pregnant individuals are clotrimazole and miconazole.
  2. Recommend a full 7-day course of treatment during pregnancy.
B. Adults:
  1. Consider immunosuppression in all adults with oral candidiasis (HIV, diabetes, chemotherapy, leukemia).
  2. Adults with oral lesions need to be assessed for leukoplakia, especially if the client has a history of smoking or using chewing tobacco.
C. Geriatrics:
  1. Risk factors for fungal infections in older adults include denture wearers, intravascular catheters, residence in long-term care facility, renal failure, prolonged ICU stay, systemic administration of broad-spectrum antibiotics, parenteral nutrition, recent abdominal surgery, neutropenia, and use of corticosteroids.
  2. Studies posit that older adult populations are susceptible to bloodstream *Candida* infections (candidemia) characterized by a high mortality rate. To avoid candidemia, ▶

practitioners must treat fungal infections promptly, encourage prevention measures, and continually assess for relapses.

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CELLULITIS AND ABSCESS

Abscess

DEFINITION

A. An abscess is a pocket of pus under the skin tissue that may invade deeper skin tissue.

PATHOGENESIS

A. An abscess develops when a collection of bacteria builds up in the tissue of the body. There may be several types of bacteria present. One common organism is methicillin-resistant *Staphylococcus aureus* (MRSA).

COMMON COMPLAINTS

A. Painful, swollen, erythemic pocket of pus under the skin tissue.

SUBJECTIVE DATA

A. Ask the client when signs and symptoms of infection, discomfort, redness, or swelling began.

B. Has the client experienced fever, chills, or other symptoms?

C. What has been used to treat the abscess? Has heat, ice, topical antibiotics, lotions, and so forth been used?

D. What treatments make the symptoms worse or better?

PHYSICAL EXAMINATION

A. Inspect:

1. Assess site of abscess.

2. Measure: length x width.

3. Note erythema, edema, warmth/fever of tissue, tenderness with palpation. Does pain radiate to other areas? If so, where?

4. Is abscess open and draining fluid? If so, note color, consistency and odor of the drainage.

DIAGNOSTIC TESTS

A. Laboratory testing (complete blood count [CBC], comprehensive metabolic panel [CMP]).

B. Open or draining abscess: culture and sensitivity, and Gram stain.

DIFFERENTIAL DIAGNOSES

A. Wound abscess.
B. Cellulitis.
C. Folliculitis.
D. Pilonidal cyst.
E. Pressure injury.
F. Necrotizing fasciitis.
G. Gas gangrene.
H. Acute gout.

PLAN

A. General interventions:

1. Incision and drainage (I&D) can successfully be performed for an uncomplicated abscess.

2. Precautions should be considered for contraindications to an I&D. These contraindications include the following:

a. Abscess location (perirectal, neck, hand, central area of the face, breasts, and in close proximity to major nerves and blood vessels).

b. Recurrent abscesses.

3. After completing the I&D, the area should be allowed to close by secondary intention.

4. Sterile packing should be inserted into the cavity for abscesses larger than 5 cm in diameter and for clients with compromised immune systems.

B. Client teaching:

1. Educate the client regarding home care for the site of the abscess. If packing is required, instruct client to return to the physician's office for packing, or consider home healthcare to perform dressing changes.

2. New packing should be replaced every 24 to 48 hours until drainage has resolved and until new granulation tissue is present.

3. Once packing is no longer necessary, the client will clean the cavity with warm, wet compress two to three times a day until healed.

C. Pharmaceutical therapy:

1. Oral antibiotic treatments: Antibiotics should be prescribed per the culture and sensitivity results if available; if not available use antibiotics that are sensitive to MRSA. See pharmacologic treatment options in the "Cellulitis" section of this chapter.

FOLLOW-UP

A. The client should return in 24 to 48 hours to evaluate the cavity and insert new packing as needed.

B. If symptoms worsen prior to follow-up, recommend that the client proceed to the ED for further assessment.

CONSULTATION/REFERRAL

A. Consult with a physician for all abscesses requiring I&D with packing.

B. Consult a physician for all abscesses that are not responding to antibiotic treatment for further evaluation.

C. See the "Cellulitis" section for other treatment options.

CELLULITIS

DEFINITION

A. Cellulitis is an acute inflammation and infection of the skin and subcutaneous tissue.

INCIDENCE

A. Cellulitis is not a reportable infection; therefore, the exact incidence is not known. However, some studies show that the incidence of cellulitis is approximately 200 individuals per 100,000 client years.

PATHOGENESIS

A. Cellulitis occurs when bacteria attack the skin tissue, which involves the dermis and subcutaneous tissue. The most common organisms causing this bacterial infection include beta-hemolytic streptococci (groups A, B, C, G, and F) and *Staphylococcus aureus*.

B. The most common organisms causing an abscess include *S. aureus* (either methicillin-susceptible *S. aureus* or MRSA [methicillin-resistant *S. aureus*]), which occur in up to 75% of cases, and group A streptococci.

C. Bacteria may invade healthy tissue but commonly occurs in damaged tissue, where there is a break in the skin, allowing invasion of the tissue that can lead to bacterial infection.

PREDISPOSING FACTORS

A. Age (adults older than 45 years, geriatrics).
B. Immunocompromised immune system.
C. Chronic medical conditions (diabetes).
D. History of intravenous (IV) drug abuse.
E. Alcoholism.
F. Peripheral vascular disease.
G. History of cellulitis.

COMMON COMPLAINTS

A. Erythema, pain, swelling, and warmth are the classic symptoms.
B. Fever, malaise, and chills.
C. Lymphadenopathy of lymph nodes adjacent to site of infection.
D. Trauma to skin tissue.

OTHER SIGNS AND SYMPTOMS

A. Drainage/discharge at wound site.
B. Erythemic streaks that progress away from the infection site.
C. Systemic signs of infection (fever, tachycardia, tachypnea, hypotension, change in mental status).

POTENTIAL COMPLICATIONS

A. Sepsis.
B. Necrosis of tissue.
C. Osteomyelitis.

SUBJECTIVE DATA

A. Ask the client if there was any activity/injury that preceded the episode.
B. Inquire regarding the onset, location, and duration of pain and symptoms. Has the area increased in size or spread to other areas?
C. Ask the client to describe pain, for example, crushing, stabbing, or burning sensation.
D. Have the client rate pain on a scale of 0 to 10, with 0 being no pain.
E. Inquire regarding any previous trauma to affected area. Have any close contacts had similar symptoms?

PHYSICAL EXAMINATION

A. Check vital signs: Check temperature, pulse, respirations, and blood pressure.
B. Inspect:
  1. Inspect general appearance of skin. Note location of involved skin and note erythemic streaking if present.
  2. Measure size (mm/cm) of skin involvement. Include pictures if applicable. Outline the erythema with a marker for ease of assessment between medical professionals as well as client if being discharged for home monitoring.
  3. Note presence of erythema, edema, and color of skin tissue involved (e.g., pink, erythemic, necrotic, pale).
C. Palpate:
  1. Palpate site for warmth, tenderness, firmness, and fluctuance.
  2. Palpate adjacent lymph nodes to assess for lymphadenopathy.
D. Auscultate:
  1. Auscultate the heart.
  2. Auscultate the lungs.

DIAGNOSTIC TESTS

A. Laboratory (blood cultures, Gram stain, culture specimen from the abscess, complete blood count [CBC] with differential, C-reactive protein [CRP] creatinine, bicarbonate, creatine phosphokinase):
  1. Blood cultures: Recommended for clients presenting with signs of systemic toxicity, extensive skin involvement, a history of comorbid conditions, history of recurrent cellulitis, and/or trauma (e.g., dog/cat bite, puncture wounds).
  2. Culture and sensitivities should be performed on clients with signs of systemic infection, severe skin impairment, history of chronic conditions, recurrent infections, and history of animal or water injury.
B. Imaging studies:
  1. Ultrasound to assess for abscess.
  2. X-ray may be performed if suspicious for osteomyelitis.
  3. CT or MRI may be considered when assessing for necrotizing fasciitis.

DIFFERENTIAL DIAGNOSES

A. Cellulitis.
B. Abscess.
C. Dermatitis.
D. Erysipelas. (This is a superficial skin infection that commonly occurs on the face, ears, and/or lower legs. It commonly presents with erythema of the skin with a distinct demarcated border commonly caused by group A *Streptococcus* bacteria. Erysipelas is more commonly seen in older adults.)

PLAN

A. General interventions:
  1. If there are signs of systemic infection, hospitalization is required.
  2. Educate the client regarding care of the site.
  3. Instruct client on reporting worsening symptoms as soon as possible to prevent sepsis.
B. Client teaching:
  1. Instruct client to keep site clean and dry, recommending elevation of extremity if applicable.

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2. Recommend over-the-counter (OTC) pain medications such as acetaminophen (Tylenol) or ibuprofen (Advil, Motrin) as needed for pain.

3. Advise client to notify the primary provider for the following symptoms:

a. Fever of more than 100.0°F.
b. Chills.
c. Increase in size of redness or change in size/consistency of site (i.e., abscess formation).
d. Erythemic streaks.
e. Lymphadenopathy.
f. Increase in pain.

4. Instruct the client on use of antibiotics and stress the importance of finishing the full course of prescribed antibiotics.

5. Once the infection has resolved, stress the importance of notifying the primary provider for reoccurring symptoms.

6. Recurrent cellulitis should be evaluated for the cause. Chronic underlying conditions, such as edema, should be treated to prevent future episodes. Edema may be treated with compression stocking therapy and/or use of diuretics.

C. Pharmaceutical therapy:

1. Uncomplicated cellulitis: Oral treatments include cephalexin 500 mg tablet Q6H; or dicloxacillin 500 mg tablet Q6H; or clindamycin 300 to 450 mg tablet every 6 to 8 hours. Oral medications are prescribed for 5 to 10 days.

a. Empiric treatment for MRSA should be considered for the following clients:

i. Clients who do not respond to initial treatment.
ii. Clients with signs of sepsis.
iii. Clients who have recurrent cellulitis.
iv. Clients who have a history of MRSA.

2. Cellulitis MRSA: Oral treatments include doxycycline 100 mg tablet BID or clindamycin 300 to 450 mg tablet TID or trimethoprim-sulfamethoxazole (TMP-SMX) double strength (DS) tablet BID or minocycline 200 mg tablet once, then 100 mg tablet BID or linezolid 600 mg tablet BID or tedizolid 200 mg tablet daily. Oral medications are prescribed for 5 to 10 days.

a. Dosing should be individualized according to the underlying conditions. Obese clients may have a higher failure rate if not prescribed adequate dosing.
b. Clients with purulent drainage should be treated for MRSA pending culture results.
c. Clients with renal impairment will require dosage adjustments.

3. Complicated cellulitis: IV antibiotics should be prescribed to clients with complicated cellulitis and/or erysipelas with signs of sepsis (fever/chills). Antibiotic treatments may include cefazolin, oxacillin, nafcillin, vancomycin, daptomycin, and ceftaroline.

FOLLOW-UP

A. Client needs to follow up in 24 to 48 hours for reassessment.

B. If worsening symptoms are present, recommend that the client proceed to the ED for evaluation and treatment.

CONSULTATION/REFERRAL

A. If necrotizing fasciitis is suspected, referral for a surgical consult is imperative.

B. Inpatient hospitalization is recommended for the following:

1. Hypotension.
2. Elevated creatinine level.
3. Creatine elevated two to three times over normal values.
4. CRP more than 13 mg/L.
5. Low serum bicarbonate level.
6. Elevation in CBC with left shift.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Suppressive antibiotic therapy is recommended for clients with predisposing risk factors who have a history of recurrent cellulitis.

2. Geriatric clients are at increased risk of developing thrombophlebitis following an acute episode of cellulitis.

3. Geriatrics often develop stasis dermatitis associated with diminished circulation causing skin breakdown, which could result in cellulitis. A Doppler ultrasound will rule out blood clots. To differentiate between stasis and allergic dermatitis is to understand that cellulitis generally tends to be unilateral while allergic/contact dermatitis is usually bilateral. However, cultures and imaging will help make the definitive diagnosis.

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CONTACT DERMATITIS

DEFINITION

A. Contact dermatitis is a cutaneous response to direct exposure of the skin to irritants (irritant contact dermatitis) or allergens (allergic contact dermatitis):

1. Irritant contact dermatitis is a nonimmunologic response of the epidermis.

2. Allergic contact dermatitis is an immunologic response after one or more exposures to a particular agent.

INCIDENCE

A. Occurs in all ages. People who work with chemicals daily and wash their hands numerous times a day have a higher incidence of irritant dermatitis. Irritant contact dermatitis is seen in older adults due to dry skin.

PATHOGENESIS

A. Irritant contact dermatitis is caused by an alteration in the outer layer of the dermis due to exposure to chemicals, lotions, cold, dry air, soaps, detergents, or organic solvents.

B. Allergic contact dermatitis is caused by an alteration in the epidermis when, after exposure to an allergen, the immune system responds by producing inflammation of the cutaneous tissue. Common allergens include poison ivy, poison oak, sumac, nickel jewelry, hair dye, rubber and leather chemicals (latex gloves), cleaning supplies, harsh soaps, detergents, and topical medicines.

PREDISPOSING FACTORS

A. Occupation (e.g., hairdressers, nurses, housecleaners, food handlers, mechanical industry workers).

B. Jewelry.

C. Activities in yard or woods.

COMMON COMPLAINTS

A. Irritation of the skin, ranging from redness to pruritic inflammation, with possible progression of blisters:

1. Poison oak, ivy, and sumac induce classic presentation: lesions (vesicles) and papules on an erythemic base presenting in a linear fashion with sharp margins.

2. Diffuse pattern with erythema may be seen when oleoresin is contracted from pets or smoke from burning fire.

B. Exposure to some type of irritant known to the client. Round or annular lesions may have an internal cause, such as a drug reaction.

OTHER SIGNS AND SYMPTOMS

A. Chronic:

1. Erythema with thickening.

2. Scaling.

3. Fissures.

4. Inflammation; with chronic dermatitis, lichenification may occur with scales and fissures.

5. Lichenification may occur with scales and fissures.

B. Candidiasis diaper rash:

1. Bright red rash with satellite lesions at margins.

2. Inflammation and excoriations present.

3. Creases may be involved.

SUBJECTIVE DATA

A. Ask the client when irritation began and how it has progressed.

B. Elicit history of exposure to allergens.

C. Question the client regarding activity and skin contact with irritants prior to outbreak (e.g., cleaning agents, walking in woods, hobbies, change in soap/laundry detergent, shaving cream, lotions).

D. List occupation and family history of allergens.

E. Review medication list, including prescription, over-the-counter (OTC), and herbal treatments to evaluate for an interaction.

F. List medications and OTC remedies used to relieve symptoms and results.

PHYSICAL EXAMINATION

A. Check temperature (if indicated).

B. Inspect:

1. Inspect skin, noting types and locations of lesions. **Note the pattern of inflammation. The shape of irritation may mimic the shape of the irritant, such as the skin under a ring or watch, for example.**

2. Determine progression of lesions.

3. Differentiate between primary and secondary lesions.

DIAGNOSTIC TESTS

A. Consider none if source is known.

B. Wet mount (potassium hydroxide [KOH] saline) to rule out fungal infection if candidiasis is suspected.

C. Culture/sensitivity of pustules.

D. Patch test to rule out allergic contact dermatitis.

DIFFERENTIAL DIAGNOSES

A. Irritant contact dermatitis.

B. Allergic contact dermatitis.

C. Candidiasis.

D. Tinea pedis, corporis, and cruris.

E. Drug reactions.

F. Pityriasis rosea.

G. Scabies.

PLAN

A. General interventions:

1. Irritant contact dermatitis: Remove the irritating agent.

a. Topical soaks with saline or Burow solution (1:40 dilution) for weeping areas.

b. Recommend lukewarm baths (not hot) with oatmeal (Aveeno), as needed.

c. For dry erythematous skin, use Eucerin or Aquaphor ointments to rehydrate skin.

d. Remind the client to avoid scratching skin and to keep nails short.

e. Suggest use of mild soaps and cleansers.

2. Allergic contact dermatitis:

a. Instruct the client to avoid contact with the agent.

b. Have the client wash with cool water immediately after exposure.

c. Recommend lukewarm baths with oatmeal (Aveeno) three to four times per day.

d. Tell the client to apply calamine lotion after baths.

B. Client teaching: *See Client Teaching Guide for this chapter, "Dermatitis."*

C. Pharmaceutical therapy:

1. Irritant contact dermatitis:

a. Hydrocortisone 2.5% ointment three to four times per day for 2 weeks.

2. Allergic contact dermatitis:

a. Low-potency topical steroids: hydrocortisone 2.5% ointment three to four times per day for 1 to 2 weeks after blistering stage; triamcinolone acetonide 0.025% (Kenalog) ointment/cream BID.

b. Medium-potency topical steroids: triamcinolone acetonide 0.1% (Kenalog) cream BID.

c. High-potent topical steroids: fluocinonide 0.05% (Lidex) ointment three to four times per day; not to be used on face or skin folds.

d. Hydroxyzine 25 to 50 mg QID and, diphenhydramine (Benadryl) 25 to 50 mg QID.

e. If rash is severe (face, eyes, genitalia, mucous membranes), consider prednisone 60 to 80 mg/d to start and taper over 10 to 14 days.

f. Triamcinolone acetonide (Kenalog) 40 to 60 mg by intramuscular (IM) injection.

3. Secondary bacterial infections:

a. Erythromycin 250 mg QID or amoxicillin-clavulanate acid (Augmentin) 875 mg BID for 10 days.

4. Secondary candidiasis infections:

a. Miconazole nitrate 2% cream, miconazole powder, or nystatin cream.

b. Clotrimazole (Lotrimin) or ketoconazole (Nizoral) cream three to four times per day for 10 days.

c. If inflammation is present along with yeast, use Mycolog II.

d. If secondary bacterial infection is present, use mupirocin (Bactroban) ointment TID for 7 to 10 days.

FOLLOW-UP

A. None required if case is mild.

B. See client again in 2 to 3 days for severe cases, or phone to assess progress.

CONSULTATION/REFERRAL

A. Consult with a physician when steroid treatment is necessary or if worsening symptoms develop despite adequate therapy.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
  1. If medications are necessary during pregnancy, consider the gestational age of fetus and category of medication.

C. Geriatrics:
  1. Clients may only exhibit scaling as the prominent irritation rather than erythema and inflammation. Topical medications (neomycin, vitamin E, lanolin) and acrylate adhesives are common causes of contact dermatitis.
  2. Avoid first-generation anticholinergics due to risk of confusion, dry mouth, constipation, and other anticholinergic effects or toxicity.
  3. Pruritus that persists ≥6 weeks in older adult population 65 years old and older is considered a chronic dermatitis condition. Studies indicate that the aging epidermal barrier, nervous system, and immune system predispose geriatrics to a sensation of "itchiness." Topical medications are considered first-line treatment for geriatrics; however, systemic treatments might be considered after evaluation of the client's comorbid diseases.

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ERYTHEMA MULTIFORME

DEFINITION

A. This dermal and epidermal inflammatory process is characterized by symmetric eruption of erythematous, iris-shaped papules ("target" lesions) and vesiculobullous lesions.

INCIDENCE

A. Erythema multiforme accounts for up to 1% of dermatology outpatient visits.
B. Adults older than 50 are rarely affected.
C. It may occur in seasonal epidemics.
D. Approximately 90% of cases of erythema multiforme minor follow a recent outbreak of herpes simplex virus (HSV)-1 or *Mycoplasma* infection.

PATHOGENESIS

A. The disorder is thought to be an immunologic reaction in the skin, possibly triggered by circulating immune complexes.

PREDISPOSING FACTORS

A. Infections: recurrent HSV, mycoplasmal infections, and adenoviral infections.
B. Drugs: sulfonamides, phenytoin, barbiturates, phenylbutazone, penicillin, and nonsteroidal anti-inflammatory drugs (NSAIDs).
C. Idiopathic: greater than 50%; consider occult malignancy.

COMMON COMPLAINTS

A. Rash with intense pruritus.
B. Nonspecific upper respiratory infection followed by rash.
C. General malaise, body aches, joint pain.
D. Fever.

OTHER SIGNS AND SYMPTOMS

A. Primary: macules, papules, plaques.
B. Secondary: erythema, dull red, target-like lesions blanch to pressure; distribution is symmetric, primarily on flexor surfaces. **Classic target lesions develop abruptly and symmetrically and are heaviest peripherally; they often involve palms and soles.**
C. Swelling of hands and feet.
D. Painful oral lesions.
E. Eye discomfort (redness, itching, burning, pain, visual changes).

SUBJECTIVE DATA

A. Ask if the client has ever been diagnosed with erythema multiforme.
B. Determine whether the onset of symptoms was sudden or gradual.
C. Assess for any associated discharge (blood or pus).
D. Identify the location of the symptoms.
E. Complete a drug history. Has the client recently taken any antibiotics or other drugs? Question the client regarding use of any topical medications.
F. Determine the presence of any preceding systemic symptoms (fever, sore throat, anorexia, vaginal discharge).
G. Rule out any possible exposure to industrial or domestic toxins.
H. Question the client concerning any possible contact with venereal disease.
I. Ask the client about any close physical contact with others with skin disorders.
J. Elicit information concerning any possible exposure to HIV.
K. Rule out sources of chronic infection, neoplasia, or connective tissue disease.
L. Inquire if client has received a COVID-19 vaccine or booster recently.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.
B. Inspect:
  1. Inspect the skin for lesions.
  2. Inspect the mouth and mucous membranes for lesions.
C. Palpate:
  1. Palpate the abdomen for masses and tenderness.
D. Auscultate:
  1. Auscultate the heart, lungs, and abdomen.
F. Perform neurologic exam.

DIAGNOSTIC TESTS

A. Punch biopsy of skin.
B. Complete blood count (CBC).
C. Urinalysis.

DIFFERENTIAL DIAGNOSES

A. Erythema multiforme:
  1. Erythema multiforme minor: pruritus, swelling of hands and feet, painful oral lesions.
  2. Erythema multiforme major: Fever, arthralgias, myalgias, cough, oral erosions with severe pain.
B. Urticaria.
C. Viral exanthems.
D. Stevens–Johnson syndrome (SJS): **a severe, life-threatening, systemic reaction with fever, malaise, cough, sore throat, chest pain, vomiting, diarrhea, myalgia, arthralgia, and severe skin manifestations with painful bullous lesions on mucous membranes.** ▶

E. Pemphigus vulgaris.
F. Bullous pemphigoid.
G. Other bullous diseases.
H. Staphylococcal scalded skin syndrome.

PLAN

A. General interventions:
1. Identify and treat precipitating causes or triggers.
2. Burow solution or warm compress may be used for mild cases as needed.
3. Oral lesions may be treated with saline solution, warm saltwater, and/or Mary's mouthwash (Benadryl, lidocaine, and Kaopectate).
4. Discontinue any medications suspected of precipitating symptoms.
5. Provide adequate pain relief if skin or oral lesions are painful. Lesions remain fixed at least 7 days.
6. Maintain nutrition and fluid replacement for this hypercatabolic state.
7. Consider chronic viral suppression therapy for recurrent HSV infections.
B. Client teaching: *See Client Teaching Guide for this chapter, "Erythema Multiforme."*
C. Pharmaceutical therapy:
1. Antihistamines such as Benadryl or Claritin may be used for itching.
2. Acetaminophen may be used to reduce fever and general discomfort/pain.
3. Potent topical corticosteroids include betamethasone dipropionate 0.05% or clobetasol propionate 0.05% BID for up to 2 weeks. Avoid use on face and groin.
4. Open lesions should be treated like open burn wounds. Stop offending medications that may cause blistering of the wounds and treat with steroids.
5. Oral antibiotics may be needed to control secondary bacterial skin infection.
6. Systemic corticosteroids include prednisone 40 to 60 mg daily in divided doses, tapered over 2 to 3 weeks.
7. Hospitalization for severe cases. Intravenous (IV) immunoglobulins may be needed.

FOLLOW-UP

A. See the client in office in 1 to 2 days to evaluate initial treatment.

CONSULTATION/REFERRAL

A. If client has recurrent or chronic infection, refer them to a physician.
B. Immediate consultation and/or hospital admission is critical if SJS is suspected.
C. Clients with ocular involvement should be referred immediately to an ophthalmologist.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. Avoid first-generation anticholinergics due to risk of confusion, dry mouth, constipation, and other anticholinergic effects or toxicity.
2. Geriatrics taking alendronate (Fosamax) for osteoporosis therapy are at an increased risk of an adverse effect that presents as mucosa erythema multiforme. The recommendation for geriatrics taking this medication is to evaluate any hypersalivation history, swollen lips (macrocheilia), and mouth ulcers that have ill-defined borders and serofibrinous exudates. Treatment measures are local cleaning, cold compress, and clinical follow-up. Discontinue Fosamax and consider alternative treatment for osteoporosis.

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FOLLICULITIS

DEFINITION

A. Folliculitis is inflammation of the superficial or deep portion of the hair follicle, causing an infection. This can be caused by bacteria, fungi, viral, or parasitic processes.

INCIDENCE

A. A very common disorder, folliculitis occurs in all ages and is seen more frequently in males.
B. *Malassezia* folliculitis is commonly seen in clients with immunosuppression, diabetes, and antibiotic use.

PATHOGENESIS

A. Bacterial organisms (most commonly *Staphylococcus aureus*) invade the follicle wall and cause an infectious process.
B. Fungal organisms, *Malassezia* folliculitis, also known as *Pityrosporum*, invade the follicle wall and cause an infectious process.

PREDISPOSING FACTORS

A. Break in the skin tissue.
B. Use of razors on skin.
C. Poor hygiene.
D. Diabetes.
E. Long-term oral antibiotic therapy for acne.
F. Exposure to hot tubs or heated swimming pools.
G. Immunosuppression.

COMMON COMPLAINT

A. Outbreak of pustules on the face, scalp, or extremities that does not resolve despite proper hygiene and care.

OTHER SIGNS AND SYMPTOMS

A. Tenderness and itching at site.
B. Furuncle (abscess): a deep pustule, tender, firm or fluctuant, found in groin, axilla, waistline, buttocks.
C. Carbuncle: a group of follicles coalescing into one larger, painful, infected area; may see fever and chills.
D. Excoriated folliculitis: chronic thickened, excoriated papules or nodules.

SUBJECTIVE DATA

A. Elicit the initial outbreak of lesions and onset and progression of lesions.
B. Identify what makes the lesions better or worse.

C. Ask the client what medications, soaps, or lotions have been used on the lesions.
D. Complete a medical history. Ask if the client has had a similar outbreak before.
E. Describe systemic symptoms if they have occurred (e.g., fever chills).
F. Does the client have a beard, shave their face, or use a razor frequently?
G. Is there a recent history of use of a hot tub? (Commonly seen 1 to 4 days after use of hot tub, whirlpool, or swimming pool.)
H. Does the client wear tight pants/jeans or use oils that clog pores in the groin area?
I. Is the client currently being treated with antibiotics for acne? (Flare of Gram-negative folliculitis may be seen with chronic use of antibiotics.)

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.
B. Inspect:
1. Assess the skin for lesions and describe.
C. Palpate:
1. Palpate the lesions and associated lymph nodes.

DIAGNOSTIC TESTS

A. Culture and sensitivity to verify appropriate antibiotic coverage.
B. Gram stain.
C. Potassium hydroxide (KOH)/wet prep.
D. Fungal culture hair if fungi suspected (tinea of the scalp).
E. Skin biopsy for diagnosis of *Malassezia* folliculitis.

DIFFERENTIAL DIAGNOSES

A. Folliculitis.
B. Acne vulgaris.
C. Ingrown hair follicle.
D. Keratosis pilaris.
E. Contact dermatitis.
F. Papulopustular rosacea.
G. Hidradenitis suppurativa.

PLAN

A. General interventions:
1. Apply warm, moist compress to the site for comfort.
B. Client teaching: *See Client Teaching Guide for this chapter, "Folliculitis."*
1. If razors are used on the area, have the client use clean, sharp razors; throw old razors away and do not share razors. Avoid use of irritating creams or lotions on affected area.
2. Encourage proper hygiene, such as frequent washing of hands and skin with antibacterial soap.
3. Warm compress three to four times a day is encouraged at site for 15 to 20 minutes.
4. Bleach bath (1/2–1 cup of bleach to 20-L water) reduces spread of staph infection.
C. Pharmaceutical therapy:
1. Mild cases: Apply mupirocin (Bactroban) ointment to the affected area TID until resolved.
2. *S. aureus:*
a. Dicloxacillin (Dynapen) 250 to 500 mg PO QID for 7 to 10 days.
b. Cephalexin (Keflex) 250 to 500 mg PO BID for 7 to 10 days.
3. Methicillin-resistant *S.aureus* (MRSA):
a. Trimethoprim-sulfamethoxazole one to two double strength (DS) tablets BID for 7 to 10 days.
b. Doxycycline 100 mg BID for 7 to 10 days.
4. *Pseudomonas aeruginosa*:
a. Ciprofloxacin (Cipro) 500 to 750 mg PO BID for 10 days.
b. Ofloxacin 400 mg PO BID for 10 days.
5. Antistaphylococcal antibiotics:
a. Cephalexin 250 to 500 mg QID.
b. Clindamycin 150 to 300 mg QID.
c. Dicloxacillin 125 to 500 mg QID.
d. Erythromycin 250 to 500 mg QID.
6. Bacteria caused by organisms other than *Staphylococcus* may be treated for an extended period of time, 4 to 8 weeks. These areas may include axilla, chest, back, beard, and groin.
7. Severe cases may be treated with oral antibiotics with topical permethrin Q12H every other night for a 6-week period or itraconazole 400 mg daily, isotretinoin 0.5 mg/kg/d for up to 4 to 5 months with UVB light therapy. Consider dermatology referral for severe cases.
8. Treatment for *Malassezia* folliculitis is as follows:
a. Antifungal treatment: Oral antifungal medications (itraconazole, fluconazole, or ketoconazole) should be prescribed for at least 4 weeks for treatment.

FOLLOW-UP

A. If not resolved in 2 weeks, further evaluation is needed.
B. Severe cases, in which carbuncles are not improved with treatment of antibiotic therapy, may warrant incision and drainage.
C. Continue to follow every 2 weeks until resolved.
D. Test for diabetes mellitus if severe cases occur.

CONSULTATION/REFERRAL

A. Refer the client to a specialist for testing for immunodeficiency if severe cases occur or if resistance is seen.
B. Refer to a dermatologist.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. Skin tissue in older adults is very thin and fragile. Precautions should be taken to avoid further breakdown. Eradicating the infection quickly and healing skin tissue are imperative to prevent long-term complications.

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HERPES SIMPLEX VIRUS TYPE 1

DEFINITION

A. Herpes simplex virus (HSV)-1 infection of the cutaneous tissue manifests itself by vesicular lesions on the mucous membranes and skin. HSV-1 is most often associated with oral lesions (mouth, lips), while HSV-2 is associated with genital lesions. The virus appears in three stages:
1. Primary.
2. Latency.
3. Recurrent infections.

INCIDENCE

A. HSV-1 is seen in clients of all ages and in equal numbers of males and females.

B. There are approximately 500,000 new cases of herpes diagnosed annually in the United States.

PATHOGENESIS

A. Viral infection can be transmitted by direct contact from a vesicular lesion or fluid (saliva) containing the virus to the skin or mucosa of another person, with an incubation period of 2 to 14 days. The trigeminal ganglia are the host of the oral virus. The virus can be reactivated, whereupon it travels along the affected nerve route and produces recurrent lesions. Common sites of infection are the lips, face, buccal mucosa, and throat.

PREDISPOSING FACTORS

A. Immunocompromised clients.

B. Prior HSV infections.

C. Exposure to virus.

COMMON COMPLAINTS

A. Painful lips, gums, and oral mucosa.

OTHER SIGNS AND SYMPTOMS

A. Primary lesion: fever, blisters on lips, malaise, and tender gums.

B. Recurrent episodes: Fever blisters with prodrome of itching, burning, and tingling sensation at site before vesicles appear.

SUBJECTIVE DATA

A. Ask questions regarding location, onset, and duration of lesions.

B. Elicit description of prodromal symptoms.

C. Ask the client if systemic symptoms occur with vesicular outbreak.

D. Determine when the initial outbreak of lesions occurred (commonly seen in childhood).

E. Inquire whether the client has been exposed to anyone with similar lesions.

F. If the lesion(s) is recurrent, ask the client if stress, skin trauma, or sun exposure stimulates outbreak of fever blisters.

PHYSICAL EXAMINATION

A. Inspect:

1. Inspect the skin, noting the location, appearance, and stage of the vesicles.

B. Palpate:

1. Palpate the lymph nodes for lymphadenopathy.

DIAGNOSTIC TESTS

A. Viral cultures.

DIFFERENTIAL DIAGNOSES

A. HSV-1.

B. Impetigo (appears as amber-colored vesicular lesions with crusting).

C. Stomatitis (appears as erythemic or erosion lesions in the mouth and lips).

D. Herpes zoster (causes vesicles that run along a single dermatome).

E. Stevens–Johnson syndrome (SJS).

F. Herpangina: Vesicles can be noted on the soft palate, tonsillar area, and uvula area, usually caused by the coxsackievirus.

PLAN

A. General interventions:

1. Comfort measures. Ice may be used to reduce swelling as needed.

2. Vaseline or other lip ointments may be applied as needed and lip ointment with sun protection factor (SPF) 30 or greater when exposed to sunlight.

B. Client teaching:

1. Educate the client regarding the disease process of HSV-1.

2. Instruct client to wash hands frequently.

3. Suggest proper care of lips to prevent drying and to reduce pain.

4. Educate regarding transmission of virus to others.

5. Teach client to expect recurrences at variable times.

C. Pharmaceutical therapy:

1. Precautions should be used when administering medication to clients who are immunocompromised and who have a history of renal insufficiency:

a. Lidocaine 2% as needed for comfort.

b. Diphenhydramine (Benadryl) elixir to rinse mouth as needed.

c. Acetaminophen (Tylenol) as needed for pain.

d. Campho-Phenique application as needed.

e. Initial episode: acyclovir 400 mg PO TID for 7 to 10 days or until resolved.

f. Recurrent episodes: Begin one of the following when prodrome begins or within 2 days of onset of lesions to get maximum effect:

i. Acyclovir 400 mg PO TID for 5 days.

ii. Acyclovir 800 mg PO BID for 5 days.

g. Other alternative antivirals: Dosage depends on renal function:

i. Famciclovir (Famvir) 750 mg PO x 1 dose.

ii. Valacyclovir (Valtrex) 2 g PO x 1 dose.

h. Suppressive therapy is recommended for clients with frequent lesions that affect everyday life:

i. Acyclovir 400 mg PO BID.

ii. Valacyclovir 500 mg PO once daily.

FOLLOW-UP

A. None needed if resolved without complications.

CONSULTATION/REFERRAL

A. Refer the client to a physician if treatment is unsuccessful or further complications arise.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Educate regarding the diagnosis of HSV-1 and precautions to take to avoid transmission to the genital area. Limit the number of sexual contacts, use a latex condom during sexual activity, use water-based lubricants (avoid oil-based lubricants), and avoid spermicides to prevent transmission. Advise the client that if a lesion in the genital area presents at any time, they are to notify their healthcare provider for testing. ▶

2. Pregnant individuals with history of human papillomavirus after 36 weeks should start suppressive therapy to avoid transmission during delivery. C-section may be the best option for delivery if flare is present.

B. Geriatrics:

1. Avoid first-generation anticholinergics due to risk of confusion, dry mouth, constipation, and other anticholinergic effects or toxicity.

2. HSV-1 has been implicated as a major component in Alzheimer disease in those who carry type 4 allele of the apolipoprotein E gene (APOE). Studies indicate that the virus remains latent in the brain of older adults and can be triggered by stress, immunosuppression, and infection. The cumulative damaging effects could further progress the disease and cognitive decline. Practitioners must look for any signs of HSV-1 outbreaks in clients suffering from Alzheimer disease and quickly treat.

3. Research does not support obtaining viral culture unless it is a first time active lesion without drying or a healed lesion.

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HERPES ZOSTER (SHINGLES)

DEFINITION

A. Herpes zoster is a viral infection manifested by painful, vesicular lesions on the skin, limited to one side of the body, following one body dermatome.

INCIDENCE

A. Infection may occur at any age; however, it is more common in older adults. It is estimated by the Centers for Disease Control and Prevention (CDC) that approximately 30% of persons in the United States will experience herpes zoster in their lifetime.

PATHOGENESIS

A. After the primary episode of chickenpox (varicella zoster), the virus remains dormant in the body. Herpes zoster occurs when the varicella virus has been stimulated and reactivated in the dorsal root ganglia, producing the clinical manifestations of herpes zoster. Infection usually lasts 14 to 21 days but may be longer in older adults or debilitated clients.

PREDISPOSING FACTORS

A. Adulthood.
B. Immunocompromised clients.
C. Spinal cord trauma or injury.
D. Autoimmune disease.

COMMON COMPLAINTS

A. Prodrome: itching, burning, tingling, or painful sensation at lesion sites.
B. Active: malaise, fever, headache, pruritic rash on the skin.

OTHER SIGNS AND SYMPTOMS

A. Lesions: clusters of vesicles on an erythemic base that burst and produce crusted lesions; commonly seen on the chest and back area; distribution that typically appears along a single dermatome.
B. Motor weakness (may be seen in approximately 5% of clients).

SUBJECTIVE DATA

A. Determine onset, location, and progression of rash.
B. Ask the client about prodromal symptoms: burning, itching, tingling, or painful sensation at site prior to lesions breaking out.
C. Evaluate client status regarding immunosuppressive agents, diseases, and others.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.
B. Inspect:

1. Observe skin for lesions, noting characteristics and distribution.

2. Inspect eyes, ears, nose, and throat.

C. Auscultate:

1. Auscultate the heart and lungs.

DIAGNOSTIC TESTS

A. Usually none.
B. Culture vesicular lesions.
C. Consider Tzanck smear.
D. Consider and test for HIV in young clients with herpes zoster.

DIFFERENTIAL DIAGNOSES

A. Herpes zoster.
B. Varicella.
C. Poison ivy.
D. Herpes simplex virus (HSV).
E. Contact dermatitis.
F. Coxsackievirus.
G. Postherpetic neuralgia.

PLAN

A. General interventions:

1. Comfort measures. Instruct the client to apply wet dressings (Burow solution) on site for 30 to 60 minutes at least four times a day. Calamine lotions may be used as needed; oatmeal (Aveeno) bath for comfort; acetaminophen (Tylenol) as needed for malaise, temperature, and comfort.

B. Client teaching: *See Client Teaching Guide for this chapter, "Herpes Zoster (Shingles)."*

1. Educate the client regarding the rash, which usually lasts approximately 2 to 3 weeks.

2. Instruct the client to monitor for signs/symptoms of postherpetic neuralgia.

3. Instruct the client to call if symptoms worsen or do not improve, or if signs of bacterial infection occur.

4. Emphasize to the client that the virus is easily transmitted to vulnerable persons who have never had the chickenpox.

C. Pharmaceutical therapy:

1. Antiviral medications should be initiated within 24 to 48 hours after outbreak:

a. Acyclovir (Zovirax) 800 mg five times daily for 7 days.

b. Famciclovir (Famvir) 500 mg PO TID for 7 days.
c. Valacyclovir (Valtrex) 1,000 mg PO TID for 7 days.

2. Acetaminophen (Tylenol) or ibuprofen as needed for pain or discomfort.

3. Narcotics may be used for severe pain as needed.

4. Postherpetic neuralgia may be treated with the following:

a. Narcotics or other pain-relieving medications.
b. Long-term medications may be needed for control of pain.
i. Gabapentin 300 to 600 mg TID, titrate up to achieve optimal dose for relief.
ii. Amitriptyline 10 mg every bedtime or other low-dose tricyclic antidepressants; may titrate up to 50 mg every bedtime if needed for relief.

5. If secondary bacterial infection of the skin occurs, apply silver sulfadiazine (Silvadene) topically to site until resolved.

6. Use of steroids is controversial. Corticosteroids may be used with caution. May increase risk of dissemination.

FOLLOW-UP

A. As needed for complications.

B. Monitor the client for complications: postherpetic neuralgia, Guillain–Barré syndrome, motor weakness, secondary infection, meningoencephalitis, ophthalmic and facial palsy, corneal ulceration, and so forth.

CONSULTATION/REFERRAL

A. Ramsay Hunt syndrome occurs when a shingles outbreak affects the facial nerve near one of the ears. This can cause facial paralysis and hearing loss in the affected ear. Consult a physician.

B. Hutchinson sign refers to vesicles in the periorbital region. These clients require an ophthalmologist referral.

C. Consult with a physician if secondary infection occurs or if secondary complications arise.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Acyclovir and valacyclovir can be used and their safety and efficacy show no issue.

2. Advise caution on the use of the antiviral medication famciclovir during pregnancy.

B. Geriatrics:

1. Postherpetic neuralgia occurs in approximately 15% of clients. It is commonly seen in older adults.

2. The CDC recommends the shingles vaccine for those 50 years and older, irrespective of whether they have had the chickenpox or shingles infection in the past. There are two types available; the CDC recommends the recombinant vaccine (Shingrix) over the live vaccine unless contraindicated:

a. Guidelines for Shingrix: a recombinant zoster vaccine:
i. Administration dosing:
1) Two doses administered intramuscularly (IM): first dose given on day 1 and the second dose administered 2 to 6 months after the first.
ii. Indications:
1) Immunocompetent clients older than 50 years.
2) Clients who previously received the varicella vaccine and the (live) zoster vaccine. The CDC recommends waiting 5 years to administer the Shingrix vaccine if Zostavax was previously administered.
3) Clients with comorbid conditions.
iii. Contraindications:
1) Clients with known allergy to components of the vaccine.
b. Guidelines for Zostavax: a zoster live vaccine guidelines:
i. Administration dosing:
1) One-time subcutaneous injection.
ii. Indications:
1) Older than 60 years.
iv. Contraindications:
1) Clients with anaphylactic reactions to gelatin or neomycin.
2) Immunocompromised clients.
3) Planned immunosuppression.
4) Clients receiving low-dose immunosuppressive therapy.
5) Household contacts of immunocompromised hosts.

3. Geriatric red flags:

a. Avoid first-generation anticholinergics due to risk of confusion, dry mouth, constipation, and other anticholinergic effects or toxicity.
b. Avoid use of nonsteroidal anti-inflammatory drugs (NSAIDs) in clients who have chronic kidney disease stage IV or less (creatinine clearance <30 mL/min).

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LICE (PEDICULOSIS)

DEFINITION

A. Pediculosis (lice) is an infestation of the louse on human beings in one of three areas:

1. Head (pediculosis capitis).
2. Pubic area (pthirus pubis).
3. Body (pediculosis corporis).

INCIDENCE

A. Pediculosis capitis is most common in children. It is estimated that head lice infestations occur in school systems anywhere from 10% to 40% of the time.

B. Pthirus pubis infestation is more common in adults.

C. Lice affect all demographics: all social, racial, and economic groups.

PATHOGENESIS

A. Head and body lice are transmitted by direct contact from person to person; this is the primary mode of transmission. The sharing of hats, combs, brushes, and so forth for transmission is controversial. The parasite hatches from an egg, or nit. Once hatched, the lice live on humans by sucking blood through the skin. The average female adult louse lives about ▶

1 month and lays seven to 10 eggs per day. The nits appear as small white eggs on the hair shaft. Nits are very difficult to remove and survive up to 55 hours after removal from the host. Body lice lay nits in the seams of clothing.

B. Pubic lice are found at the base of the hair shaft, where they lay nits. Pubic lice are transmitted through sexual contact.

PREDISPOSING FACTORS

A. Head and body lice: exposure to crowded public areas, such as schools; inability to clean and launder clothing, bed linens, and so forth.

B. Pubic lice: sexual contact with infected people.

C. Poor hygiene.

COMMON COMPLAINTS

A. Head lice: severe itching and scratching of the head, neck area, and commonly behind the ears.

B. Body lice: severe itching on the body, which may lead to secondary infections of the skin.

C. Pubic lice: severe itching of genital area.

OTHER SIGNS AND SYMPTOMS

A. Excoriated skin from intense scratching.

B. Visible lice or nits in hair, body, or clothing.

C. Papules with an erythemic base, which may develop on the genital area, axilla, chest, beard, or eyelashes.

SUBJECTIVE DATA

A. Inquire as to exposure to anyone known to have lice.

B. Identify whether the client attends a crowded environment, such as school, day care, and so forth.

C. Ask if lice and nits have been seen by the client.

D. Determine onset, duration, and course of symptoms. Ask: When were lice or nits first discovered?

E. Assess whether the client has been symptomatic (itching, scratching).

F. Inquire about social habits of cleaning, laundry, and so forth.

PHYSICAL EXAMINATION

A. Check temperature to rule out any secondary infection.

B. Inspect:

1. Inspect hair, body, pubic area, and clothing seams for nits or lice.

2. Note excoriation of skin.

3. Examine eyelashes of children.

4. Examine skin for secondary bacterial infection.

DIAGNOSTIC TESTS

A. None.

B. Culture the excoriated area if secondary bacterial infection is suspected.

DIFFERENTIAL DIAGNOSES

A. Lice.

B. Seborrheic dermatitis.

C. Scabies.

PLAN

A. General interventions:

1. Treat immediately with appropriate pediculicides (see the "Pharmaceutical therapy" section).

2. After treatment, it is imperative to remove each nit and louse; use fine-tooth comb for nit removal.

3. Evaluate the entire family for lice.

4. Treat secondary bacterial infection as needed.

B. Client teaching: *See Client Teaching Guide for this chapter, "Lice (Pediculosis)."*

1. Specific instructions need to be given to clients on how to get rid of lice and nits.

2. Reinforce good hygiene; teach not to share combs, brushes, hats, and hair accessories.

C. Pharmaceutical therapy:

1. Malathion lotion 0.5% (Ovide); pediculicidal and partially ovicidal: Leave on hair for 8 to 12 hours, then wash with a nonmedicated shampoo. A single application may be effective; repeat in 7 to 9 days if live lice are seen. Recommended for ages 6 and up.

2. Permethrin lotion 1% (Nix); pediculicidal only: Available over-the-counter (OTC). Leave on hair for 10 minutes, rinse and repeat on day 9. Recommended for ages 2 months and up.

3. Synergized pyrethrins (Rid 0.3%): Available OTC. Leave on hair for 10 minutes, rinse and repeat on day 9. Recommended for ages 2 years and up.

4. Do not use a shampoo/conditioner or conditioner before using head lice treatments. Do not wash hair for 1 to 2 days after using lice treatment regimen.

5. Pthirus pubis: permethrin (Nix) or pyrethrins; apply to the pubic area as directed.

6. Lindane (Kwell) toxicity may occur from ingestion or overuse and is exhibited by headaches, dizziness, and convulsions.

7. Eyelash manifestation: After removing nits, apply petroleum jelly to lashes three to four times a day for 8 to 10 days. **Eyelashes should never be treated with pediculicides.**

FOLLOW-UP

A. None recommended.

B. Some institutions require follow-up to evaluate whether infestation has resolved.

CONSULTATION/REFERRAL

A. If lice are a repeated problem, contact social services or the health department to have a visiting nurse or aide visit the home to evaluate home conditions and to teach the family how to prevent infestations.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Lindane (Kwell) is contraindicated during pregnancy. Permethrin topical is category B and is the only recommended for treatment of lice during pregnancy.

B. Geriatrics:

1. For adult/older adult clients with a diagnosis of lice, consider self-neglect, unsuitable living conditions, cognitive/psychiatric impairments, and/or abuse/neglect of older adults. Consider a home visit for evaluation of the client.

LICHEN PLANUS

DEFINITION

A. Lichen planus is a relatively uncommon condition of acute or chronic inflammatory dermatosis. It affects skin and mucous membranes with characteristic flat-topped, shiny, violaceous (purplish color), pruritic papules with lacy lines on the skin and milky-white papules in the mouth.

INCIDENCE

A. Lichen planus accounts for 0.1% to 1.2% of office visits to dermatologists.
B. It exhibits no racial preference.

PATHOGENESIS

A. Etiology is unknown, although it is possibly a cell-mediated immune response. Most cases remit within 7 years. Lesions may heal with significant postinflammatory hyperpigmentation.

PREDISPOSING FACTORS

A. Severe emotional stress.
B. Drugs may induce lichenoid plaques.

COMMON COMPLAINTS

A. Rash with or without pruritus.
B. Primary lesions: small, flat-topped papules or plaques that are polygonal, lightly scaly, and violaceous.
C. Secondary lesions: erythema, scales, and erosions.

OTHER SIGNS AND SYMPTOMS

A. Distribution: volar aspect of wrists, ankles, mouth, genitalia, and lumbar region.
B. Wickham striae (white, lacelike pattern on surface).
C. Scalp: atrophic skin with alopecia.
D. Nails: destruction of nail fold and bed, especially in large toe.
E. Males: lesions of glans penis.
F. Females: erosive lesions of labia and vulva.

SUBJECTIVE DATA

A. Determine whether the onset was sudden or gradual.
B. Ask the client to describe if the skin is itchy or painful.
C. Assess lesions for any associated discharge (blood or pus).
D. Identify the location(s) of the problem.
E. Complete a drug history. Ask the client if they have recently taken any antibiotics or other drugs. Ask if they have used any topical medications, lotions, or other creams.
F. Determine the presence of any preceding systemic symptoms (fever, sore throat, anorexia, or vaginal discharge).
G. Rule out insect bites.
H. Identify any possible exposure to industrial toxins, domestic toxins, or color-film-developing chemicals.
I. Ask if the client has had any possible sexual contacts with persons with HIV or sexually transmitted infections (STIs).
J. Ask if the client has had close physical contact with others with skin disorders.

PHYSICAL EXAMINATION

A. Inspect:
  1. Inspect skin and note lesion distribution.
  2. Inspect mucous membranes: buccal mucosa, tongue, and lips.
  3. Examine hair and nails.
  4. Observe genitalia.

DIAGNOSTIC TESTS

A. Dermoscopy to accentuate Wickham striae.
B. If necessary to confirm diagnosis, deep shave or punch biopsy of developed lesions.
C. HIV or STI testing if indicated.
D. Hepatitis testing should be completed to assess for hepatitis C as lichen planus has been shown to have a correlation.

DIFFERENTIAL DIAGNOSES

A. Lichen planus.
B. Lichenoid drug eruptions.
C. Leukoplakia.
D. Chronic graft versus host disease.
E. Candidiasis (thrush).
F. Lupus erythematosus.
G. Contact dermatitis.
H. Bite trauma.
I. Secondary syphilis.

PLAN

A. General interventions:
  1. Discontinue any suspected drug agent.
B. Client teaching: *See Client Teaching Guide for this chapter, "Lichen Planus."*
  1. Instruct the client that the disease may be chronic; most cases resolve spontaneously.
  2. Encourage the client to avoid severe emotional stress.
  3. Encourage the client to avoid scratching and prevent secondary infection.
  4. Reassure the client that lichen planus is not contagious.
C. Pharmaceutical therapy:
  1. Oral antihistamines to assist with pruritus: hydroxyzine hydrochloride 10 to 50 mg QID as needed for pruritus, or cetirizine HCl (Zyrtec) 10 mg daily.
  2. Medium- to high-potency topical corticosteroids:
    a. Mouth lesions: fluocinonide 0.05%, ointment or gel, two or three times daily.
    b. Body lesions: betamethasone dipropionate (Diprolene) 0.05%, triamcinolone (Kenalog), or other class 1 cream or ointment BID. **Caution clients about steroid atrophy**.
    c. Genital lesions: desonide cream 0.05% BID initially, although higher potency creams may be necessary. Topical corticosteroids should be used on genitalia in short bursts only.
    d. Hypertrophic lesions: Intralesional injections, such as injecting triamcinolone 2.5 to 10 mg/mL, 0.5 to 1 mL per 2-cm lesion, are helpful for pruritus relief. Use cautiously in dark-skinned clients due to risk of hypopigmentation. Limit the total dose to no more than 40 mg per treatment session. Injections may be repeated after 4 to 6 weeks.
  3. Oral prednisone is rarely used, but if necessary use with a short course only and taper.

FOLLOW-UP

A. See the client in 1 week for evaluation of treatment.

CONSULTATION/REFERRAL

A. Refer the client to a dermatologist if there is no response to initial treatment.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
  1. Use caution with medications prescribed.
B. Geriatrics:
  1. Red flags:
    a. Avoid first-generation anticholinergics due to risk of confusion, dry mouth, constipation, and other anticholinergic effects or toxicity.

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PITYRIASIS ROSEA

DEFINITION

A. Pityriasis rosea is an acute, self-limiting, benign skin eruption characterized by a preceding "herald patch" followed by widespread papulosquamous lesions.

INCIDENCE

A. Pityriasis rosea is relatively common, with more than 75% of cases in individuals from 10 to 35 years of age.
B. Incidence is slightly higher in females than in males.
C. Incidence is higher during spring and autumn.

PATHOGENESIS

A. Disease is idiopathic; some evidence exists to support viral origin or autoimmune disorder.

PREDISPOSING FACTOR

A. Recent acute infection.

COMMON COMPLAINTS

A. Rash: salmon-, pink-, or tawny-colored lesions generally concentrated in lower abdominal area, but may develop on arms, legs, and rarely on the face.
B. Mild pruritus.

OTHER SIGNS AND SYMPTOMS

A. Earliest lesions may be papular but may progress to 1- to 2-cm oval plaques.
B. Long axes of oval lesions run parallel to each other, hence the term "Christmas tree distribution."
C. Preceding herald patch (2–10 cm with central clearing) closely resembles ringworm; usually appears abruptly a few days to several weeks prior to the generalized eruptive phase.

SUBJECTIVE DATA

A. Elicit information about occurrence of initial, single, 2- to 10-cm round-to-oval lesion.
B. Question the client as to known contacts with similar symptoms. Small epidemics have been identified in fraternity houses and military bases.

PHYSICAL EXAMINATION

A. Check temperature to rule out any infection.
B. Inspect:
1. Examine all body surfaces with client unclothed.
2. Look for characteristic lesions and distribution.
3. Check the mucous surfaces, palms, and soles, which are usually spared by pityriasis rosea.

DIAGNOSTIC TESTS

A. Generally, none required; however, potassium hydroxide (KOH) wet preparation may be useful to distinguish herald patch from tinea corporis.
B. If necessary to confirm diagnosis, deep shave or punch biopsy of developed lesions.
C. Order serology to rule out syphilis, if applicable.
D. If unable to identify herald patch, a serologic test for syphilis should be ordered because syphilis may be clinically indistinguishable from pityriasis rosea.
E. White blood count normal; no specific lab markers for pityriasis rosea.

DIFFERENTIAL DIAGNOSES

A. Pityriasis rosea.
B. Nummular eczema.
C. Tinea corporis.
D. Tinea versicolor.
E. Viral exanthems.
F. Drug eruptions:
1. Captopril.
2. Bismuth.
3. Barbiturates.
4. Clonidine.
5. Metronidazole.

G. Secondary syphilis.
H. Lichen planus.
I. Psoriasis.

PLAN

A. General interventions:
1. Direct sunlight to point of minimal erythema hastens disappearance of lesions and reduces itching. UVB light in five consecutive daily exposures can reduce pruritus and shorten rash, particularly if administered within the first week of eruption.
2. Not proven to be contagious and relatively harmless, so isolation is not required.

B. Client teaching: *See Client Teaching Guide for this chapter, "Pityriasis Rosea."*
1. Advise the client that the disease is self-limiting and clears spontaneously in 2 to 3 months.

C. Pharmaceutical therapy:
1. Generally, none required, but for itching the following recommendations exist:
   a. Group V topical steroids and oral antihistamines as per usual dosing.
   b. Prednisone 20 mg BID for 1 to 2 weeks in rare cases of intense itching.

FOLLOW-UP

A. None required unless secondary bacterial infection develops. Reoccurrence is unusual after disease resolution.

CONSULTATION/REFERRAL

A. Consult or refer the client to a physician when disease persists beyond 3 months.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Disease has not been shown to affect fetus.

B. Geriatrics:
1. Disease rarely seen in geriatric clients. Strongly consider other differential diagnoses, particularly drug reactions.
2. Red flags:
a. Avoid first-generation anticholinergics due to risk of confusion, dry mouth, constipation, and other anticholinergic effects or toxicity.

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PRECANCEROUS OR CANCEROUS SKIN LESIONS

DEFINITION

A. Skin lesions are skin cell changes that occur in the body, which may be benign, precancerous, or cancerous.

INCIDENCE

A. Actinic keratosis is the most common precancerous skin disorder, occurring in approximately 58 million Americans, which is an estimate because it is not commonly reported and/or often occurs without diagnosis.

B. Basal cell carcinoma (BCC) is the most common form of skin cancer, with approximately 400,000 new cases per year in the United States. It is often seen in the sixth or seventh decade of life.

C. Squamous cell carcinoma (SCC) accounts for 20% of all skin cancers and occurs mostly in the middle-aged and older adult populations.

D. Malignant melanoma accounts for less than 5% of all skin cancers. The median age at diagnosis is 61 years. The rate of new melanoma skin cases increased to 27.6 per 100,000 in 2008.

PATHOGENESIS

A. Actinic keratoses: A precancerous skin lesion commonly caused by damage to the skin as a result of exposure to UV rays or tanning beds. If not treated, these skin lesions may turn into a form of skin cancer, such as SCC.

B. BCC: Skin lesions that result from abnormal cells of the basal layer of the epidermis that progress into the surrounding stroma and support basal cell growth. UV rays (sunlight) are the major contributor to BCC. BCC is a slow-growing tumor that rarely metastasizes.

C. SCC: Abnormal cells of the epidermis penetrate the basement membrane of the epidermis and move into the dermis, producing SCC. This often begins as actinic keratosis that undergoes malignant change.

D. Malignant melanoma: Abnormal cells proliferate from the melanocyte system. Initially, the cells grow superficially and laterally into the epidermis and papillary dermis. After a certain period of time, the cells begin moving into the reticular dermis and subcutaneous fat. Malignant tumors occur because of the inability of the damaged cells to protect themselves from long-term exposure to UV rays.

PREDISPOSING FACTORS

A. Advanced age (older than age 50).

B. Median age of 40 years for malignant melanoma.

C. Exposure to UV light (sun exposure).

D. Fair complexion (blonde or red hair, blue, green, or gray eyes).

E. Smokers (damaged lips).

F. Skin damaged by burns and/or chronic inflammation.

G. History of blistering sunburns before 18 years of age increases risk.

COMMON COMPLAINTS

A. New lesions found on the skin.

B. Ulcer/sore that does not heal.

OTHER SIGNS AND SYMPTOMS

A. Actinic keratosis (solar keratosis): scaly, crusted lesions commonly found on sun-exposed skin areas such as the face, ears, scalp, lips, and hands that are usually rough in texture and appearance.

B. BCC: Tumors arising from the basal cell layer of the epidermis; may be seen on face and neck; may appear as an open sore, pink growth, or nodule that is greater than 1 cm and which appears shiny, pearly in color with telangiectasia; center may cave in.

C. SCC: skin lesions seen in sun-exposed areas of the skin, or skin damaged by burns or chronic inflammation; lower lip lesions common; firm, irregular papules with scaly, bleeding, friable surface like sandpaper; grows rapidly.

D. Malignant melanoma: asymmetrical tumor of skin with irregular border, variation in color, greater than 6 mm in diameter; can metastasize to any organ.

E. Bowen disease (SCC in situ): chronic, nonhealing erythemic patch with sharp, irregular borders; occurs on skin and/or the mucocutaneous tissue; resembles eczema but does not respond to steroids.

SUBJECTIVE DATA

A. Have the client identify when lesion was first noted.

B. Ask the client to describe any changes in size, color, or shape of the lesion.

C. Determine whether the client has noted any new lesions.

D. Ascertain any family history of malignant melanoma.

E. Determine the client's history of skin exposure to the sun or any other UV rays.

F. Ask the client about smoking history. If the individual smokes, ask how many packs per day.

PHYSICAL EXAMINATION

A. Inspect:
1. Perform full-body exam of the skin for lesions.
2. Note the surface, size, shape, border, color, and diameter of lesion.
3. Examine scalp and ears for lesions.

DIAGNOSTIC TESTS

A. Biopsy suspicious lesions.

DIFFERENTIAL DIAGNOSES

A. Actinic keratosis.

B. BCC.

C. SCC.

D. Malignant melanoma.

E. Solar lentigo.

F. Seborrheic keratosis.

G. Common nevus.

H. Leukoplakia.

PLAN

A. General interventions:

1. Monitor progress/change of lesions detected.

2. Biopsy any suspicious lesions. Excise lesion with narrow margins, making sure to include all margins. If biopsy results of specimen are inadequate for accurate histologic diagnosis or staging, repeat biopsy. Include all clinical history information on the pathology report with the specimen when sending to pathology.

B. Client teaching: *See Client Teaching Guide for this chapter, "Skin Care Assessment."*

1. Educate clients regarding the importance of early identification of lesions and monthly assessment of skin. The U.S. Preventive Services Task Force (USPSTF) recommends counseling adolescents and young adults less than 24 years of age regarding reducing the amount of UV radiation exposure, especially between the hours of 10 a.m. and 3 p.m. In adults older than 24 years of age, the USPSTF did not find sufficient evidence to determine the effects of counseling these clients regarding the use of sun protection.

2. Instruct clients on monthly skin evaluation. Teach the "ABCD" method of exam for changes in lesions: **A**symmetry, **B**order, **C**olor, and **D**iameter. A body map may be used to mark skin changes and monitor progress. Mark the site of the lesion on the body map, including measurements and date found. A body map can be accessed at www.skincancer.org/skin-cancer -information/early-detection/body-map.

C. Pharmaceutical therapy:

1. None indicated.

FOLLOW-UP

A. When a diagnosis is made, follow up according to diagnosis. Differentiate between benign skin lesions and malignant melanoma. Follow-up/treatment depends on diagnosis (nonsurgical vs. surgical removal for diagnosis). Surgical removal is recommended for recurrent and suspicious lesions.

CONSULTATION/REFERRAL

A. Refer all clients to a dermatologist if skin cancer is suspected.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Skin cancer screening is important during pregnancy if new lesions develop.

2. Melanoma should be assessed without delay. The same anesthetics used for adult biopsy is also safe in pregnancy. Treatment with radiation to head and neck only and use of interferon for treatment may be safe in pregnancy.

B. Geriatrics:

1. Older adults are at higher risk of skin cancer due to their decreased immune system as they age, as well as their cumulative chronic disease risk factors. Educate these clients regarding their risk factors and teach them how to assess the skin for lesions. Encourage frequent skin assessment and early diagnosis for improved outcomes.

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PSORIASIS

DEFINITION

A. A common benign, chronic, inflammatory skin disorder, psoriasis is characterized by whitish/silver scaly patches commonly seen on the scalp, knees, and elbows.

INCIDENCE

A. Psoriasis occurs in 1% to 3% of the world population.

B. Psoriasis affects 7.5 million people in the United States, with 80% of them having plaque psoriasis.

C. It occurs at any age:

1. Peak of onset seen in adolescence.

2. Young adult (16–22 years old).

3. Adult (50–60 years old).

PATHOGENESIS

A. Etiology is unknown; this is a multifactorial disease with a definite genetic component. Hyperproliferation of the epidermis and inflammation of the epidermis and dermis are seen, with epidermal transit time rapidly increased (six- to nine-fold). A T-lymphocyte-mediated dermal immune response may be due to a microbial antigen or autoimmune process.

PREDISPOSING FACTORS

A. Family history.

B. Drugs that exacerbate condition:

1. Lithium.

2. Beta-blockers.

3. Nonsteroidal anti-inflammatory drugs (NSAIDs).

4. Antimalarials.

5. Sudden withdrawal of systemic or potent topical corticosteroids.

C. Stress (common triggering factor).

D. Local trauma or irritation.

E. Recent streptococcal infection.

F. Alcohol use.

G. Tobacco use.

H. HIV association; suspected if onset is abrupt.

I. Obesity.

J. Vitamin D deficiency.

COMMON COMPLAINT

A. Dry scaly rash.

OTHER SIGNS AND SYMPTOMS

A. Pruritic and/or painful lesions.

B. Silvery scales on discrete erythematous plaques.

1. Onset commonly occurs as a guttate form with small, scattered, teardrop-shaped papules and plaques after a streptococcal infection in a young adult.

2. Larger, chronic plaques occur later in life.

C. Lesions are commonly seen on scalp, elbows, and knees, but may involve any area of the body.

D. Glossitis or geographic tongue: small pits or yellow-brown spots (oil spots).

E. Positive Auspitz sign: punctate bleeding points with removal of scale.

F. Onycholysis.

G. Stippled nails and pitting; approximately 50% of affected individuals have nail involvement.

H. Periarticular swelling of small joints of fingers and toes. **Joint pain and involvement signals psoriatic arthritis.**

I. Pustular variant with predominant involvement of hands and/or feet, including nails.

SUBJECTIVE DATA

A. Question the client regarding any predisposing factors listed earlier to identify risk factors.
B. Ask the client if there have been changes in the course of symptoms.
C. Ascertain whether the symptoms worsen in winter and clear in summer.
D. Determine site of lesion and whether the onset is sudden or painful.
E. Ask the client to describe the skin. Is it itchy or painful?
F. Assess lesions for any associated discharge (blood or pus).
G. Ask if the client is using any new soaps, creams, or lotions.
H. Rule out any exposure to industrial or domestic toxins.
I. Ask the client about any possible contact with venereal disease (sexually transmitted infections [STIs]).
J. Review whether there was close physical contact with others with skin disorders.
K. Elicit information regarding any preceding systemic symptoms (fever, sore throat, and anorexia).

PHYSICAL EXAMINATION

A. Check temperature (if indicated).
B. Inspect:
  1. Inspect skin; note type of lesion and distribution. Assess oral mucosa, nails, and nail beds.
  2. Assess joints for erythema and/or synovitis (inflammation of the synovial membrane).
C. Palpate:
  1. Palpate joints for tenderness and synovitis.

DIAGNOSTIC TESTS

A. Skin biopsy may be performed if the diagnosis remains uncertain.
B. If HIV infection is suspected, order HIV test.
C. If joint inflammation is present, consider rheumatoid factor, erythrocyte sedimentation rate, C-reactive protein, and uric acid.
D. If there is a history of streptococcal infection, order antistreptolysin O titer.

DIFFERENTIAL DIAGNOSES

A. Psoriasis.
B. Scalp: seborrheic dermatitis.
C. Body folds: candidiasis.
D. Trunk: pityriasis rosea, tinea corporis.
E. Hand dermatitis.
F. Squamous cell carcinoma (SCC).
G. Cutaneous lupus erythematosus.
H. Eczema.

PLAN

A. General interventions:
  1. This is a chronic disorder that requires long-term treatment, a high degree of client involvement, and therapy that is simple and inexpensive.
  2. Aim of treatment is control, not cure.
  3. Exposure to sunlight may be beneficial. However, a small percentage of clients worsen with exposure to sunlight.
  4. The sequence of agents for involvement of less than 20% body surface is as follows:
    a. Emollients (Eucerin cream or Aquaphor cream).
    b. Keratolytic agents (salicylic acid gel or ointment).
    c. Topical corticosteroids: Use lowest potency to control disease.
    d. Calcipotriene ointment: vitamin D analogue (calcipotriene ointment 0.005%).
    e. Anthralin: Use as short-contact therapy 1% to 3%.
    f. Coal tar (Estar, Psorigel): Use in conjunction with topical steroids or anthralin. It can be applied at bedtime or in the morning for 15 minutes and then shower off.
    g. Medicated shampoos: Useful for scalp psoriasis, in conjunction with topical steroids and other treatments.
B. Client teaching: *See Client Teaching Guide for this chapter, "Psoriasis."*
  1. Help the client understand the chronic nature of this disease characterized by flares and remission. Teach stress monitoring and control. Assist with coping techniques.
  2. A trial of a gluten-free diet may be tried to help symptoms. See Appendix B for Gluten-Free Diet.
  3. Encourage the client to reach out to the National Psoriasis Foundation for education and resources for them and their family.
C. Pharmaceutical therapy:
  1. If disease is not controlled with first agent, then an alternative agent may be tried.
    a. Mild to moderate disease: Topical steroids are first-line therapy.
    b. Emollients to start treatment (e.g., Eucerin Plus lotion or cream, Lubriderm Moisture Plus, Moisture).
    c. Scalp: Use coal tar shampoo (Zetar, T/Gel, Pentrax) in place of regular shampoo two times per week.
      i. Apply lather to scalp, allow to soak for 5 minutes, and then rinse.
      ii. If the scale is very thick, use P and S Liquid (over-the-counter [OTC]). Massage in at night and wash out in the morning.
    d. For additional treatment as needed, apply triamcinolone acetonide 0.1% (Kenalog 0.1%) lotion or equivalent to scaly, stubborn areas once or twice daily until controlled. Avoid the face.
    e. Dovonex scalp solution: Apply on dry scalp as directed.
    f. Face and skin folds: Apply hydrocortisone cream 1% sparingly up to 4 weeks, preferably no more than 2 weeks. If lesions are unresponsive, consider increasing to 2.5% and taper quickly with improvement.
    g. Body, arms, and legs: Use triamcinolone acetonide 0.025% (Aristocort A) cream BID up to 2 weeks. **Avoid normal skin.**
    h. For thick plaques, try Keralyt gel (6% salicylic acid), then corticosteroids.
    i. Coal tar (Estar gel) once or twice daily in combination with corticosteroids.
    j. Anthralin (Dritho-Creme) is beneficial as an alternate to steroid lotion for scalp psoriasis. **Avoid sunlight.**
    k. Vitamin D_3 analog (Calcipotriol), BID up to 8 weeks, is comparable to midpotency corticosteroids. **Avoid the face and skin folds.**

FOLLOW-UP

A. See the client in 2 to 3 weeks to evaluate treatment.
B. Follow up in 2 months to monitor side effects.
C. Follow-up must be individualized for each client.

CONSULTATION/REFERRAL

A. Medical management: For involvement greater than 20% of the body, refer the client to a dermatologist for the following:
  1. Light therapy with UVA and UVB. UVB light therapy is often used in conjunction with keratolytic agents.
  2. Synthetic retinoids: etretinate or acitretin.
  3. Low-dose cyclosporine or Azulfidine.
B. Refer clients with extensive disease, psoriatic arthritis, or inflammatory disease to a rheumatologist. Medications are used to suppress the immune system's response, and include adalimumab (Humira), alefacept (Amevive), etanercept (Enbrel), infliximab (Remicade), and ustekinumab (Stelara).
C. Cases of generalized pustular psoriasis of exfoliative erythroderma should be referred immediately to a dermatologist.
D. All systemic therapies should be given under supervision of a dermatologist or a rheumatologist.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
  1. Psoriasis may get better or worse during pregnancy. Use of moisturizers and emollients is strongly encouraged.
  2. Corticosteroids are safe to use if using low or medium strength. Use may cause an increase in developing stretch marks.
  3. Treatment with phototherapy is safe using broadband UVB and narrowband UVB.
  4. Avoid use of methotrexate, acitretin, and tazarotene.
B. Geriatrics:
  1. First-line therapy for mild to moderate psoriasis in the older adult population is topical treatments. Systemic steroid therapies place geriatrics at a higher risk of adverse effects, including atrophy, rebound phenomenon, tachyphylaxis, secondary skin infections, telangiectasia, and purpura.
  2. If topical treatment is not effective, before considering other treatments, do an assessment for client adherence and physical function abilities. Verify the client is using the medication correctly and consistently.
  3. If the medication is being implemented appropriately, then consider second-line treatments of systemic medications or UVB phototherapy. Second-line therapies must have a thorough review of comorbidities, any client needs for assistance, and an evaluation of functional status before initiating the systemic/phototherapy.

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SCABIES

DEFINITION

A. Scabies is a contagious skin infestation by the mite *Sarcoptes scabiei.*

INCIDENCE

A. Scabies occurs mainly in individuals in close contact with many other individuals, such as nursing home residents. It is rare among African Americans.

PATHOGENESIS

A. Scabies is transmitted through close contact with an individual who is infested with the mite *S. scabiei.* Transmission may occur through sexual contact or contact with mite-infested clothing or sheets. The fertilized female mite burrows into the stratum corneum of a host and deposits eggs and fecal pellets. Larvae hatch, mature, and repeat the cycle.
B. A hypersensitivity reaction is responsible for the intense pruritus.

PREDISPOSING FACTORS

A. Close contact with large numbers of individuals.
B. Institutionalization.
C. Poverty.
D. Sexual promiscuity.

COMMON COMPLAINTS

A. Intense itching, worse at night.
B. Skin excoriation.
C. Generalized pruritus.
D. Rash.

OTHER SIGNS AND SYMPTOMS

A. Mites burrow in finger webs, wrists, sides of the hands and feet, axilla and buttocks, and in the penis and scrotum in males.
B. Discrete vesicles and papules, distributed in linear fashion.
C. Erythema.
D. Secondary bacterial infections due to scratching (pustules and pinpoint erosions).
E. Nodules in covered areas (buttocks, groin, scrotum, penis, axilla), which may have slightly eroded surfaces that persist for months after mites have been eradicated.
F. Diffuse eruption that spares face.

SUBJECTIVE DATA

A. Elicit information regarding housing conditions, close contact, or sexual contact with potentially infected individuals.
B. Question the client regarding onset, duration, and location of itching.

PHYSICAL EXAMINATION

A. Check temperature.
B. Inspect:
  1. Examine all body surfaces with client unclothed.
  2. Use a magnifying lens to identify characteristic burrows in finger webs, wrists, and penis.
  3. Inspect adult pubic area for lesions.

DIAGNOSTIC TESTS

A. Three findings are diagnostic of scabies:
  1. Microscopic identification of *S. scabiei* mites.
  2. Eggs.
  3. Fecal pellets (scybala).

B. Burrow identification: Ink the suspected area with a blue or black felt-tipped pen, then wipe with an alcohol swab. The burrow absorbs the ink, while the surface ink is wiped clean.

C. A tiny black dot may be seen at the end of a burrow, which represents the mite, ova, or feces, and can be transferred by means of a 25-gauge hypodermic needle to immersion oil on a slide for microscopic identification.

D. Place a drop of mineral oil on a suspected lesion, scrape lesion with a #15 blade, and transfer the shaved material to a microscope slide for direct examination of the mite under low power.

DIFFERENTIAL DIAGNOSES

A. Scabies.
B. Atopic dermatitis.
C. Insect bites.
D. Pityriasis rosea.
E. Eczema.
F. Seborrheic dermatitis.
G. Syphilis.
H. Pediculosis.
I. Allergic or irritant contact dermatitis.

PLAN

A. General interventions:
  1. Implement comfort measures to reduce pruritus.
  2. Treat secondary bacterial infection(s) with antibiotics.
  3. Household members should be treated simultaneously as a prophylactic measure and to reduce the chance of reinfection.
  4. The client should be advised that pruritus may continue for up to a week even with a successful treatment due to local irritation.

B. Client teaching: *See Client Teaching Guide for this chapter, "Scabies."*

C. Pharmaceutical therapy:
  1. First-line of therapy, due to its low toxicity, is 5% permethrin (Elimite cream) applied to all body areas from neck down and washed off in 8 to 14 hours. One application is highly effective, but some dermatologists recommend retreatment in 1 week.
  2. An oral dose of the anthelmintic agent ivermectin (200 mcg/kg) followed by a repeat dose in 1 to 2 weeks has been shown to be effective and to rapidly control pruritus in healthy clients and individuals with HIV.
  3. Diphenhydramine (Benadryl) 25 to 50 mg may be given PO every 4 to 6 hours if indicated for pruritus. Other nonsedating antihistamines may be used. Toxicity is usually a result of client overtreatment (failure to follow prescribed regimen). Advise the client of this danger.

FOLLOW-UP

A. Follow up in 2 weeks to assess treatment response.

CONSULTATION/REFERRAL

A. Consult or refer the client to a physician if, at 2-week follow-up, pharmaceutical therapy has been ineffective.

INDIVIDUAL CONSIDERATIONS

A. Partners:
  1. All intimate contacts within the past month and close household and family members should be treated.

B. Geriatrics:
  1. Older adults tend to have more severe pruritus despite fewer lesions.
  2. They are at risk of extensive infections due to an age-related decline in immunity.
  3. The excoriations may become severe and may be complicated by cellulitis.
  4. Red flags:
    a. Avoid first-generation anticholinergics due to risk of confusion, dry mouth, constipation, and other anticholinergic effects or toxicity.
    b. Geriatric clients who have dementia or declining functional abilities need a thorough home environment assessment and an evaluation for any issues of neglect or abuse.

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SEBORRHEIC DERMATITIS

DEFINITION

A. A common chronic, erythematous, scaling dermatosis, seborrheic dermatitis occurs in areas of the most active sebaceous glands, such as the face and scalp, body folds, and presternal region.

INCIDENCE

A. Seborrheic dermatitis is very common, affecting approximately 1% to 5% of the general population.

B. Incidence is higher in HIV-infected individuals, ranging from 34% to 83%.

PATHOGENESIS

A. Etiology is unknown. There is a possibility that it is hormonally dependent, has a fungal *(Pityrosporum ovale* or *Candida albicans)* component, is neurogenic, or may reflect a nutritional deficiency.

B. Currently, it is identified as an inflammatory disorder that most probably results from a dysfunction of sebaceous glands.

PREDISPOSING FACTORS

A. Possible link between infantile and adult forms.
B. Possible familial trend.
C. High association with HIV-infected individuals.

COMMON COMPLAINTS

A. "Dandruff," dry flaky scalp.
B. Rash with "sticky flakes."

Often no presenting complaints are found on a routine physical exam.

OTHER SIGNS AND SYMPTOMS

A. Variable pruritus, often increased with perspiration and winter.

B. Oily, flaking skin on an erythemic base around ears, nose, eyebrows, and eyelids. ▶

C. Red, cracking skin in body folds, axilla, groin, or anogenital, submammary, or umbilical areas.
D. Primary lesions: plaques.
E. Secondary lesions: erythema, scales, fissures, exudate, and symmetric eyelid involvement.
F. Lesions with drainage or crusting may indicate secondary bacterial infection.
G. Distribution area in adults: scalp, eyebrows, paranasal area, nasolabial fold, chin, behind ears, chest, and groin.

SUBJECTIVE DATA

A. Identify location, onset, and progression of symptoms.
B. Ask the client to describe symptoms. Ask if the skin is itchy or painful.
C. Assess lesions for any associated discharge (blood or pus).
D. Elicit information regarding use of topical medications, soaps, creams, or lotions. Quiz client regarding any oral medications being taken.
E. Determine whether there were any preceding systemic symptoms (fever, sore throat, anorexia, or vaginal discharge).
F. Rule out any possible exposure to industrial or domestic toxins.
G. Ask the client to identify what improves or worsens this condition.

PHYSICAL EXAMINATION

A. Inspect:
  1. Inspect skin; note areas of lesions and distribution.
  2. Assess eyes for blepharitis.
  3. Inspect ears and nose.
B. Palpate:
  1. Palpate the skin, noting texture and moisture.

DIAGNOSTIC TESTS

A. None required.
B. May consider possible skin biopsy to rule out other conditions if suspicious.

DIFFERENTIAL DIAGNOSES

A. Seborrheic dermatitis.
B. Atopic dermatitis.
C. Candidiasis.
D. Dermatophytosis.
E. Histiocytosis X.
F. Psoriasis vulgaris.
G. Rosacea.
H. Systemic lupus erythematosus.
I. Tinea capitis.
J. Tinea versicolor.
K. Vitamin deficiency.
L. Impetigo.
M. Eczema.
N. Secondary syphilis.

PLAN

A. General interventions:
  1. Shampooing is the foundation of treatment.
    a. Use medicated shampoos as directed (Selsun Blue, Exsel, or Nizoral).
    b. Shampoo daily with baby shampoo using a soft brush.
    c. If scalp does not clear after 1 to 2 weeks of treatment, it is appropriate to use ketoconazole 2% cream.
  2. Seborrheic blepharitis:
    a. Hot compress plus gentle debridement with cotton-tipped applicator and baby shampoo twice a day.
    b. For secondary bacterial infection, sulfacetamide sodium 10% (ophthalmic Sodium Sulamyd).
    c. Continue treatment for several days after lesions disappear.
B. Client teaching: *See Client Teaching Guide for this chapter, "Seborrheic Dermatitis."*
C. Pharmaceutical therapy:
  1. Most shampoos should be used two times per week. Those with coal tar can be used three times per week.
  2. Medicated shampoos:
    a. Coal tar (Denorex, T/Gel, Pentrax, Tegrin) shampoo: Apply as directed.
    b. Salicylic acid (Ionil Plus, P and S) shampoo: Apply as directed.
    c. Selenium sulfide (Exsel, Selsun Blue): Shampoo daily.
    d. Ketoconazole 2% (Nizoral) cream: Apply to the affected area BID for 4 to 6 weeks.
    e. Combination shampoos: coal tar and salicylic acid (T/Sal); salicylic acid and sulfur (Sebulex). These shampoos may be used one to two times a week, alternating with other shampoos during the week. Always apply corticosteroids as thin layer only; avoid eyes.
  3. Topical corticosteroid lotions or solutions: Use in combination with medicated shampoo if 2 to 3 weeks of treatment with shampoo alone fails.
    a. Adults: scalp:
      i. Start with medium potency, for example, betamethasone valerate 0.1% lotion 20 to 60 mL BID.
      ii. If treatment is not effective in 2 weeks, increase potency, for example, fluocinonide 0.05% solution 20 to 60 mL BID, or fluocinolone acetonide 0.01% oil 120 mL nightly with shower cap.
      iii. As dermatitis is controlled, reduce to mild potency, for example, hydrocortisone 1% to 2.5% lotion 60 to 20 mL once or twice daily.
    b. Adults: face or groin:
      i. Low-potency agents, for example, hydrocortisone 1% cream or desonide 0.05% cream once or twice daily.
      ii. Consider lotion for eyebrows for easier application.
      iii. Metronidazole 1% gel on face once or twice daily.
  4. Recalcitrant disease:
    a. Add ketoconazole 2% cream (15, 30, or 60 g) every day.
    b. Sulfacetamide sodium 10%, with sulfur 5%, lotion 25 g once or twice daily.

FOLLOW-UP

A. Advise the client to call the office in 5 to 6 days to report progress.
B. The client should return to the office if no improvement is seen.

CONSULTATION/REFERRAL

A. Refer the client to a dermatologist if the condition does not clear in 10 to 14 days.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Ketoconazole is not recommended.

B. Geriatrics:

1. Seborrheic dermatitis is one of the most common inflammatory skin disorders in the older adult population. Clients with light dry skin are at greater risk of an occurrence, especially during the winter.

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TINEA

DEFINITION

A. Tinea corporis (ringworm) is a fungal infection of the skin tissue (keratin) commonly seen on the trunk and extremities.

B. Tinea capitis is a fungal infection of scalp hair.

C. Tinea cruris is a fungal infection of the groin.

D. Tinea pedis is a fungal infection of the foot.

E. Tinea unguium (dermatophyte onychomycosis) is a fungal infection of the nail.

INCIDENCE

A. Tinea is a fairly common fungal infection seen in adults.

PATHOGENESIS

A. The causative fungal species varies, depending on the location of the infection. Three common organisms are *Epidermophyton, Microsporum*, and *Trichophyton.*

B. The infection can be obtained from other people, environmental contact with inanimate objects, animals (puppies, kittens), and the soil.

PREDISPOSING FACTORS

A. Exposure to person or facilities (e.g., locker rooms) infected with the fungus.

B. Poor nutrition.

C. Poor health.

D. Poor hygiene.

E. Warm climates.

F. Immunosuppression.

COMMON COMPLAINTS

A. Scaly, itchy skin.

B. When circular in shape, usually tinea corporis (ringworm).

C. When nails are thickened and yellow, usually tinea unguium.

OTHER SIGNS AND SYMPTOMS

A. Tinea capitis: erythema, scaling of scalp, with hair loss at site, in which client is asymptomatic of hair loss.

B. Tinea corporis: circular, erythematous, well-demarcated lesion on the skin with hypopigmentation at the center of the lesion; usually pruritic.

C. Tinea cruris: well-demarcated scaling lesions on groin (not scrotum) or thigh; usually pruritic.

D. Tinea pedis: scaly, erythemic vesicles on feet, between toes, and in arch, with extreme pruritus.

E. Tinea unguium (onychomycosis): thickening and yellowing of toenail or fingernail, often with other fungal infection or alone.

SUBJECTIVE DATA

A. Ask the client about onset, duration, and progression of patch or rash on skin.

B. Assess the client for other areas of skin involvement.

C. Ask if the lesion is pruritic.

D. Inquire as to the client's exposure to anyone with similar symptoms.

E. Determine whether the client has a history of similar lesions.

F. Query the client regarding predisposing factors.

G. Review with the client what remedies were used and with what results.

PHYSICAL EXAMINATION

A. Check temperature (if indicated).

B. Inspect:

1. Examine all areas of skin.

2. Note type of lesions present.

DIAGNOSTIC TESTS

A. Obtain scrapings of the border of the lesion for evaluation.

1. Potassium hydroxide (KOH).

2. Wet prep.

3. Fungal cultures.

DIFFERENTIAL DIAGNOSES

A. Tinea corporis.

B. Dermatitis.

C. Alopecia areata.

D. Psoriasis.

E. Contact dermatitis.

F. Atopic eczema.

G. Subacute cutaneous lupus erythematosus.

H. Pityriasis rosea.

PLAN

A. General interventions:

1. Identify type of lesion.

2. Identify other infected family members or sexual partners for treatment.

B. Client teaching: *See Client Teaching Guide for this chapter, "Tinea."*

1. Reinforce medication regimen for a 4- to 8-week period for resolution.

C. Pharmaceutical therapy:
1. Tinea capitis:
a. Adults: griseofulvin 500 mg PO each day for 4 to 6 weeks.
2. Tinea corporis, pedis, and cruris:
a. Clotrimazole 1% (Lotrimin) cream or econazole nitrate 1% cream, BID for 14 to 28 days.
b. Terbinafine 1% cream (Lamisil), topical; apply once or twice daily for 1 to 4 weeks.
3. Onychomycosis: Successful treatment is difficult.
a. Itraconazole (Sporanox) 100 mg, two tablets PO BID for 7 days. Repeat in 1 month, then repeat again in 1 more month.

Monitor liver function tests (LFTs) at 6 weeks after starting medication.

b. Terbinafine.
i. Fingernail: 250 mg once daily for 6 weeks.
ii. Toenail: 250 mg daily for 12 weeks.
c. Home cure: Apply Vicks VapoRub on toenail bed and cover with a sock every night at bedtime for approximately 4 to 6 months or until resolved. This treatment offers a safe, cost-effective alternative to oral medications.

FOLLOW-UP
A. A 2- to 4-week follow-up is recommended to evaluate progress.
B. When using Sporanox, monitor liver function tests (LFTs) at 6 weeks. If the medication is continued, monitoring LFTs every 6 to 8 weeks is recommended.

CONSULTATION/REFERRAL
A. Consult a physician if the infection has not improved.

INDIVIDUAL CONSIDERATIONS
A. Pregnancy:
1. Oral antifungal medications are not recommended during pregnancy.
B. Adults:
1. Tinea capitis is rare in adults.
2. Tinea cruris is more common in obese males but rare in females.
3. Transient and/or permanent hearing loss has been documented in some clients with use of itraconazole. These cases were reported when itraconazole was used with quinidine, which is contraindicated.
C. Geriatrics:
1. Itraconazole should be used cautiously in older adults.
2. Recommendation for tinea treatment with the older adult population is terbinafine due to its decreased risk for cardiac complications and drug interactions if on multiple other medications.

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TINEA VERSICOLOR

DEFINITION
A. Tinea versicolor is a fungal infection of the skin which may be chronic in nature. It is most commonly seen on the upper trunk; however, it may spread to extremities.

INCIDENCE
A. Tinea versicolor is seen most frequently in adolescents and young adults.

PATHOGENESIS
A. Tinea versicolor is a fungal infection of the skin caused by an overgrowth of *Malassezia* (formerly known as the *Pityrosporum orbiculare)*, part of the normal skin flora.
B. Discoloration of the skin is seen, forming round or oval maculae, which may become confluent.
C. Maculae range from 1 cm to very large, greater than 30 cm.

PREDISPOSING FACTORS
A. Immunosuppressed
B. Pregnancy.
C. Warm temperatures.
D. Corticosteroid therapy.
E. Genetic Predisposition.

COMMON COMPLAINTS
A. Scaly rash on the upper trunk with occasional mild itching.

OTHER SIGNS AND SYMPTOMS
A. Annular maculae with mild scaling.
B. Asymptomatic or pruritic.
C. Pink-, white-, or brown-colored rash.

SUBJECTIVE DATA
A. Ascertain when and where the rash began.
B. Have the client describe how the rash has changed.
C. Assess the client for any associated symptoms with the rash, such as itching and burning.
D. Identify what products the client has used on the skin to treat rash and with what results.
E. Elicit information regarding a history of similar rashes.
F. Query the client regarding current medications.
G. Review any medical history for comorbid conditions.

PHYSICAL EXAMINATION
A. Inspect:
1. Inspect skin and note type of lesion.
2. Examine other areas of skin for similar lesions.

DIAGNOSTIC TESTS
A. Wet prep/potassium hydroxide (KOH).
B. Wood lamp: Wood light is useful in examining skin to determine the extent of infection. Inspection of fine scales with Wood lamp reveals scales with a pale yellow-green fluorescence that contain the fungus.

C. Culture lesion: When obtaining a sample scraping, obtain sample from edge of lesion for best sample of hyphae. (Hyphae and spores have a "spaghetti and meatball" appearance.)

DIFFERENTIAL DIAGNOSES

A. Tinea versicolor.
B. Tinea corporis.
C. Pityriasis alba.
D. Pityriasis rosea: Herald patch is clue to diagnosis.
E. Seborrheic dermatitis.
F. Vitiligo.
G. Secondary syphilis.

PLAN

A. General interventions:
  1. Apply medication as directed.
C. Client teaching: *See Client Teaching Guide for this chapter, "Tinea Versicolor."*
  1. Because causative species is a normal inhabitant of skin flora, recurrence is possible.
  2. Skin pigmentation returns after infection has cleared up. This may take several months to resolve.
D. Pharmaceutical therapy:
  1. Selenium sulfide 2.5% (Selsun Blue):
    a. Advise client to shower at bedtime. Then apply selenium sulfide 2.5% cream to skin, covering entire body from chin down to toes. Leave treatment on skin for 8 to 10 hours. Shower off in the morning.
    b. A second treatment option includes applying the selenium sulfide 2.5% cream to skin lesions daily for 12 days. Leave treatment on skin for 30 minutes, then shower off.
    c. Treatment may be needed monthly until desired results are obtained. **Encourage use of Selsun Blue on entire body surface except on the face and head.**
  2. Other medications used:
    a. Clotrimazole 1% cream BID for 4 weeks.
    b. Ketoconazole 2% (Nizoral) cream daily for 14 days.
    c. Fluconazole 300 mg once weekly for 2 weeks. **When using fluconazole as treatment, caution the client regarding liver damage with toxicity.**

FOLLOW-UP

A. None required if resolution occurs.
B. Monitor liver function tests (LFTs) every 6 weeks while on oral medication.

CONSULTATION/REFERRAL

A. Consult with a physician if current treatment is unsuccessful.

INDIVIDUAL CONSIDERATIONS

A. Adults:
  1. Commonly seen in young adults.
B. Geriatrics:
  1. Rare in the geriatric population.

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WARTS

DEFINITION

A. A wart is an elevation of the epidermal layer of the skin (skin tumor). Warts are caused by the human papillomavirus (HPV).

INCIDENCE

A. Warts occur in people of all ages, more commonly in children and those in early adulthood.
B. By adulthood, 90% of all people have positive antibodies to the virus.
C. Warts are seen more frequently in females than in males.

PATHOGENESIS

A. A circumscribed mass develops on the skin that is limited to the epidermal layer. The virus, papillomavirus, is located within the nucleus of the cell.
B. The virus may be transmitted by touch and is commonly seen on the hands and feet.
C. Most warts resolve without treatment within 12 to 24 months.

PREDISPOSING FACTORS

A. Skin trauma.
B. Immunosuppression.
C. Exposure to public showers, pools, locker rooms, and so forth.

COMMON COMPLAINTS

A. Bump on the skin or specific area of the body (hands, feet, arms, and legs).
B. Usually painless unless present on the bottom of the foot.

OTHER SIGNS AND SYMPTOMS

A. Common wart (verruca vulgaris): flesh-colored, irregular lesion with rough surface; black dots in center of lesion occasionally seen, which is thrombosed capillaries; can occur on any body part.
B. Filiform wart (verruca filiformis): thin, threadlike, projected papule on face, lips, nose, or eyelids.
C. Flat wart (verruca plana): flat-topped, flesh-colored papule, 1 to 3 mm in diameter, with smooth surface; seen in clusters or in a line, on face and extremities.
D. Plantar wart (verruca plantaris): firm papule, 2 to 3 cm in diameter, indented into skin with verrucous surface; painful with ambulation, when placed on ball or heel of foot.
E. Genital warts: See the topics on sexually transmitted infections in Chapter 17.

SUBJECTIVE DATA

A. Determine onset, location, and duration of wart.
B. Elicit information regarding a history of warts.
C. Identify with the client what treatment has been used in the past and what the results were. Question the client regarding ▶

length of time over-the-counter (OTC) medications were used and how aggressive they were with the treatment.

PHYSICAL EXAMINATION

A. Inspect:
  1. Assess skin for lesions, noting location, appearance, size, and surface texture of wart.
  2. Examine the entire body for other lesions.

DIAGNOSTIC TESTS

A. None indicated.

DIFFERENTIAL DIAGNOSES

A. Wart:
  1. Verruca vulgaris.
  2. Filiform wart.
  3. Verruca plana.
  4. Verruca plantaris.
B. Seborrheic keratosis.
C. Callus.
D. Molluscum contagiosum: flesh-colored group of firm papules found on the face, trunk, and/or extremities. A white core may be expressed from lesion. Lesion may be successfully removed by curettage or cryotherapy.
E. Corn.
F. Skin cancer.

PLAN

A. General interventions:
  1. Identify type of wart.
  2. Treatment is based on the type of wart.



B. Client teaching: *See Client Teaching Guide for this chapter, "Warts."*
C. Pharmaceutical therapy:
  1. Common wart:
    a. After soaking and filing wart with a nail file, apply one of the following:
      i. Salicylic acid 17% (Compound W) gel BID for up to 12 weeks, if needed. Keep site covered with adhesive and bandage.
      ii. Apply duct tape to site after treatment. Repeat this treatment every night for up to 12 weeks or until resolved.
    b. Apply cryotherapy with liquid nitrogen to the site. Repeat every 3 to 4 weeks until resolved. Apply adhesive tape over the site and keep covered.
  2. Flat wart:
    a. Retinoic acid: Apply to the site BID for 4 to 6 weeks.
    b. Imiquimod (Aldara) 5% cream may be applied by the client at home. Although labeled use is for genital warts, the client may consider off-label use at bedtime and wash off after 6 to 8 hours every other day until resolved. Precautions should be stressed regarding caustic nature of the cream to healthy skin.
  3. Plantar wart:
    a. Salicylic acid 40% (Mediplast): Apply over wart. Remove in 24 to 48 hours and remove dead skin with stone or by scraping or using nail file. Repeat every 24 to 48 hours until wart is removed. May take up to 6 to 8 weeks.
  4. Filiform wart:
    a. Apply cryotherapy with liquid nitrogen to site. Repeat every 3 to 4 weeks until resolved. Apply adhesive tape over the site and keep it covered.
    b. Refer to a dermatologist for surgical removal.
  5. Educate client to throw away emery board and nail file after each use. If using nail file, cleanse after each use with alcohol.

FOLLOW-UP

A. Follow the client every 4 to 6 weeks until resolved.

CONSULTATION/REFERRAL

A. If diagnosis is unclear, refer the client to a dermatologist for surgical excision and biopsy.

INDIVIDUAL CONSIDERATIONS

A. Warts occur in all ages but are less common in older adults. Use caution if client elects cryotherapy to treat a wart secondary to thinness of aging skin. Always assess if the client is taking aspirin or anticoagulant therapy prior to incisional removal or cryotherapy.

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WOUND CARE: LOWER EXTREMITY ULCER

DEFINITION

A. Vascular ulcer:
  1. Arterial/ischemic ulcer:
    a. Skin ulcers usually found from the toes to the ankle region; ulcers fail to heal due to inadequate arterial flow.
  2. Venous ulcer:
    a. Chronic skin and subcutaneous lesions are usually found on lower extremity between the ankle and knee, thought to occur from intracellular edema or inflammatory processes.
B. Diabetic foot ulcer:
  1. Skin ulcers usually found on the plantar surface of the feet, or metatarsals, most commonly occurring from trauma or plantar pressure.

INCIDENCE

A. Diabetic foot ulcers precede over 80% of lower extremity amputations in the United States.
B. The financial burden of venous ulcers is estimated to be $2 billion per year in the United States.
C. Up to 20% of lower extremity ulcers have been shown to have mixed etiology disease.

PATHOGENESIS

A. An ulcer that is found between the knees and toes constitutes a lower extremity ulcer, and guidelines are based according to the etiology. The thing to remember with lower ▶

extremity ulcers is that they often may have more than one cause. The most common etiologies are venous insufficiency, arterial insufficiency, diabetic foot ulcer, and/or pressure.

PREDISPOSING FACTORS

A. Arterial insufficiency.
B. Congestive heart failure.
C. Coronary heart disease.
D. Diabetes.
E. Edema.
F. Dyslipidemia.
G. Obesity.
H. Age: older than 65 years.
I. Venous insufficiency.
J. Peripheral neuropathy.
K. Protein calorie malnutrition.
L. Immobility.

COMMON COMPLAINTS

A. Lower extremity or foot pain.
B. Bleeding.
C. Drainage.
D. Hyperglycemia.

SUBJECTIVE DATA

A. Ask the client to describe the location and onset. What do they think may have caused an ulcer in that area? Was the onset sudden or gradual? How have the symptoms continued to develop?
B. Assess if the area is pruritic or painful. Does the client have feeling in the area or is there a decrease in sensation noted?
C. Assess for any associated drainage. Ask about the color and if any odor is present.
D. Complete a drug history. Ask the client if they are taking any steroids or anticoagulants.
E. Has the client been treated for this location in the past? If so, ask to describe.
F. Determine whether the client has attempted to treat this at home. If yes, inquire about previous treatments and for how long.
G. Does the client have any numbness or tingling in the lower extremities? Does the client wake up at night with pain? Do they have any pain with ambulation? Do they have sensation to their feet?
H. Rule out any possible exposure to industrial or domestic toxins or insect bites.
I. Assess for iodine and sulfa allergies before starting treatment.

PHYSICAL EXAMINATION

A. Check temperature, pulse, pulse ox, respirations, and blood pressure.
B. Inspect:
1. Assess the lower extremities, feet, and metatarsals:
a. Color of the skin:
i. Assess skin, beginning at the top of the legs; move down the legs to the metatarsals for changes in color that may exhibit signs of ischemia.
ii. Hemosiderin staining may exhibit venous insufficiency.
b. Temperature of the skin.
c. Sensation of the skin.
d. Capillary refill.
e. Pulses.
f. Edema.
2. Inspect the ulcer.
a. Measure length × width × depth.
i. Undermining (destroyed tissue below the wound margin): Measure and note location, using the face of a clock to document the site of undermining: for example, 12 o'clock, 3 o'clock, 6 o'clock, or 9 o'clock.
ii. Tunneling (destroyed tissue pathway that creates dead space underneath the skin): Measure and note the location, using the face of a clock to document the site of tunneling: for example, 12 o'clock, 3 o'clock, 6 o'clock, or 9 o'clock.
b. Describe the wound bed.
i. Tissue in the wound bed:
1) Necrotic tissue, slough tissue, granulation tissue, epithelial tissue (percentage of each should together equal 100%).
ii. Drainage:
1) Amount:
a) None, scant, moderate, copious.
2) Color:
a) Serous, purulent, yellow, serosanguineous, or green.
iii. Odor:
1) Odor presence: yes or no.
iv. Periwound:
1) Intact.
2) Not intact.
a) Describe periwound. Note erythema, fever, induration, maceration, excoriation, calloused area, or epiboly (rolled edges).

DIAGNOSTIC TESTS

A. For perfusion:
1. Ankle brachial index (ABI).
2. Arterial Doppler.
3. Venous Doppler.
B. For infection:
1. Partial or full thickness:
a. Wound culture and sensitivity.
2. Full thickness with concern of underlying structure infection:
a. X-ray.
b. MRI
c. Bone scan.
C. For pathology:
1. Wound biopsy.
D. For medical management:
1. Complete blood count (CBC) basic metabolic panel (BMP).
2. Hemoglobin A1c (Hgb A1c).
3. Protein, albumin, prealbumin, and zinc level.

DIFFERENTIAL DIAGNOSES

A. Vascular ulcer:
1. Arterial/ischemic ulcer.
2. Venous ulcer.
B. Diabetic foot ulcer.
C. Abscess.
D. Atypical ulcers.
E. Dermatologic disorder.
F. Necrotizing fasciitis.
G. Skin cancers.
H. Pressure ulcer.

I. Trauma.
J. Pyoderma gangrenosum.

PLAN

A. General interventions:
1. Vascular ulcers:
a. Arterial ulcer:
i. Refer to vascular surgery for assessment to improve arterial flow.
ii. Refer to a wound care specialist.
b. Venous ulcer:
i. Establish arterial flow.
1) Refer to a vascular surgeon if deficiency is found.
ii. For signs and symptoms of infection, treat the infection first with tissue culture and sensitivity. Treat per pharmaceutical recommendations. Treat with silver alginate to the site for moderate drainage and silver gel to the site for scant drainage.
iii. Once arterial flow has been established as sufficient and infection has been ruled out, compression therapy is the mainstay of treatment for venous ulcers. Recommendations for compression therapy:
1) ABI: 0.8 to 1.0 full compression:
a) Profore.
b) Apply calcium alginate to ulcer, then wrap with an Unna Boot and cover with a Coban wrap: Change in 3 days; if tolerating well, then change weekly.
2) ABI: 0.6 to 0.8 light compression:
a) Profore Lite.
b) Apply calcium alginate to ulcer, then wrap with an Unna Boot and cover with a Coban wrap: Change in 3 days; if tolerating well, then change weekly.
2. Diabetic foot ulcer:
a. Establish arterial flow.
i. Refer to a vascular surgeon if deficiency is found.
b. For signs and symptoms of infection, use a sterile culturette to obtain a tissue culture and sensitivity first to assess what organism is present and to determine sensitivities. Treat per pharmaceutical recommendations. Treat with silver alginate to the site for moderate drainage and silver gel to the site for scant drainage.
c. Initiate offloading to site.
i. Refer to an orthotist for assessment if devices are required.
d. Treatment options:
i. To debride, cleanse with normal saline (NS), apply Santyl, and change dressing daily and as needed.
ii. To granulate an ulcer with scant drainage, cleanse with NS, apply a collagen dressing and cover with a secondary dressing, and change daily as needed.
iii. To granulate an ulcer with moderate drainage, cleanse with NS, apply calcium alginate, and change dressing daily and as needed.

B. Client teaching: *See Client Teaching Guide for this chapter, "Wound Care: Lower Extremity Ulcers."*
C. Pharmaceutical therapy:
1. If culture and sensitivity is performed, antibiotics may be used as recommended per sensitivity.

FOLLOW-UP

A. Follow up in 1 to 2 weeks to evaluate therapy.
B. See the client every 1 to 2 weeks until healing well; then may reduce to a 2- to 4-week evaluation until complete closure.

CONSULTATION/REFERRAL

A. Consult or refer the client to a wound care specialist for the following.
1. Client has extensive ulcer that you are not comfortable with:
a. Visible bone, muscle, or tendon.
2. Client has multiple medical comorbidities (especially diabetes).
3. Client is not responding to treatment of 2 to 4 weeks.
4. Ulcer is showing decline on follow-up visit.
5. Infection is present.

INDIVIDUAL CONSIDERATIONS

A. Adults:
1. Ischemic ulcers warrant immediate referral.
2. Complaints of severe pain, lack of pulse, cool digit, or new onset of purplish/bluish discolorations to the feet require immediate workup for arterial clot to lower extremity.

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WOUND CARE: PRESSURE INJURIES/ULCERS

DEFINITION

A. "A pressure injury is localized damage to the skin and underlying soft tissue usually over a bony prominence or related to a medical or other device. The injury can present as intact skin or an open ulcer and may be painful. The injury occurs as a result of intense and/or prolonged pressure or pressure in combination with shear. The tolerance of soft tissue for pressure and shear may also be affected by microclimate, nutrition, perfusion, co-morbidities and condition of the soft tissue" (National Pressure Ulcer Advisory Panel, 2016).

INCIDENCE

A. Acute care: 0.4% to 38%.
B. Long-term care: 2.2% to 23.9%.
C. Home care: 0% to 17%.

PATHOGENESIS

A. Pressure ulcers occur when an area of tissue remains in surface contact for a period of time. This contact causes occlusion of microvascular vessels, which leads to tissue hypoxia and eventually may cause ischemia. Over time, a pressure ulcer is developed. The amount of time this takes is client-dependent and can be altered by physical and/or environmental factors.

PREDISPOSING FACTORS

A. Acute illness.
B. Fecal/urinary incontinence.

C. Malnutrition.
D. Weight loss.
E. Failure or inability to offload:
  1. Fracture, elevation of head of bed (HOB), lack of education, or noncompliance.
F. Chronic medical conditions.
G. Advanced age.
H. Medical devices.

COMMON COMPLAINTS

A. Pain.
B. Bleeding.
C. Drainage.

SUBJECTIVE DATA

A. Ask the client to describe the location and onset. What did they think may have caused the ulcer in the area? Was the onset sudden or gradual? How have the symptoms continued to develop?
B. Assess if the area is itchy or painful.
C. Assess for any associated drainage. Ask about the color and if any odor is noted.
D. Complete a drug history. Ask the client if they are taking any steroids or anticoagulants.
E. Has the client been treated for this location before? If so, describe treatments.
F. Determine whether the client has attempted to treat this problem at home. If yes, ask with what.
G. Rule out any possible exposure to industrial or domestic toxins or insect bites.
H. Assess for iodine and sulfa allergies before starting treatment.

PHYSICAL EXAMINATION

A. Check temperature, pulse, pulse ox, respirations, and blood pressure:
B. Inspect:
  1. Inspect the pressure ulcer/injury.
    a. Measure length × width × depth.
      i. Undermining: Measure and note location, using the face of a clock to document site of undermining: for example, 12 o'clock, 3 o'clock, 6 o'clock, or 9 o'clock.
      ii. Tunneling: Measure and note location, using the face of a clock to document site of tunneling: for example, 12 o'clock, 3 o'clock, 6 o'clock, or 9 o'clock.
    b. Describe the wound bed.
      i. Tissue in the wound bed:
        1) Necrotic tissue, slough tissue, granulation tissue, epithelial tissue (percentage of each should together equal 100%).
      ii. Drainage:
        1) Amount:
          a) None, scant, moderate, copious.
        2) Color:
          a) Serous, purulent, yellow, serosanguineous, or green.
      iii. Odor:
        1) Odor present: yes or no.
      iv. Periwound:
        1) Intact.
        2) Not intact.
          a) Erythema, fever, induration, maceration, excoriation, calloused, epiboly.

DIAGNOSTIC TESTS

A. Complete blood count (CBC).
B. Wound culture and sensitivity.
C. Wound biopsy.
D. X-ray.
E. MRI.
F. Bone scan.

DIAGNOSIS

A. Staging pressure injuries/ulcers:
  1. Deep-tissue pressure injury (DTPI):
    a. Intact or nonintact skin with persistent nonblanchable discoloration (purple, deep red, maroon) or epidermal separation revealing a blood-filled blister or darkened wound bed.
    b. Changes in pain/temperature may precede changes in skin color. Discoloration may be difficult to detect in highly pigmented skin.
    c. DTPI may be due to intense pressure and shear forces at the bone–muscle interface.
    d. The wound may resolve without tissue loss, or rapid evolution may occur to reveal extent of damage.
    e. If fascia, muscle, necrotic/subcutaneous/granulation tissue, or other underlying structures are visible, this is likely a full-thickness pressure injury (stage 3, 4, or unstageable).
    f. Do not use DTPI staging to describe neuropathic, vascular, dermatologic, or traumatic conditions.
  2. Stage 1 pressure injury:
    a. Intact skin with nonblanchable, localized erythema. Discoloration may be difficult to detect in highly pigmented skin.
    b. Changes in sensation, temperature, or firmness may precede visual changes.
    c. Do not use stage 1 to describe purple, deep red, or maroon discoloration; these may indicate DTPI.
  3. Stage 2 Pressure Injury:
    a. Partial-thickness loss of skin with exposed dermis.
    b. The wound is moist, pink or red, viable, and may present as an intact or ruptured serum-filled blister.
    c. Deeper tissues and adipose (fat) are not visible.
    d. Eschar, slough, and granulation tissue are not present.
    e. Stage 2 pressure injuries are the result of adverse microclimate or shear forces.
    f. Do not use stage 2 to describe the following:
      i. Skin tears, burns, or abrasions.
      ii. Moisture-associated skin damage (MASD), including:
        1) Incontinence-associated dermatitis (IAD).
        2) Intertriginous dermatitis (ITD).
        3) Medical adhesive-related skin injury (MARSI).
  4. Stage 3 pressure injury:
    a. Full-thickness loss of skin, with visible adipose.
    b. Granulation tissue, slough, eschar, and epibole (rolled wound edges) may be present.
    c. The extent of tissue damage varies by anatomic location; deep wounds may develop in areas of significant adiposity.
    d. Undermining/tunneling may occur.
    e. Muscle, tendon, ligament, cartilage, fascia, and/or bone are not exposed.

▶

f. Do not use stage 3 if eschar or slough obscures the depth of tissue loss; this indicates an unstageable pressure injury.

5. Stage 4 pressure injury:
a. Full-thickness loss of skin and tissue.
b. Muscle, tendon, ligament, cartilage, fascia, and/or bone are exposed or directly palpable.
c. Epibole (rolled wound edges), undermining, and/or tunneling often occur.
d. The extent of tissue damage varies by anatomic location.
e. Eschar or slough may be present. If either or both obscure the depth of tissue loss, this indicates an unstageable pressure injury.

6. Unstageable pressure injury:
a. Full-thickness loss of skin and tissue.
b. Eschar or slough obscures the extent of tissue loss; removing these will reveal a stage 3 or 4 pressure injury.
c. Do not soften or remove stable eschar (dry, adherent, intact without erythema or fluctuance) on the heel or ischemic limb unless you have established client has adequate perfusion to ensure potential for wound healing.

7. Medical device-related pressure injury:
a. This is the result of using devices intended for diagnostic or therapeutic purposes.
b. The pressure injury generally conforms to the shape or pattern of the device.
c. The injury should be staged using the staging system.

8. Mucosal Membrane Pressure Injury:
a. Found on mucous membranes with a history of medical device use at the injury's location.
b. These ulcers cannot be staged due to the anatomy of the tissue.

Source: Adapted from National Pressure Ulcer Advisory Panel (NPUAP). (2016). NPUAP pressure injury stages. www.npuap.org/resources/educational-and-clinical-resources/npuap-pressure-injury-stages/.

DIFFERENTIAL DIAGNOSES

A. Abscess.
B. Trauma.
C. Skin cancer.
D. Vascular ulcer.
E. Diabetic foot ulcers.
F. Dermatologic disorder.

PLAN

A. General interventions:
1. Identify the cause of pressure and alleviate.
2. To debride, cleanse with normal saline (NS), apply Santyl ointment, and change dressing daily and as needed throughout.
3. To granulate an ulcer with scant drainage, cleanse with NS, apply collagen, and change dressing daily as needed.
4. To granulate an ulcer with moderate drainage, cleanse with NS, apply calcium alginate, and change dressing daily as needed.

FOLLOW-UP

A. Follow up in 1 to 2 weeks to evaluate therapy.
B. See the client every 1 to 2 weeks until healing well; then may reduce to a 2- to 4-week evaluation until complete closure.

CONSULTATION/REFERRAL

A. Consult or refer the client to a wound care specialist for the following:
1. Extensive ulcer that you are not comfortable with.
2. Client with multiple medical comorbidities (especially diabetes).
3. Client not responding to treatment of 2 to 4 weeks.
4. Ulcer showing decline on follow-up visit.
5. Infection is present.

INDIVIDUAL CONSIDERATIONS

A. Adults:
1. Clients at end of life may develop pressure ulcers related to the dying process. This type of ulcer is referred to as a Kennedy terminal ulcer. These clients are treated for comfort.

B. Geriatrics:
1. Research suggests that pressure ulcers in the older adult population (especially those suffering from dementia) appear to accelerate health deterioration, which leads to lower survival and higher mortality rates. Educating the client and surrogates regarding good nutrition, fall prevention, skin care, if bed-bound or wheelchair-bound frequent position change, and meticulous wound care techniques could help reduce pressure ulcer complications and increase survival time.

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WOUNDS OF THE SKIN

DEFINITION

A. Wounds are breaks in the external surface of the body.

PATHOGENESIS

A. Wounds can be caused by any one of innumerable objects that breach the skin. Lacerations and abrasions typically heal by a four-stage process of clotting, inflammation, skin cell proliferation, and remodeling. The most common pathogens of wound infections are *Staphylococcus aureus* and beta-hemolytic streptococcus.

PREDISPOSING FACTORS

A. Exposure to accidental or intentional injury.
B. Accident prevention failure.
C. High-risk behaviors.
D. Conditions that predispose to poor wound healing:
1. Diabetes.
2. Corticosteroid therapy.
3. Immunodeficiency.
4. Advanced age.
5. Undernourishment.
6. Comorbidities.

COMMON COMPLAINTS

A. Bleeding.

B. Pain.
C. "Cut" in the skin integrity.
D. Drainage.

OTHER SIGNS AND SYMPTOMS

A. Signs and symptoms of infection: Deep wounds and dirty wounds have increased risk of infection.
B. Soft tissue damage: Wounds with tissue necrosis have increased risk of infection.

SUBJECTIVE DATA

A. Elicit the client's description of how the wound occurred, including where and when the injury was sustained.
B. Ascertain how much time has elapsed until treatment. If 6 hours have elapsed, bacterial multiplication is likely.
C. Ask if the client is currently immunized for tetanus.
D. Complete a drug history; include any allergies to medications, anesthetics, or dressings.
E. Ask if the client is taking any medications, especially steroids or anticoagulants.
F. Assess iodine and sulfa drug allergies before starting treatment.
G. Review with the client whether anything significant in the medical history may interfere with the healing process (e.g., immunodeficiency).

PHYSICAL EXAMINATION

A. Check temperature, pulse, pulse ox, respirations, and blood pressure.
B. Inspect:
1. Inspect wound.
2. Measure wound for length × width × depth. Wounds with untidy edges may heal more slowly and with disfigurement.
3. Assess underlying bony structures.
4. Inspect for foreign objects.
C. Palpate:
1. Palpate extremities for neurovascular function and sensation.
2. Palpate tissue distal to wound.
3. Palpate lymph nodes surrounding injured area.
D. Perform neurologic exam:
1. Assess motor function distal to wound.

DIAGNOSTIC TESTS

A. Culture wound site if suspicious of infection.
B. Take x-ray films for deep or crushing wounds.

DIFFERENTIAL DIAGNOSES

A. Wound, minor.
B. Accidental self-inflicted injury.
C. Self-inflicted injury.
D. Domestic violence.
E. Bite.

PLAN

A. General interventions:
1. Wounds that require open-wound management:
a. Abrasions and superficial lacerations.
b. Wounds with great amount of tissue damage.
c. Wounds more than 6 hours old.
d. Contaminated wounds.
e. Large area of superficial skin denudation.
f. Puncture wounds.
2. For wounds that do not require sutures:
a. Cleanse the wound well with warm water and soap; remove all dirt and foreign bodies.
b. Forceful irrigation may be needed; use finepore sponge (Optipore) with a surfactant, such as poloxamer 188 (Skin-Clens). Approximate wound edges if possible, cover with xeroform and nonadherent dressing change Q72H and as needed.
c. If inflammation is present, soak and wash for 15 to 20 minutes three to four times per day. Cover with clean, dry dressing. **Do not use Steri-Strips.**
3. For wounds that require sutures:
a. Clean with warm water and soap. Irrigate with sterile saline solution.
b. Anesthetize with 1% to 2% lidocaine (Xylocaine). Do not use solution with epinephrine at fingertips, nose, or ears. Probe wound for any remaining foreign bodies. Approximate wound edges.
c. Suture the following with technique appropriate to the site:
i. Skin sutures: nonabsorbable material (e.g., nylon, prolene, silk).
ii. Subcutaneous and mucosal sutures: absorbable material (e.g., Dexon, Vicryl, or plain or chromic gut).
iii. Extremities: 4-0 nylon.
iv. Soles of feet: 2-0 nylon.
d. Cover with clean, dry dressing; change after first 24 hours.
e. Suture removal is based on location:
i. Head and trunk: 5 to 7 days.
ii. Extremities: 7 to 10 days.
iii. Soles and palms: 7 to 10 days.
f. Tetanus prophylaxis.
B. Client teaching: *See Client Teaching Guide for this chapter, "Wound Care: Pressure Injuries/Ulcers."*
C. Pharmaceutical therapy:
1. Control pain with acetaminophen (Tylenol) or ibuprofen, as needed.
2. Topical antibiotic ointments include bacitracin and neomycin.
3. Clients with severe wound contamination, involvement of structures, immunocompromised, or vascular insufficiency would be given oral antibiotics for prophylaxis:
a. Amoxicillin, clavulanate acid (Augmentin) 875 mg orally BID for 7 to 10 days.
b. With penicillin allergy, use erythromycin; 500 mg orally BID for 7 to 10 days.
4. Other alternatives include cephalexin (Keflex), cefadroxil (Duricef), ciprofloxacin.
5. Tetanus toxoid 0.5 mL by intramuscular (IM) injection in deltoid, if no booster has been administered in the past 5 years.
6. Wounds diagnosed with methicillin-resistant *S. aureus* (MRSA) should be treated with the following oral antibiotics:
a. Trimethoprim-sulfamethoxazole (Bactrim).
b. Minocycline or doxycycline.
c. Clindamycin rifampin (should be used in combination with one of the previous antibiotics).
d. Linezolid.
7. Antibiotics not recommended due to high resistance include the following:
a. Beta-lactams.

TABLE 7.2 RECOMMENDED PROTEIN NEEDS PER DAY FOR WOUND HEALING

Older adults: baseline protein needs	1.0–1.2 g/kg body weight/d
Healthy adults	0.8–1.0 g/kg body weight/d
Large burns	1.5–3.0 g/kg body weight/d (varies by extent of the burn)
Post surgery	1.0–1.5 g/kg body weight/d (varies by extent of the surgery)
Pressure ulcers/injuries	1.0–2.0 g/kg body weight/d

b. Fluoroquinolones.
c. Dicloxacillin.
d. Cephalexin.

8. Treating the nares with Bactroban Ointment BID and having the client use Hibiclens soap when showering will help prevent recurrent infections.

9. For severe cases of infection, the client requires hospitalization for aggressive antibiotic treatment.

FOLLOW-UP

A. Have the client return for evaluation and dressing change in 24 to 48 hours.

CONSULTATION/REFERRAL

A. Refer the client to a physician for the following wounds:

1. Facial wounds.
2. Subcutaneous tissue penetration.
3. Functional disturbance of tendons, ligaments, vessels, or nerves.
4. Grossly contaminated wounds.
5. Wounds requiring hospitalization or aggressive antimicrobial therapy for evidence of pyogenic abscess, cellulitis, and ascending lymphangitis.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. Adults with compromised immune systems and chronic conditions are at greater risk of developing a secondary bacterial infection.

B. Geriatrics:

1. Avoid use of nonsteroidal anti-inflammatory drugs (NSAIDs) in clients who have chronic kidney disease stage IV or less (creatinine clearance <30 mL/min).
2. Medications commonly used in the older adult population are associated with poor wound healing: cytotoxic antineoplastic agents, corticosteroids, aspirin, NSAIDs, anticoagulants, bisphosphonates, some antibiotics, anticonvulsants, angiogenesis inhibitors, and pain medications that contain morphine.

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WOUND HEALING: HIGH-PROTEIN NUTRITION

A. Nutrition is an essential component in the healing of any wounds. Protein, carbohydrates, fats, vitamins, minerals, and amino acid needs are increased for tissue repair. Table 7.2 summarizes the recommended amount of protein intake needed under different circumstances for tissue repair.

1. Consult with a dietitian for medical nutrition therapy (MNT).
2. Evaluate protein intake.
3. Evaluate the client's weight.
a. Perform serial weights: Use the same scales.
b. Use diet diary or 24-hour recall.
c. The dietary and weight history is used to determine additional needs.
d. Excessive protein potentially leads to dehydration.
4. Perform lab tests.
a. Complete blood count (CBC).
b. Basic metabolic panel (BMP).
c. Protein: albumin, total protein, and prealbumin.
d. Renal: blood urea nitrogen (BUN) and creatinine.
5. Evaluate wound stage/healing.
6. Supplemental protein may be required.
a. Sources of protein:
i. **Whole eggs are considered the "gold standard" protein.**
ii. Meat: red and white meat.
iii. Fish.
iv. Liver.
v. Dairy: milk, cheese, yogurt.
vi. Soybeans.
vii. Legumes.
viii. Seeds.
ix. Nuts.
x. Grains.
b. Encourage meals and snacking, double portions of meat and eggs, commercial supplements, and fluids.
i. Eat five to six smaller meals throughout the day.
ii. Keep ready-to-eat meals and snacks.
iii. Drink shake-type beverages that contain protein, vitamins, and minerals.
iv. Add protein powder to drinks.

▶

c. Consider appetite stimulants.
d. Enteral nutrition should be considered if oral food and fluid intake is not adequate.

OTHER CONSIDERATIONS

A. Older adults have multiple risk factors for intake of insufficient protein for wound healing.
1. Financial:
a. Expense of high-protein foods.
b. Expense of oral supplemental nutrition drinks.
2. Difficulty chewing:
a. Dental problems.
b. Loose-fitting dentures.
c. Edentulous.
3. Assistive needs:
a. Inability to prepare foods.
b. Need to have a caregiver feed older adults.
c. Given inadequate time to consume food if being fed.
4. Dysphagia.
5. Anorexia.
6. Fatigue.
7. Food sensitivities and allergies.
8. Psychosocial:
a. Depression.
b. Loneliness.
B. Consultation with a dietitian for MNT, wound care specialist, social worker, occupational therapist, and behavioral health evaluations is advised.

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XEROSIS (WINTER ITCH)

DEFINITION

A. Xerosis, often called "winter itch," is dry skin.

INCIDENCE

A. Xerosis occurs in 48% to 98% of clients with atopic dermatitis.
B. It occurs more frequently in older adult clients.

PATHOGENESIS

A. Dry skin may fissure, appear shiny and cracked, and leave subsequent inflammatory changes.

PREDISPOSING FACTORS

A. Frequent bathing with hot water and harsh soaps.
B. Cold air.
C. Low humidity.
D. Central heating or cooling.
E. Alcohol use.
F. Poor nutrition.
G. Cholesterol-lowering drugs.
H. Systemic disease manifested by thyroid, renal, or hepatic disease, anemia, diabetes, or malignancy.

COMMON COMPLAINTS

A. Dry, rough skin, especially on legs.

OTHER SIGNS AND SYMPTOMS

A. Pruritic, scaling skin, particularly on legs, with cracks and/or fissures.
B. Pruritus may be associated with systemic disorders or other infections. **Itching of scabies is particularly intense at night**.
C. Plaques 2 to 5 cm in diameter.
D. Erythema.
E. Wheal-and-flare response typical of urticaria.

SUBJECTIVE DATA

A. Obtain the client's description of the onset of symptoms and whether it was sudden or gradual.
B. Ask the client to identify any discomfort. Ask if the skin is itchy or painful.
C. Assess lesions for any associated discharge (blood or pus).
D. Determine whether the client has recently ingested any new medicines (antibiotics, cholesterol-lowering medications, or other drugs), alcohol, or new foods.
E. Ask the client about use of any topical medications.
F. Identify any preceding systemic symptoms (fever, sore throat, anorexia, or vaginal discharge).
G. Ask the client about bathing in hot water and if they are bathing regularly.
H. Review the client's full medication history for comorbid conditions.

PHYSICAL EXAMINATION

A. Inspect:
1. Inspect the skin for lesions, noting texture of skin.
B. Palpate:
1. Palpate abdomen for masses and hepatosplenomegaly.
2. Palpate lymph nodes.

DIAGNOSTIC TESTS

A. There are no diagnostic tests for xerosis.

DIFFERENTIAL DIAGNOSES

A. Xerosis.
B. Scabies.
C. Atopic dermatitis.

PLAN

A. General interventions:
1. Hydration and lubrication of skin.
2. Assess for and treat secondary infection.
B. Client teaching: *See Client Teaching Guide for this chapter, "Xerosis (Winter Itch)."*
1. Avoid alkaline soaps: Use Dove, Basis, mild soap, or soap substitutes such as Cetaphil or Aquanil.
C. Pharmaceutical therapy:
1. Apply emollient cream or lotion (Sarna, Lac-Hydrin, or Eucerin).
2. Use over-the-counter (OTC) skin lubricants (petroleum jelly, mineral oil, or cold cream).
3. Topical corticosteroid:
a. Triamcinolone 0.025% two to four times daily or 0.1% two to three times daily; apply sparingly. Advise not to use steroid treatments any longer than 2 weeks without notifying the primary provider.
b. Hydrocortisone 1% or 2.5% two to four times daily for no longer than 2 weeks. Recommend applying a thin film and avoiding the face. ▶

4. Systemic antihistamine to control pruritus, such as diphenhydramine (Benadryl) 25 to 50 mg or hydroxyzine 25 mg every 4 to 6 hours as needed.

FOLLOW-UP

A. Follow up as indicated until resolved.

CONSULTATION/REFERRAL

A. Consult or refer the client to a dermatologist if no improvement is seen.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Monitor the client for possible skin breakdown and/or ulceration.

2. Xerosis is common among older adult population. Several nonpharmacologic treatments to consider before using topical hydrocortisone would include increasing home environment humidity, avoiding excessive bathing and bath oils, encouraging oatmeal baths, and using emollient ointment (e.g., Aquaphor or Eucerin) and ceramide lotions (e.g., AmLactin) to smooth cracked rough skin and reduce pruritus.

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CLIENT TEACHING GUIDE

ACNE ROSACEA

PROBLEM Acne rosacea is a skin condition that affects primarily the nose and face, causing redness, flushing, pimples, and bumps. The blood vessels may be more prominent on the face, causing the skin to appear reddened.

CAUSE The cause is not known but is thought to be due to the blood vessels in the face being too active, causing flushing and redness of the skin.

PREVENTION/CARE

A. Avoid rubbing or massaging the face, as it can irritate the skin.
B. Avoid alcoholic beverages.
C. Avoid using harsh soaps/creams on the face, including cosmetics that irritate the skin.
D. Wash face with a mild soap daily, such as Cetaphil or Purpose soap. Other cleansers suggested are sulfa-based cleansers (Rosanil) or benzoyl peroxide cleansers daily.
E. Protect the skin when outdoors, wearing protective clothing, as well as hats and others to shield the face. Use sunscreen with a sun protection factor (SPF) 30 or a zinc-based ointment, such as zinc oxide, on the skin for protection.
F. You may be prescribed an antibiotic by mouth or an antibiotic cream/gel to place on the skin. Use medications as prescribed by your provider.
G. Avoid using steroid creams on your face unless prescribed by your provider.
H. If your skin condition begins to affect your eyes, you need to notify your provider immediately. Do not apply any medications or creams to your eyes that have not been prescribed by your provider.

TREATMENT PLAN

A. Use antibiotics/medications as prescribed by your provider.
B. Wash face with mild cleanser daily.

Activity: As tolerated. No limitations in physical activity.

Diet:

A. Drink plenty of fluids daily.
B. Avoid alcoholic beverages.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:

A. You have a reaction to any of the medications or cleansers prescribed.
B. Rash appears on your eyes or other new places.
C. Symptoms worsen or new signs or symptoms present before your next follow-up appointment.
D. Other: _____

Phone: _____

RESOURCE www.rosacea.org

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ACNE VULGARIS

PROBLEM Acne vulgaris refers to blackheads, whiteheads, or red nodules noted on the face, back, chest, and arms.

CAUSE Accumulation of cells and bacteria clogs the pores and stimulates an inflammatory response, resulting in papule or pustule (pimple or blackhead) formation.

PREVENTION/CARE

A. Wash area with mild soap (Purpose or Basis soap) no more than two times per day.
B. Avoid oil-based makeup and creams. Use matte-finish makeup or pore minimizer.
C. Use facial cleansers and moisturizers such as Cetaphil and Moisturel. These prevent the skin from drying out. Benzoyl peroxide 5% lotion or gel may be used at bedtime to help open pores and kill bacteria.
D. To prevent scarring, do not pick the lesions.
E. Avoid excessive sun exposure. Use oil-free sunscreen with a sun protection factor (SPF) 15 or greater.
F. Do not become frustrated if lesions return. Do not stop medications without the direction of your provider.
G. Stress can influence outbreaks of lesions. Practice exercise programs, stress management tactics, and other measures that reduce stress levels in your daily routine.
H. For mask use, wash or replace frequently.

TREATMENT PLAN

Activity: As tolerated. Physical activity encouraged.

Diet: Eat a well-balanced diet. Drink 8 to 10 glasses of water a day to help keep your skin well hydrated. Cocoa and chocolate do not have an effect on the development of acne vulgaris.

Medications: Antibiotics may need to be prescribed.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. You have a reaction to any of the medications prescribed.
B. You are unable to tolerate the prescribed medications.

Phone: _____

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DERMATITIS

PROBLEM Inflammation of the skin that occurs from contact with an irritant substance (poison ivy, soaps, etc.).

CAUSE Skin contact with irritating agent.

PREVENTION/CARE

A. Avoid aggravating agents.
B. Learn to recognize all plants (poison ivy, poison oak, etc.).
C. Flare-ups are common.
D. Avoid all known stimuli (poison ivy, soaps, etc.).
E. Do not wear tight, restrictive clothing.
F. When around irritating substances, wear gloves for protection.
G. For poison ivy, wash all clothes, shoes, pets, or other substances that may have come in contact with the poison ivy oil.

TREATMENT PLAN

Activity: As tolerated. Take cool baths as needed for itching. Oatmeal baths (Aveeno bath) help soothe the itching.

Diet: Regular diet.

Medications: Take Benadryl as needed for itching. Use calamine lotion as needed. Steroid creams may also be prescribed if the reaction is severe. Steroid dose packs may be needed if you are not getting better.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Worsening symptoms.
B. Sores on your face, eyes, or ears.
C. More redness, swelling, pain, or drainage.
D. Secondary bacterial infection.
E. Fever.
F. Other: _____

Phone: _____

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ECZEMA

PROBLEM Red, itching, scaling, and thickening of the skin occur in patches. You may have papules (bumps) with vesicles (clear fluid) that can be found especially on the hands, scalp, face, back of the neck, or skin creases of the elbows and knees.

CAUSE The cause is unknown. If it is an allergic reaction, it may be caused by foods such as eggs, wheat, milk, or seafood; wool clothing; skin lotions and ointments; soaps; detergents; cleansers; plants; tanning agents used for shoe leather; dyes; and topical medications. The risk of developing eczema increases with stress, medical history of other allergic conditions, clothing made of synthetic fabric (which traps perspiration), and weather extremes (cold, hot).

PREVENTION/CARE

A. Avoid risk factors.
B. Wear rubber gloves for household cleaning tasks.
C. Wear loose, cotton clothing to help absorb perspiration.
D. Keep fingernails short and wear soft gloves during sleep.
E. Scratching worsens eczema.
F. Bathe less frequently to avoid excessive skin dryness.
G. Use special nonfat soaps (Purpose or Basis soap) and tepid water.
H. Do not use soap on inflamed areas.
I. Lubricate the skin after bathing; avoid lubricants with alcohol in the ingredients.
J. Recommended creams include Eucerin, Keri Lotion, and Lubriderm. Steroid creams may be prescribed.
K. Avoid extreme temperature changes.
L. Avoid anything that has previously worsened the condition.

TREATMENT PLAN

Activity: No restrictions.

Diet: You may be told to try a special diet. Eliminate any foods known to cause flare-ups.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. You have a reaction to any of the medications prescribed.
B. You cannot take the medications.
C. New symptoms develop.

Phone: _____

RESOURCE National Eczema Association, 4460 Redwood Highway, Ste. 16-D, San Rafael, CA 94903, United States, Phone: 415-499-3474, www.nationaleczema.org

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ERYTHEMA MULTIFORME

PROBLEM An acute inflammatory disorder of the skin and mucous membranes, erythema multiforme is usually self-limited and benign.

A severe form is known as Stevens–Johnson syndrome or erythema multiforme major, and the less severe form is referred to as erythema multiforme minor.

CAUSE The cause is unknown in 50% of cases. Erythema multiforme has been associated with viral infections, particularly the herpes simplex virus; bacterial and protozoan infections; an immunologic reaction of the skin; medications (sulfonamides, penicillins, anticonvulsants, salicylates, barbiturates, nonsteroidal anti-inflammatory drugs [NSAIDs]), with reactions occurring up to 7 to 14 days after using the medication; pregnancy; premenstrual hormone changes; malignancy; or radiation therapy. Risk increases with history of erythema multiforme.

PREVENTION/CARE

A. Avoid suspected causes.
B. Seek prompt treatment of any illness or infection.
C. Prevent herpes simplex virus outbreaks by avoiding sun exposure and reducing stress.
D. Seek treatment immediately if at any time symptoms seem to be worsening or increasing.
E. Discontinue any implicated medication.
F. Apply wet dressings or soaks with Burow solution or apply lotions to soothe the skin.
G. Bathe in lukewarm to cool water three times a day for 30 minutes.
H. Monitor yourself for any eye involvement and report it to your healthcare provider immediately.
I. If mouth sores are present, use good oral hygiene (brush two to three times a day using a soft brush) and rinse frequently with cool water.
J. Hospitalization may be required if there is extensive skin involvement.

TREATMENT PLAN

Activity: As tolerated by the extent of the symptoms. Restrict yourself to bedrest if fever is present.

Diet: Usually no special diet is necessary, although if mouth sores are present a soft or liquid diet may be better tolerated. Increase fluid intake above the general 8 to 10 glasses per day.

Medications: May be prescribed to control symptoms and pain.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. You have an adverse reaction to or cannot tolerate any of the prescribed medications.
B. Symptoms worsen during treatment, or the rash does not clear in 3 weeks (usual course: rash evolves over 1–2 weeks, usually clears in 2–3 weeks, but may take 5–6 weeks).
C. New or unexplained symptoms develop.
D. You have any questions or concerns.

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FOLLICULITIS

PROBLEM A bacterial (or fungal) infection of the hair follicle. Folliculitis is seen when a pustule develops, commonly on the arms, legs, scalp, and face (beard).

CAUSE

A. Bacterial: infection commonly caused by *Staphylococcus* bacteria.
B. Fungal: may be caused by yeast infection.

PREVENTION/CARE

A. Keep skin clean and dry.
B. Avoid warm, moist conditions.
C. Healing generally occurs in 10 to 14 days after proper treatment with medications.
D. Practice good handwashing technique, using antibacterial soaps.
E. Use clean razors daily.
F. Throw old razors away.
G. Do not share razors.
H. Shampoo scalp daily.
I. Avoid use of hot tubs or heated swimming pools.
J. Folliculitis usually resolves within 4 to 6 weeks after proper treatment.

TREATMENT PLAN

Activity: As tolerated.

Diet: Regular diet.

Medications: Topical and/or oral antibiotics as prescribed.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:

A. You notice lesions worsening or spreading despite adequate medication treatment.
B. You have a fever higher than 101°F.
C. Your condition is not getting better.
D. You have a reaction to your medication.

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HERPES ZOSTER (SHINGLES)

PROBLEM Shingles is a reactivation of the viral infection common in childhood known as chickenpox. **The virus is contagious to those who have not had chickenpox.**

CAUSE Varicella zoster virus is stimulated and produces a blister-like rash, commonly seen on the chest and trunk area. The rash is commonly confined to one side of the body.

PREVENTION There are two vaccines available for prevention of shingles. The Centers for Disease Control and Prevention (CDC) recommends the recombinant zoster vaccine (RZV; Shingrix) as the preferred vaccine for anyone age 50 years and older; however, Zostavax is also still available if there is a shortage of the preferred vaccine. Shingrix is a two-dose vaccine, given 2 months apart. Zostavax is a one-time vaccine.

TREATMENT PLAN

A. Zostavax cannot be used to treat shingles breakouts or the painful sensations (postherpetic neuralgia) after you develop shingles.
B. The shingles rash usually lasts 2 to 3 weeks; however, symptoms may persist beyond this period.
C. The goal is to relieve the itching.
D. Apply warm soaks of Burow solution three times a day to lesions.
E. Notify family and friends of active virus. Advise anyone who has had contact with you that you have shingles, especially pregnant individuals and those who have never had chickenpox.

Activity: Avoid touching the shingles. Wash your hands. Your partners should not touch the area, especially when blisters are present. Use separate bath towels.

Diet: There is no special diet for shingles.

Medications: Oral and topical medications may be prescribed to soothe the itching. Take acetaminophen (Tylenol) as needed for comfort. Antiviral medications are available to help slow down the virus if started within 48 to 72 hours after the initial outbreak. You may be prescribed medications to help with the painful sensations (neuralgia).

CDC guidelines recommend all immunocompetent adults 50 years of age and older receive the Shingrix vaccine.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. Severe pain at lesion sites.
B. Any new symptoms relating to the shingles, such as excruciating pain, headaches, numbness, tingling sensation, or other symptoms.
C. Any questions regarding the shingles.
D. Any new facial involvement.

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INSECT BITES AND STINGS

PROBLEM Insect bites or stings cause skin changes and other reactions.

A. Seek immediate help if you or a family member have any symptoms of allergic reaction or anaphylaxis, either immediately after the bite or 8 to 12 hours after the bite.

B. You may need to call 911 or your local emergency response service.

C. If you have had a previous life-threatening allergic reaction, carry an anaphylaxis kit for emergency treatment.

1. *Local skin reactions* include red bumps on the skin that usually appear within minutes after the bite or sting but may not appear for 6 to 12 hours. Itching and discomfort may occur at the site.

2. *Systemic (body) reactions* include nausea or vomiting; headache, fever, dizziness, or lightheadedness; swelling; or convulsions.

3. *Allergic reactions* include itchy eyes, facial flushing, dry cough, wheezing, and chest or throat constriction or tightness.

CAUSE Bites or stings can be caused by mosquitoes, fleas, chiggers, bedbugs, ants, spiders, bees, scorpions, and other insects.

Risk increases with exposure to areas with heavy insect infestation, warm weather in spring and summer, lack of protective measures, use of perfumes or colognes, and previous sensitization.

PREVENTION/CARE

A. Institute first-aid measures and activate emergency services if severe, life-threatening reactions occur.

B. Avoid risk factors.

C. Wear protective clothing.

D. Use insect repellents with diethyltoluamide (DEET), avoiding the head, face, eyes, and mouth.

Specific Insect Care

A. All stings: Remove the stinger.

B. Bee, wasp, yellow jacket, or hornet stings: Rub a paste of meat tenderizer and water into the site.

C. Ant bites: Rub bite with ammonia and repeat as often as necessary.

D. Spider and scorpion bites: Capture the arachnid if possible and seek medical attention.

E. Mites: Apply a petroleum product (Vaseline) until the animal withdraws from the skin.

F. Ticks: You should also be given the *Client Teaching Guide for "Lyme Disease and Removal of a Tick."*

General Care For All Bites

A. Clean wound with soap and water.

B. Apply ice pack (no ice directly on skin; use towel or cloth to protect skin).

C. Elevate and rest the affected body part.

D. Immerse affected part or apply warm water soaks to site. However, if site itches, cool water feels best.

E. For minor discomfort, you may use nonprescription oral antihistamines (Benadryl) or topical steroid preparations (hydrocortisone cream).

F. Use only low-potency topical steroid products without fluorine on the face and groin area.

G. You may be prescribed more potent, prescription medications.

TREATMENT PLAN

Activity: No restrictions.

Diet: Eat a regular diet. Maintain adequate hydration with 8 to 10 glasses of water per day.

Medications: You may be prescribed an EpiPen to use for future major reactions. You need to keep this with you at all times.

You Have Been Prescribed: _____

(*continued*)

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CLIENT TEACHING GUIDE

INSECT BITES AND STINGS (*CONTINUED*)

You Need to Take: _____

You Need to Notify the Office If:

A. Self-care treatment does not relieve symptoms or if no improvement is noticed after 2 to 3 days.
B. A bitten area becomes red, swollen, warm, and tender to the touch. These symptoms indicate infection.
C. You have a temperature higher than or equal to 101°F.
D. You have a reaction or cannot tolerate any of the prescribed medications.

Phone: _____

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CLIENT TEACHING GUIDE

LICE (PEDICULOSIS)

PROBLEM A parasite called a louse has been found on your body or hair. Lice tend to live on the scalp, eyebrows, or genital area, or in warm moist areas of your skin. You may notice you have intense itching, swelling, or reddened areas of the skin, and sometimes even enlarged lymph glands.

CAUSE The lice bite the skin and cause intense itching. Lice and their eggs (called nits) may be difficult to see on the skin and shafts of hair. Lice look like small, 2- to 3-mm, tan-colored bugs. The eggs are tiny white eggs that stick to the hair shaft.

PREVENTION You can prevent repeated episodes of lice if you bathe daily; avoid crowded living conditions; change bed linens frequently; and do not share hats, combs, brushes, or other belongings. When family members have been in contact with others diagnosed with lice, check them closely for lice and treat as appropriate.

TREATMENT PLAN

A. Use medicated shampoo as directed.
B. Machine-wash all linens, stuffed animals, and any other items with which the lice may have come in contact.
C. Wash clothes in hot, soapy water.
D. Dry all linens in a hot dryer for at least 30 minutes.
E. Items that cannot be washed must be taken to the dry cleaner or wrapped and sealed in a plastic bag for 14 days.
F. Boil all hair accessories and clean well.
G. Do not share hats and combs.
H. Spray all furniture with appropriate products that kill all nits and lice.
I. Vacuum.

Activity: There is no activity restriction.

Diet: There is no special diet.

Medications:

A. Food and Drug Administration (FDA)-approved over-the-counter (OTC) products:
  1. Pyrethrins combined with piperonyl butoxide (Rid, Triple X, A-200, Pronto, R&C): approved for children 2 years and up. Avoid if allergic to ragweed or chrysanthemums.
  2. Permethrin lotion 1% (Nix): FDA-approved for children 2 months and older Repeat application on day 9 of initial dose.

You Have Been Prescribed to Use: _____

A. Treat as directed on the bottle.
B. After shampooing as directed, make sure to remove each single nit from each shaft of hair. Any nits left in the hair will hatch and start the cycle over again. Comb any dead or remaining live lice out of the hair with a fine-tooth comb.
C. Repeat on day 9 of initial dose.
D. Do not use a shampoo/conditioner or conditioner prior to use of the lice medications. Do not wash hair for 1 to 2 days after lice medication treatment.

You Need to Notify the Office If You Have:

A. Any questions regarding the removal of the nits and lice, or if you need any assistance. If other family members need to be evaluated, please let us know. If secondary infection occurs, call the office.
B. Precautions: Do not overuse medications or combine different head lice medications. Use only as directed. These medications are insecticides and can be dangerous if used incorrectly.

Phone: _____

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LICHEN PLANUS

PROBLEM A chronic skin eruption, lichen planus is not cancerous or contagious. It frequently appears as small, slightly raised, itchy, purplish bumps with a whitish surface. Sudden hair loss from the head may occur. Lichen planus may involve the skin of the legs, trunk, arms, wrists, scalp, or penis; the lining of the mouth or vagina; and the nail beds of the toenails and fingernails.

CAUSE The cause is unknown, but it may be caused by a virus. In a few cases, this may be an adverse reaction to certain drugs. The risk of developing lichen planus increases with stress, fatigue, or exposure to drugs or chemicals.

PREVENTION/CARE Currently, there are no known preventive measures.

A. The goal of treatment is to relieve symptoms.
B. Use cool-water soaks to relieve itching.
C. Reduce stress; this may help prevent recurrences. Learn relaxation techniques or obtain counseling if necessary.
D. Speak with your healthcare provider if you suspect a drug to be the cause.

TREATMENT PLAN

Activity: No restrictions.

Diet: Eat a well-balanced diet; drink 8 to 10 glasses of water every day.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:

A. You have a reaction to any of the prescribed medications.
B. You are unable to tolerate the prescribed medications.
C. Hair loss or nail destruction occurs.
D. New lesions appear as old lesions resolve.
E. Other: _____

Phone: _____

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PITYRIASIS ROSEA

PROBLEM Pityriasis rosea is a very common condition characterized by a rash, which may or may not itch. You may have noticed a large scaly patch prior to breaking out with the more generalized rash. **It is not known to be contagious and you do not need to isolate yourself.**

CAUSE The cause of pityriasis rosea is unknown.

PREVENTION Because the cause of pityriasis rosea is unknown, there are no recommended preventive measures.

TREATMENT PLAN Good hygiene and avoidance of scratching are recommended to prevent a secondary infection.

Activity: It is not necessary for you to limit your activity. Sunlight exposure to skin for short periods of time daily for 5 consecutive days will reduce itching and improve rash. Care should be taken in not burning skin with short-term exposure to the sun.

Diet: No changes are required in your diet.

Medications: You may be prescribed an antihistamine medication to take by mouth and topical steroid creams to apply to the rash itself. If the itching is severe, you may have oral steroids prescribed.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Any new symptoms.
B. Any reaction to your medication.

Phone: _____

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PSORIASIS

PROBLEM Psoriasis is a chronic, scaly, thickened skin disorder with frequent remissions and recurrences. The skin of the scalp, elbows, knees, chest, back, arms, legs, toenails, fingernails, and fold between the buttocks may be involved.

CAUSE The cause of psoriasis is unknown.

PREVENTION/CARE

A. There is no known prevention, but symptoms can be controlled.
B. Moving to a warmer climate might be beneficial. Severity increases with cold.
C. Maintain good skin hygiene with daily baths or showers.
D. Avoid harsh soaps.
E. Avoid skin injury, including harsh scrubbing, which can trigger new outbreaks.
F. Avoid skin dryness.
G. To reduce scaling, use nonprescription, waterless cleansers, as well as hair preparations containing coal tar (Zetar, T/Gel, Pentrax), emollients (Eucerin Plus lotion or cream, Lubriderm, Moisture Plus, Moisturel), or products containing cortisone (often prescription strength).
H. Expose the skin to moderate amounts of sunlight as often as possible. Avoid long periods in the sun to prevent sunburn.
I. Oatmeal baths may loosen scales. Use one cup of oatmeal to a tub of warm water.
J. Stress may increase outbreaks of psoriasis. Consider counseling to assist in lifestyle changes, coping, or any psychological problems caused by psoriasis.

TREATMENT PLAN

Activity: There are no activity restrictions.

Diet: Eat a well-balanced diet. You may be instructed to try a gluten-free diet. Drink 8 to 10 glasses of water per day. Avoid alcohol in your diet.

Medications: You may be prescribed the following types of medications:
A. Creams to rub on the skin:
    1. Ointments containing coal tar. These may stain clothing.
    2. Salicylic acid cream, anthralin cream, or vitamin D-like cream (calcipotriene).
    3. Topical cortisone creams may also be used for short periods of time.
B. Psoralen plus UV light (PUVA; combination of a medication and exposure to UVA light).
C. Combination of tar baths with UVB light.
D. Antihistamines to relieve itching.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. You have an adverse reaction to or cannot tolerate any of the prescribed medications.
B. Symptoms recur after treatment. Notify your healthcare provider if during an outbreak, pustules erupt on the skin and/or are accompanied by fever, muscle aches, and fatigue.
C. New, unexplained symptoms develop.
D. Other: _____

Phone: _____

For severe cases, you may be referred to a dermatologist (specialist for skin disorders).

RESOURCE National Psoriasis Foundation, Suite 200, 6415 SW Canyon Ct., Portland, OR 97221, United States, Phone: 800-723-9166, www.psoriasis.org

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SCABIES

PROBLEM Scabies is a common condition characterized by severe itching. You may have noticed small burrows between your fingers and in other locations, or you may have some redness and skin irritation aggravated by scratching.

CAUSE Scabies is caused by an infestation of the skin by a mite. You have contracted scabies by coming in close contact with an individual who has the condition.

PREVENTION You can prevent reinfection of scabies by following these measures:

A. Make sure all close contacts, sexual partners, family, and household contacts are treated.

B. All bedding and clothing that have touched infected skin should be machine-washed and machine-dried on the highest heat cycle.

C. Any clothing or bedding that cannot be laundered in the previously mentioned way should be placed in a plastic bag that is securely tied for at least a week. The mites cannot live this long away from human skin.

D. Coats, furniture, rugs, floors, and walls do not require any special cleaning or treatment.

TREATMENT PLAN

A. Most clients with scabies are successfully treated with only one overnight application of a cream known as scabicide.

B. You should let your practitioner know if you are pregnant or breastfeeding.

C. You may itch for up to a week even with successful treatment.

D. If you still have symptoms after 2 weeks, you should see your practitioner, who will determine if you need a second treatment.

Activity: Affected children in day care or school can return the day after treatment is completed.

Diet: There is no special diet.

Medications: The most common medications used to treat scabies include:

A. Permethrin (Elimite cream), which is applied to all body areas from the neck down and washed off in 8 to 14 hours.

B. You may be told to use Benadryl 25 to 50 mg if needed for itching.

C. Do not use it near your eyes.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:

A. You have new symptoms.

B. You have a reaction to the medication.

C. Other: _____

Phone: _____

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SEBORRHEIC DERMATITIS

PROBLEM Seborrheic dermatitis is a skin condition characterized by greasy or dry, white, flaking scales over reddish patches on the skin. The scales anchor to the hair shafts and may itch, but they are usually painless unless complicated by infection.

CAUSE The cause is unknown. The risk of seborrheic dermatitis increases with stress; hot and humid or cold and dry weather; infrequent shampoos; oily skin and other skin disorders such as rosacea, acne, or psoriasis; obesity; Parkinson disease; use of lotions that contain alcohol; and HIV/AIDS.

PREVENTION/CARE

A. There are no specific preventive measures.
B. The goal of care is to minimize the severity or frequency of symptoms.
1. Shampoo vigorously and as often as once a day. The type of shampoo is not as important as the way you scrub your scalp. To loosen scales, scrub with your fingernails while shampooing and scrub at least 5 minutes.
2. If you suffer from minor dandruff, you may use nonprescription dandruff shampoos with selenium sulfide (Selsun Blue, Exsel) or zinc pyrithione (Zincon), as well as lubricating skin lotion.
3. For severe problems, shampoos that contain coal tar or scalp creams that contain cortisone may be prescribed.
4. To apply medication to the scalp, part the hair a few strands at a time and rub the ointment or lotion vigorously into the scalp.
5. Topical steroids may be prescribed for other affected parts of the skin.
6. Be sure to dry skin folds thoroughly after bathing.
7. Wear loose, ventilating clothing. Avoid constant cap wearing.

TREATMENT PLAN

Activity: No restrictions. Outdoor activities in the summer may help alleviate symptoms.

Diet: Eat a well-balanced diet. Drink 8 to 10 glasses of water per day. Avoid foods that seem to worsen your condition.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. You have an adverse reaction to any of the prescribed medications.
B. You are unable to tolerate any of the medications.
C. You have any secondary infection in the affected area.
D. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

SKIN CARE ASSESSMENT

PROBLEM Skin cancer is the most common type of cancer. In 2006, the Centers for Disease Control and Prevention (CDC) noted that the rate of skin cancer varies by the state where you live. A state map can be accessed at at www.cdc.gov/cancer/skin/statistics/state.htm.

CAUSE Skin cancer is frequently caused by damage from the UV rays of the sun. Some people are at a higher risk of developing skin cancer. Risk factors include the following:

A. Fair complexion; blond/red hair; blue, green, or hazel eye color.
B. Advanced age with sun-damaged skin.
C. A history of a severe sunburn.
D. A history of spending long hours outdoors.
E. A history of x-ray procedures for skin conditions.
F. Genetic susceptibility.

PREVENTION/CARE

A. Protect yourself and prevent sun exposure to your skin by staying out of the harmful rays as much as possible, especially between the hours of 10 a.m. and 3 p.m. This time frame accounts for approximately 70% of the harmful UV radiation.
B. If you are exposed to the sun, wear a sunscreen product with a sun protection factor (SPF) of 15 or greater at all times.
C. Wear hats that screen your face and neck, as well as your ears.
D. Clothing is available with sun-protective materials. Regular long shirts and long pants also help protect your skin.
E. Sit in the shade to rest.
F. Do not use a tanning booth.

STEPS TO TAKE TO PREVENT YOURSELF FROM HAVING SKIN CANCER

A. Examine your skin monthly.
1. Use a good light source and a mirror to see areas of your skin not clearly visible.
2. Examine your entire body closely.
3. Pay particular attention to areas that are frequently exposed to the sun, especially your face, lips, eyes, neck, scalp, and ears.
4. Monthly screening allows you to familiarize yourself with birthmarks and moles. Note the size, shape, and color of these marks. Note any changes in these marks using the "ABCDE" method:
a. Asymmetry: The shape of the mark should be noted. Any change in shape or irregularity of the mark needs to be evaluated by your healthcare provider.
b. Border: Look carefully at the border of the mark. If the border edge is ragged, notched, and not smooth, your healthcare provider needs to evaluate it.
c. Color: Note the color of moles. If you notice any change in color, or if you notice the mole to have several colors (brown, black, tan, red, etc.), you need to alert your healthcare provider.
d. Diameter: Measure the size of the mark and document it. Any change in size, especially if it is greater than 6 mm, should be brought to the attention of your healthcare provider.
e. Elevation: Note elevation of the lesion, change in size, and any evolving changes of the lesion. You need to alert your healthcare provider if changes occur.
5. You should also evaluate lesions on your skin for any type of change. If the lesions begin bleeding or hurting, or change in texture or in any other way, your healthcare provider needs to evaluate the change.
6. Be alerted to any skin ulcers that do not heal within 1 month. Also, any new moles or lesions need to be evaluated by your healthcare provider for proper diagnosis of the type of lesion.

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SKIN CARE ASSESSMENT (*CONTINUED*)

TREATMENT PLAN

Activity: As tolerated, but protect yourself from sun exposure.

Diet: There is no special diet that will stop skin cancer.

Medications: There are no medications that prevent skin cancer.

You Need to Notify the Office If You Have:

A. Any of the skin changes mentioned here that need to be evaluated by your healthcare provider.

Phone: _____

RESOURCE American Cancer Society, 250 Williams Street NW, Atlanta, GA 30303, United States, Phone 800-227-2345, www.cancer.org

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CLIENT TEACHING GUIDE

TINEA

PROBLEM Tinea is a fungal infection of the skin that can be found on any part of the body. It is not uncommon to have more than one tinea fungal infection at a time:

A. Tinea pedis (athlete's foot).
B. Tinea cruris (jock itch).
C. Tinea capitis (ringworm on the head).
D. Tinea unguium (onychomycosis of the nails).
E. Tinea corporis (ringworm on the trunk or extremities).

CAUSE The fungus is transmitted by direct contact. It can be transmitted from objects, shoes, locker rooms, animals, and people.

PREVENTION/CARE

A. Use good hygiene, including not sharing hairbrushes and combs.
B. Keep skin cool and dry.
C. Wear shoes in locker rooms and pools.
D. Wear loose-fitting clothing.
E. Treat pets' skin problems adequately. Ringworm is often blamed on cats but it can come from almost any animal, including horses, rabbits, dogs, and pigs.
F. Infections of fingernails and toenails may require prescription medications.

TREATMENT PLAN

Activity: As tolerated. Some contact sports may increase getting tinea (football and wrestling).

Diet: There is no special diet.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. Your symptoms get worse.
B. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

TINEA VERSICOLOR

PROBLEM A yeast infection of the skin, tinea versicolor may cause color changes of the skin, commonly on the chest, back, shoulders, arms, and trunk. During the summer, these spots usually appear pale and do not tan. During the winter, the spots may appear pinker or darker than the normal skin color.

CAUSE Tinea versicolor is caused by an increased production of yeast on the skin, which is influenced by warm, moist conditions. It is common to have recurring episodes.

PREVENTION/CARE

A. Air-dry skin as much as possible.
B. Apply medication as directed.
C. Patches on skin (color changes) may take several weeks to be resolved.
D. Monthly treatments may help prevent recurrences.

TREATMENT PLAN

Activity: There are no activity restrictions with tinea versicolor.

Diet: There is no special diet.

Medications:
A. Apply Selsun Blue shampoo or other medication as directed.
B. Most medications may be washed off of skin 30 minutes after application.
C. Selsun Blue shampoo may be used daily on the affected skin for 2 weeks.
D. Keep shampoo out of the eyes and the genital area.
E. Leave it on skin for about 20 minutes and then rinse it off.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You:
A. Do not see improvement despite proper treatment.
B. Develop new symptoms.
C. Other: _____

Phone: _____

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WARTS

PROBLEM A wart is a raised, rough growth projecting from the skin, which can be contagious.

CAUSE Warts are caused by a viral infection that stimulates the cells of the skin to multiply rapidly, resulting in an outward growth.

PREVENTION/CARE

A. Wash hands well.
B. Avoid scratching or picking warts. Warts bleed easily.
C. Some warts go away spontaneously without any treatment after time.
D. Medications may be prescribed.

To Enhance Destruction

A. Soak wart in warm water 10 to 15 minutes a day.
B. After soaking, use an emery board to file wart down.
C. Apply over-the-counter (OTC) medication (Compound W) as prescribed to site.
D. Duct tape may be applied over wart.
E. Perform these steps every night until resolved.
F. Warts may reappear at the same spot or in other areas.
G. Cryotherapy or "freezing" is another treatment option. Discuss this with your healthcare provider.

TREATMENT PLAN

Activity: There are no activity restrictions for warts.

Diet: There are no special diets for warts.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:

A. You develop an infection at the site of the wart.
B. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

WOUND CARE: LOWER EXTREMITY ULCERS

PROBLEM You have an ulcer on the body that lies on the lower extremities.

CAUSE Causes include edema, trauma, ischemia, and venous insufficiency.

PREVENTION/CARE

A. Keep the area clean and free of foreign debris.
B. Dressing changes: _____
C. You may be prescribed antibiotics; if so, take all antibiotics until they are completely gone.

TREATMENT PLAN

Activity: Do not apply direct pressure to the site of the ulcer. You may be prescribed to elevate your lower extremities: _____

Diet: Eat a well-balanced diet. Increase protein intake.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. A reaction or cannot tolerate any of the prescribed medications.
B. A fever and a general ill feeling.
C. Any new or unexplained symptoms related to the ulcer:
    1. Increase in size.
    2. New odor.
    3. Increased drainage.
    4. Change in color of the drainage.
    5. Increased pain at the site.
D. Any questions or concerns.

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WOUND CARE: PRESSURE INJURIES/ULCERS

PROBLEM You have an ulcer on the body that lies over a bony surface.

CAUSE Causes include prolonged periods of pressure to the area of the ulcer, causing a breakdown of skin integrity.

PREVENTION/CARE

A. Keep the area clean and free of foreign debris.
B. You may be prescribed dressing changes:
  1. Remove dressing.
  2. Clean the ulcer with normal saline.
  3. Apply prescribed medication (see Medications section, as follows).
  4. Cover with dry dressing; change as ordered.
C. You may be prescribed antibiotics; if so, take all antibiotics until they are completely gone.

TREATMENT PLAN

Activity: Do not apply direct pressure to the site of the ulcer.

Diet: Eat a well-balanced diet. Drink 8 to 10 glasses of water per day. Increase protein intake.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. A reaction or cannot tolerate any of the prescribed medications.
B. A fever and a general ill feeling.
C. Any new or unexplained symptoms:
  1. Increase in size.
  2. New odor.
  3. Increased drainage.
  4. Change in color of the drainage.
  5. Increased pain at the site.
D. Any questions or concerns.

Phone: _____

RESOURCE National Pressure Injury Advisory Panel, 4 Lan Drive Suite 310, Westford, MA 01886, United States, Phone: 978-364-5130, www.npiap.org

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WOUNDS OF THE SKIN

PROBLEM A wound is a break in the external surface of the body.

CAUSE Wounds are often caused by an accidental or intentional injury. Wound infection is usually caused by bacterial contamination of the site.

PREVENTION/CARE

A. Prevent accidental or intentional injury.
B. Immediately after injury, cleanse the wound well with soap and water.
C. Remove all dirt and foreign material.
D. You may be prescribed antibiotics; if so, take all antibiotics until they are completely gone.
E. You may need a tetanus shot.

TREATMENT PLAN

Activity: No restrictions. If infection is present, you may need to increase rest.

Diet: Eat a well-balanced diet. Drink 8 to 10 glasses of water per day.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. A reaction or cannot tolerate any of the prescribed medications.
B. A fever and a general ill feeling.
C. A wound/infection that seems to worsen.
D. Any new or unexplained symptoms.
E. Any questions or concerns.

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XEROSIS (WINTER ITCH)

PROBLEM Xerosis is severely chapped skin that becomes cracked, fissured, and inflamed. It can appear anywhere on the body, but it is seen most commonly on the legs.

CAUSE Xerosis is caused by insufficient oil on the skin's surface, which allows water to evaporate through the skin. Oil in the skin decreases with aging, excessive bathing, and excessive rubbing of the skin. An environment with low humidity also promotes dryness of the skin.

PREVENTION/CARE

A. Reduce water loss from the skin.
   1. Reduce the frequency and duration of baths or showers; use tepid water.
   2. Use soap sparingly.
   3. Avoid detergent soaps.
   4. Pat skin dry rather than rubbing.
   5. Apply skin lubricants (Lac-Hydrin, Eucerin, etc.) to dry skin before chapped areas become inflamed.
   6. Use ultrasonic, cool-mist humidifiers if the air is very dry.
   7. Clean the humidifier daily.
   8. Oil (such as Nivea) in the bathwater may be helpful.
   9. Apply lubricants after bathing when possible to trap additional moisture before evaporation occurs.

B. Apply hand cream four to eight times a day to hands and twice daily on the trunk and extremities.

TREATMENT PLAN

Activity: No restrictions. Avoid long-term exposure to drying environments.

Diet: Eat a well-balanced diet; drink 8 to 10 glasses of water per day.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Severely chapped skin, and self-care does not relieve the symptoms in 1 week.
B. Chapped skin that becomes inflamed or if you see any oozing.
C. Any questions or concerns.

Phone: _____

RESOURCE American Academy of Dermatology, 9500 W Bryn Mawr Avenue, Ste 500, Rosemont, IL 60018-5216, United States, Phone: (888) 462-DERM (3376), www.aad.org

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CHAPTER 8

EYE GUIDELINES

Jill C. Cash and Nancy Pesta Walsh

BLEPHARITIS

DEFINITION

A. Blepharitis is dryness and flaking of the eyelashes, resulting from an inflammatory response of the eyelid. It is considered a chronic condition exhibiting intermittent exacerbations of worsening redness and irritation of the eyelid.

INCIDENCE

A. Blepharitis affects all age and ethnic groups. Current studies indicate the mean age of clients affected with blepharitis is 50 years, and clients with staphylococcal blepharitis are approximately 42 years old and primarily female.

PATHOGENESIS

A. Seborrheic: excessive shedding of skin cells and blockage of glands.
B. *Staphylococcus:* the most common bacteria found, responsible for bacterial infection of lid margin.
C. Commonly seen with altered lipid protection and increased bacterial colonization of the ocular surface and eyelids.
D. Allergic dermatitis (contact dermatitis), eczema, psoriasis.

PREDISPOSING FACTORS

A. Diabetes.
B. *Candida*.
C. Seborrheic dermatitis.
D. Acne rosacea.
E. Eczema.
F. Aging.
G. Clients who wear contact lens and have acquired giant papillary conjunctivitis.

COMMON COMPLAINTS

A. Burning and itching.
B. Lacrimal tearing.
C. Photophobia.
D. Recurrent eye infections, styes, or chalazia.
E. Dry, flaky secretions on lid margins and eyelashes.
F. Gritty sensation to both eyes.
G. Dry eyes.

OTHER SIGNS AND SYMPTOMS

A. Seborrheic blepharitis: lid margin swelling and erythema, flaking, nasolabial erythema, and scaling.
B. *Staphylococcus aureus* blepharitis: erythema/edema, scaling, burning, tearing, itching, and recurrent stye or chalazia.
C. May have dandruff of scalp and eyebrows.

SUBJECTIVE DATA

A. Elicit onset and duration of signs and symptoms.
B. Note sensations of itching, burning, or pain in the eye.
C. Ask: What makes signs and symptoms worse? What makes signs and symptoms better?
D. Any change in soaps, creams, lotions, or shampoos?
E. Has the client had similar signs and symptoms in the past?
F. Note any visual change or pain since the last eye exam.
G. Note contributing factors involved, if present. These should include smoking, allergen exposure, contact lenses, and use of retinoid products.
H. Ask about risk for foreign bodies or contact with toxins transferred from hands to eyelids (occupational exposures).

PHYSICAL EXAMINATION

A. Inspect:
  1. Inspect eyes, noting extraocular eye movements (EOMs).
  2. Examine sclera, pupil, iris, and fundus.
  3. Examine eyes for red reflex and corneal light reflex.
  4. Note erythema or edema on lid margin; note dryness, scaling, and flakes.
  5. Examine for foreign body or corneal abrasion.
  6. Assess vision using Snellen chart.

DIAGNOSTIC TESTS

A. None; however, cultures of eyelid margins may be warranted if signs/symptoms indicate.

DIFFERENTIAL DIAGNOSES

A. Blepharitis:
  1. *S. aureus.*
  2. Seborrheic.
  3. *Demodex folliculorum.*
B. Conjunctivitis.
C. Squamous cell carcinoma.
D. Stye (hordeolum).
E. Chalazion.
F. Upper respiratory infection.
G. Sinusitis with periorbital involvement.

PLAN

A. General interventions:
  1. Assess the client and rule out bacterial infection and vision changes.
  2. Clients with frequent recurrent blepharitis need further follow-up with an ophthalmologist. ▶

The contributions of Mellisa Hall to this chapter in prior editions are acknowledged here.

B. Client teaching:
1. Wash eyelids with antibacterial soap and water. Use over-the-counter (OTC) eyelid scrub product or baby shampoo.
2. Apply warm compress to eye for comfort daily for approximately 10 to 20 minutes.
3. Stop use of contacts until the eye is healed. Avoid eye makeup while lids are inflamed.
4. Encourage good hygiene for prevention of recurrent episodes.
C. Pharmaceutical therapy:
1. Apply erythromycin ophthalmic ointment or azithromycin ophthalmic solution to margin of eye at bedtime, taking care not to contaminate the medication bottle. Bacitracin is an alternative but has been associated with contact dermatitis.
2. Oral antibiotics for severe blepharitis not responding to topical antibiotics: tetracycline 250 mg PO QID for 4 weeks; alternative: doxycycline 100 mg PO BID, or oxacillin 250 mg QID for 4 weeks (contraindicated during pregnancy and lactation).
3. Consider long-term treatment with doxycycline if infections reoccur.
4. Topical glucocorticoids, while having a role in treatment, should be prescribed only by an ophthalmologist due to potential side effects.
5. Consider use of topical cyclosporine ophthalmic drops such as Restasis.

FOLLOW-UP

A. Recommend follow-up with a primary provider in 1 to 2 weeks.
B. Consider referral to an eye specialist for recurrent episodes of blepharitis.

EMERGENT ISSUES/INSTRUCTIONS

A. Same-day referral to an ophthalmologist should be arranged for clients with severe eye pain, redness, light sensitivity, vision changes, corneal concerns, or symptoms not responding to initial therapy.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. If symptoms are unilateral only, client should be evaluated by an ophthalmologist as blepharitis is a bilateral condition. Unilateral cases may be associated with malignancy.
2. Azithromycin may lead to abnormalities of heart electrical rhythm; use with caution in clients at high risk of cardiovascular disease.
3. Geriatric clients who often rub their eyes and have a history of meibomian gland dysfunction or keratoconus (thinning, protruding cornea) will tend to suffer from blepharitis disease progression. Erythromycin ointment is recommended as a first-line treatment if staphylococcal infection is present. Keep eyes moistened during the day with artificial tear formulations and ophthalmic gel solutions at night (e.g., Systane gel drop lubricant).
4. Clear lids with gentle cleaning with dandruff shampoo or applying tea tree oil to lids; referral if no improvement within 6 weeks.

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CATARACTS

DEFINITION

A. A cataract, an opacity of the crystalline lens of the eye, causes progressive, painless loss of vision (functional impairment). Presenile and senile cataract formation is painless and progresses throughout months and years. Cataracts are frequently associated with intraocular inflammation and glaucoma.

INCIDENCE

A. Cataracts are the most common cause of blindness in the world.
B. Of people older than 60, 95% have cataracts without visual disturbance.
C. Of people older than 40, 50% have significant visual loss due to cataracts.
D. Prevalent cases in the United States are 61% female and 39% male.
E. The National Institutes of Health projected that by year 2030 approximately 40 million U.S. citizens are expected to suffer from cataracts and approximately 50 million people by 2050.

PATHOGENESIS

A. Age-related changes of the lens of the eye result from protein accumulation, which produces a fibrous thickened lens that obscures vision.

PREDISPOSING FACTORS

A. Age.
B. Trauma.
C. Medications (e.g., topical or systemic steroids, major tranquilizers, or some diuretics).
D. Medical diseases (e.g., diabetes mellitus, metabolic syndrome, Wilson disease, hypoparathyroidism, glaucoma, congenital rubella syndrome, chronic anterior uveitis).
E. Chronic exposure to UVB light (including sunlight).
F. Down syndrome.
G. Low antioxidant vitamin use.
H. Alcohol use.
I. Smoking.
J. Lower educational level.
K. Poor nutrition and sedentary lifestyle.
L. Lead exposure.
M. Family history.

COMMON COMPLAINTS

A. Decreased vision, including reading road signs or small print.
B. Blurred or foggy vision, "ghost" images.
C. Inability to drive at night.

OTHER SIGNS AND SYMPTOMS

A. Initial visual event can be a shift toward nearsightedness.
B. Visual impairment can be more marked at distances, with abnormal visual acuity exams.
C. Severe difficulty with glare.
D. Altered color perception.
E. Frequent falls or injuries.

SUBJECTIVE DATA

A. Review the onset, course, and duration of visual changes, including altered day or night vision and nearsighted versus farsighted vision. Rule out diplopia or hemianopia.
B. Assess whether involvement is in one or both eyes.
C. Determine what improves vision—use of glasses or use of extra light.
D. Review the client's medical history and current medications.
E. Review the client's history for traumatic injury.
F. Discuss the client's occupation and leisure activities to determine exposure to UV rays.
G. Review lifestyle choices that would identify risk of cataract formation.

PHYSICAL EXAMINATION

A. Inspect:
1. Conduct a funduscopic examination.
a. Check red reflex and opacity.
i. A bright red reflex is seen in the normal eye.
ii. Cataract formation is seen by the disruption of the red reflex.
iii. Lens opacities appear as dark areas against the background of the red-orange reflex.
b. Examine color of opacity. For brunescent cataracts, the nucleus acquires a yellow-brown coloration and becomes progressively more opaque.
c. Check retinal abnormalities, hemorrhage, scarring, and drusen (small yellow deposits).
d. Examine extraocular movements and papillary reactivity.

DIAGNOSTIC TESTS

A. Perform visual acuity exam.
B. Perform peripheral vision exam.
C. Perform slit-lamp exam to determine the exact location and type of cataract.
D. Perform dilated eye exam (performed by an ophthalmologist).

DIFFERENTIAL DIAGNOSES

A. Cataracts.
B. Glaucoma.
C. Age-related macular degeneration (AMD). AMD causes vision loss that is symptomatically similar to cataracts.
D. Diabetic retinopathy.
E. Temporal arteritis.
F. Cerebral vascular accident.
G. Central nervous system (CNS) lesions or neurodegenerative conditions.

PLAN

A. General interventions:
1. Monitor the client for increased interference of visual impairment on their lifestyle.
2. Cataracts do not need to be removed unless there is impairment of normal, everyday activities.
3. Surgery is the definitive treatment; however, modification of glasses may improve vision adequately to defer surgery. Contact lenses are optically superior to glasses.
B. Client teaching:
1. Prevention is important. Teach the client to use protective eyewear to prevent trauma and sun exposure.
2. Discuss modifiable lifestyle behaviors that increase the risk of cataract formation.
3. Wear a hat with a visor to protect eyes when outdoors.
4. Vitamins: Lutein and zeaxanthin are associated with reduced cataract formation in elderly females.
5. Clients may not be aware of Medicare coverage for surgical removal. Referring them to Medicare.gov will help them understand costs of surgical intervention.

FOLLOW-UP

A. Surgical removal is indicated if the visual disturbance is interfering with the client's life, such as causing falls or prohibiting reading.

CONSULTATION/REFERRAL

A. Refer client for ophthalmologic consultation.
B. Clients should be followed by an optometrist or an ophthalmologist to monitor the cataract for increased size and progressive visual impairment.
C. Contact a social worker or community resources as needed.

INDIVIDUAL CONSIDERATIONS

A. Adults:
1. Cataracts are most commonly seen after the age of 60.
2. Clients complaining of visual disturbance should be screened for cataracts.
B. Geriatrics:
1. More than half of Americans ≥80 years old have cataracts or have had cataract surgery. Cataracts in older adults tend to present as a yellow-brown color on the lens. Clients may report a problem with identifying blues, blacks, and purples. Other complaints include cloudy/blurry vision, faded colors, glare, halo around lights (home appliances or headlights), poor night vision, and double/multiple images.
2. After 60 years of age, implement a comprehensive dilated eye exam at least once every 2 years.
3. Interventions for geriatrics include the following:
a. Reduction of UV light exposure. Encourage older adults to wear sunglasses and wide-brim hats.
b. Smoking cessation and reduce/avoid alcohol use.
c. Surgery: diagnostic preoperative indications for geriatrics:
i. EKG for clients with heart disease to determine stability.
ii. Potassium and creatinine levels for clients on diuretics (renal function).
iii. Glucose trends during stress/infections with diabetics in order to prepare any short-term medicine changes.
iv. Close monitoring for clients taking scheduled anticoagulant/antiplatelet medications.
4. Secondary cataracts may develop after long-term steroid use. Be sure to monitor clients who have chronic obstructive pulmonary disease (COPD) or rheumatoid arthritis (RA)/autoimmune disorders and who have been taking daily steroids ≥1 year.

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CHALAZION

DEFINITION

A. Chalazion is a chronic lipogranulomatous inflammation of a meibomian gland located in the eyelid margin. It differs from a hordeola in that it is a result of gland obstruction rather than an infection. Inflammation occurs from occlusion of the ducts.

INCIDENCE

A. The incidence is unknown.

PATHOGENESIS

A. Meibomian glands secrete the lipid layer of the tear film that covers and protects the eye. When the glands become blocked, the oil or lipid extrudes into the surrounding tissue, causing formation of a painless, rubbery nodule.

PREDISPOSING FACTORS

A. Chalazion may occur as a secondary infection of the surrounding tissues (stye). Chalazia occur more frequently in clients with blepharitis and rosacea.

COMMON COMPLAINTS

A. Swelling, nontender palpable nodule, usually pea-sized, inside lid margin or eye.
B. Discomfort or irritation due to swelling.

OTHER SIGNS AND SYMPTOMS

A. Tearing.
B. Feeling of a foreign body in the eye.
C. If infection is present, the entire lid becomes painfully swollen.

SUBJECTIVE DATA

A. Review onset of symptoms, their course and duration, and any concurrent visual disturbance.
B. Question the client regarding possible foreign body or trauma to the eye.
C. Elicit the quality of pain or tenderness of the eyelid.
D. Review past eye problems and the treatment received.
E. Question for eye tearing, discharge, or matting.
F. Question for employment duties and hobbies predisposing to foreign bodies.

PHYSICAL EXAMINATION

A. Check temperature.
B. Inspect:
  1. Inspect the eye, sclera, and conjunctiva for a foreign body, erythema, vessel injection, discharge, matting, inflammation of the eyelids, or pterygium.
  2. Check for red- or gray-colored subconjunctival mass in the upper and lower eyelid.
C. Palpate:
  1. Palpate eyelid for masses, foreign-body sensation, and tenderness.
  2. Check for preauricular adenopathy. Usually a hard, nontender nodule is found on the middle portion of the tarsus, away from the lid border; it may develop on the lid margin if the opening of the duct is involved. Some chalazia continue to increase in size and can cause astigmatism by putting pressure on the eye globe.
  3. Chalazia may become acutely tender; however, note the difference between the chalazia and the stye, which is found on the lid margin.

DIAGNOSTIC TESTS

A. Perform visual acuity exam.

DIFFERENTIAL DIAGNOSES

A. Chronic dacryocystitis.
B. Hordeolum.
C. Blepharitis.
D. Xanthelasma.
E. Cellulitis of the eyelid.

PLAN

A. General interventions:
  1. Small chalazia do not usually require treatment.
  2. Warm, moist compress may be applied for 15 minutes four times a day.
B. Client teaching:
  1. Instruct client regarding use of warm compress and good handwashing.
  2. Use of baby shampoo and lid massage may be helpful.
C. Pharmaceutical therapy:
  1. Antibiotic treatment is not recommended as chalazion is a granulomatous condition. However, if infected, give doxycycline 100 mg PO BID for 10 days, OR minocycline 50 mg daily for 10 minutes. If unable to take tetracycline, metronidazole is recommended.
  2. Intrachalazion corticosteroid injection by an ophthalmologist may be considered.

FOLLOW-UP

A. For large chalazia, follow up with client in 1 week and then evaluate the client every 2 to 4 weeks.

CONSULTATION/REFERRAL

A. Refer to an ophthalmologist for incision and curettage if the chalazion does not resolve spontaneously.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
  1. To prevent the development of infected chalazion, have the client routinely remove makeup at night and gently wash eyes. Encourage washing their hands frequently during the day.
  2. Nonpharmacologic treatments include warm compress for 20 to 30 minutes (rewetting washcloth every 5–10 minutes), applying a gentle massage, discontinuing use of contact lens and eye makeup until healed, and encouraging clients to keep hands clean and to not rub their eyes.
  3. Pharmacologic treatments common in geriatrics include short-term nonsteroidal anti-inflammatory drugs (NSAIDs) for pain and inflammation and Tobradex ointment three to four times a day for 1 week (combination of an antibiotic and an anti-inflammatory).
  4. Recurrent chalazion in the elderly should be biopsied to rule out malignancy.

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CONJUNCTIVITIS

DEFINITION

A. Conjunctivitis is inflammation of the conjunctiva. All conjunctivitis is erythemic, but not all erythemic eyes are conjunctivitis. Differential diagnosis is key in the diagnosis.

INCIDENCE

A. Conjunctivitis may be subdivided into viral, bacterial, allergic, and nonallergic subcategories. Viral conjunctivitis is the most common type of conjunctivitis.

PATHOGENESIS

A. Primarily, four types of conjunctivitis are observed:
  1. Bacterial (*Staphylococcus aureus*, *Corynebacterium*, *Pseudomonas*, *Moraxella*, *Haemophilus influenzae*, *Streptococcus pneumoniae*, *Neisseria gonorrhoeae*, and *Chlamydia*).
  2. Viral (adenovirus, coxsackievirus, herpes zoster ophthalmicus, and enteric cytopathic human orphan [ECHO] viruses).
  3. Allergic (seasonal pollens or allergic exposure).
  4. Nonallergic (commonly related to mechanical or chemical trauma).

PREDISPOSING FACTORS

A. Contact with another person with a diagnosis of conjunctivitis.
B. Exposure to sexually transmitted infection (STI).
C. Other atopic conditions (allergies).
D. Mechanical or chemical injury.

COMMON COMPLAINTS

A. Red eyes.
B. Eye drainage.
C. Itching (with allergic conjunctivitis).
D. Matting upon awakening.

OTHER SIGNS AND SYMPTOMS

A. Bacterial:
  1. Fast onset, 12 to 24 hours of copious purulent or mucopurulent discharge.
  2. Burning, stinging, or gritty sensation in eyes.
  3. Crusted eyelids upon awakening, with swelling of the eyelid.
  4. Usually starts out unilaterally; may progress to bacterial infection.
  5. Bacterial conjunctivitis may present as beefy red conjunctiva.
B. Viral:
  1. Symptoms may begin in one eye and progress to both eyes.
  2. Tearing of eyes.
  3. Sensation of foreign body.
  4. Systemic symptoms of upper respiratory infection (runny nose, sore throat, sneezing, fever).
  5. Preauricular or submandibular lymphadenopathy.
  6. Photophobia, impaired vision.
  7. Primary herpetic infection: vesicular skin lesion, corneal epithelial defect in form of dendrite, uveitis.
C. Allergic:
  1. Itchy, watery eyes, bilateral.
  2. Seasonal symptoms.
  3. Edema of eyelids without visual change.
  4. With allergic conjunctivitis, hyperemia of eyes is always bilateral and giant papillae on tarsa may be seen.
  5. May also see eczema, urticaria, and asthma flare.
D. Nonallergic (mechanical or chemical):
  1. Itchy, watery eyes, unilateral or bilateral.
  2. Frequent causes may include smoke, dust, vapors, and foreign objects.
  3. Tearing.
  4. Vision obstructed from blurring or tearing.

SUBJECTIVE DATA

A. Elicit onset, duration, and course of symptoms.
B. Question client regarding presence of discharge upon awakening.
C. Elicit changes in vision since symptoms began.
D. Determine whether there has been any injury or trauma to the eye, including foreign body.
E. Assess whether these symptoms have appeared before.
F. Rule out exposure to anyone with conjunctivitis.
G. Ask client about any new events, such as use of contact lenses or change in contact lenses or solutions.
H. Review client and family history of allergies.
I. Question the client regarding pain and rate on a pain scale.

PHYSICAL EXAMINATION

A. Check temperature.
B. Inspect:
  1. Observe eyes for color and foreign objects. Perform complete eye exam, including acuity exam.
  2. Note lid edema.
  3. Assess pupillary reflexes.
  4. Examine eyelids and periorbital skin for erythema, vesicles, inflammation, or tenderness.
  5. Inspect ears, nose, and throat.
C. Auscultate:
  1. Auscultate heart and lungs.
D. Palpate:
  1. Palpate preauricular lymph nodes and anterior and posterior cervical chain lymph nodes.

DIAGNOSTIC TESTS

A. Gram stain testing for discharge/exudate extracted from eyes if gonococcal infection is suspected.
B. Culture if gonococcal or chlamydial infection is suspected, recurrent conjunctivitis, or treatment-resistant infection.
C. Perform fluorescein stain of eye if foreign body or corneal abrasion/ulceration is suspected.
D. Test visual acuity with the Snellen chart. Assess peripheral vision and extraocular eye movements (EOMs).

DIFFERENTIAL DIAGNOSES

A. Conjunctivitis.
B. Corneal abrasion.
C. Blepharitis.
D. Drug-related conjunctivitis.
E. Herpetic keratoconjunctivitis.
F. Iritis.
G. Gonococcal or chlamydial conjunctivitis.
H. Angle-closure glaucoma.
I. Keratitis.
J. Foreign body.

PLAN

A. General interventions:

1. Distinguish between bacterial, allergic, nonallergic, and viral infection.

2. Consider other diagnoses as earlier if eye pain is noted.

B. Client teaching: *See Client Teaching Guides for this chapter, "Conjunctivitis" and "How to Administer Eye Medications."*

1. Cool compress to affected eye should be applied several times a day.

2. Clean eyes with warm, moist cloth from inner to outer canthus to prevent spreading infection.

3. Encourage good handwashing with antibacterial soap.

4. Instruct on the proper method of instilling medication into eye.

5. Instruct the female client to discard all eye makeup, including mascara, eyeliner, and eye shadow, worn at the time of the infection.

6. Teach the client the difference between bacterial, allergic, nonallergic, and viral infections. Educate according to appropriate diagnosis.

7. If using aminoglycoside or neomycin ointments or drops, use caution and monitor closely for reactive keratoconjunctivitis.

8. Contact lens wearers should be prescribed a fluoroquinolone to cover for *Pseudomonas*.

9. Bacterial conjunctivitis is contagious up to 24 hours after starting medication.

10. Viral conjunctivitis is contagious for 48 to 72 hours, but may last up to 2 weeks. This is typically self-limiting and does not require antibiotic treatment.

11. Discuss general eye protection against recurrent exposures or trauma.

C. Pharmaceutical therapy:

1. Bacterial:

a. Aminoglycosides: These should be avoided due to risk of reactive keratoconjunctivitis.

b. Polymyxin B: Trimethoprim/polymyxin B sulfate (Polytrim) ophthalmic ointment in each eye QID for 7 days. Polymyxin B/bacitracin (Polysporin) drops may also be used, 1 gtt Q3H for 7 to 10 days.

c. Macrolides: Erythromycin (Ilotycin) ophthalmic ointment 0.5% in each eye QID for 7 days.

d. Fluoroquinolones: Ciprofloxacin 0.3% Q2H for 2 days, then Q4H for 5 days; moxifloxacin (Vigamox) 0.5% 1 gtt TID for 7 days. (Due to concerns of resistance, fluoroquinolones should be reserved for treatment of bacterial conjunctivitis primarily in contact wearers.)

2. Viral:

a. Trifluridine 1% drops are indicated for ocular herpes, one drop Q2H while awake; no more than nine drops per day; use for 10 to 14 days. Clients suspected with ocular herpes should benefit from a same-day referral to an ophthalmologist as permanent vision loss can be a sequela of herpes conjunctivitis.

b. Ganciclovir 0.15% gel one drop five times a day until epithelial heals and then TID for 1 week.

c. Oral antiviral medications (trifluridine, valacyclovir) may be used for herpes simplex keratitis. Herpes zoster ophthalmicus is often treated with acyclovir, famciclovir, or valacyclovir and lessens symptoms if started within 72 hours of onset of symptoms. **Clients should receive same-day referral** for consideration of treatment of ocular herpes.

3. Allergic:

a. Topical antihistamines/mast cell stabilizer:

i. Azelastine HCl (Optivar): for adults, one drop to the affected eye BID.

ii. Olopatadine HCl (Pataday) 0.2%: one drop to the affected eye daily.

iii. Olopatadine HCl (Patanol) 0.1%: one drop BID to the affected eye.

b. Mast cell stabilizer:

i. Cromolyn sodium (Crolom) ophthalmic solution: one to two drops four to six times daily.

c. Topical nonsteroidal anti-inflammatory drugs (NSAIDs):

i. Ketorolac tromethamine (Acular) 0.5%: for adults, one drop QID; this is used for severe symptoms of atopic keratoconjunctivitis.

d. Artificial tears can be used four to five times daily.

e. Oral antihistamines may be used in severe cases (loratadine or diphenhydramine HCl).

4. Concurrent conjunctivitis and otitis media should be treated with a systemic antibiotic; no topical eye antibiotic is needed.

FOLLOW-UP

A. If complete resolution occurs within 5 to 7 days after proper treatment, follow-up is not needed.

B. If client continues to have equal or worsening symptoms after 48 to 72 hours of treatment or if different symptoms appear, then follow-up and a same-day referral to an ophthalmologist are recommended.

CONSULTATION/REFERRAL

A. Consult or refer client to a physician if they are not responding to treatment within 48 to 72 hours.

B. Same-day referral if client is suspected of having periorbital cellulitis, iritis, keratitis, herpes zoster ophthalmicus, or acute angle-closure glaucoma.

C. Refer to an eye specialist if client has vision change or eye pain, is not responding to treatment, or if sight-threatening red flags are present.

1. Reduced visual acuity with onset of symptoms.

2. Observation of a ciliary flush.

3. Photophobia.

4. Severe foreign-body sensation.

5. Corneal opacity.

6. Fixed pupil.

7. Severe headache associated with nausea.

INDIVIDUAL CONSIDERATIONS

A. Partners:

1. Check partners for gonorrhea and chlamydia when an adolescent or adult presents with gonococcal or chlamydial conjunctivitis.

B. Pregnancy:

1. In pregnancy, change in hormones and the immune system make the client more susceptible to eye infections, especially if there are young children in the house as vectors.

2. Apply cool compress, practice good hand hygiene, and if the client has allergic conjunctivitis Pataday and Optivar are safe to use during pregnancy.

3. If antibiotic drops are needed, erythromycin (predominantly gram-positive coverage) and polymyxin B (gram-negative coverage) are safe during pregnancy.

C. Geriatrics:

1. Lid or lacrimal sac edema or proptosis with conjunctivitis is a red flag to further investigate for cellulitis or an orbital tumor.

2. First-line treatment for conjunctivitis in geriatrics is erythromycin ointment/drops. Second-line is ciprofloxacin 0.3% solution.

3. If signs/symptoms of purulent discharge do not improve within 48 hours after initiating antibiotics, refer to an ophthalmologist.

4. Avoid diphenhydramine secondary to risks of confusion, constipation, blurred vision, delirium, dry mouth, and other anticholinergic symptoms. This may be considered only for acute treatment of a severe allergic reaction.

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CORNEAL ABRASION

DEFINITION

A. A corneal abrasion is a defect of the corneal epithelial tissue, either superficial or deep.

INCIDENCE

A. In the United States, there are 2,000 work-related eye injuries daily, approximately 2.4 million annually.

B. One-third of these eye injuries require treatment in an ED or by an ophthalmologist.

PATHOGENESIS

A. Injury from trauma occurs to the epithelial tissue of the cornea.

PREDISPOSING FACTORS

A. Trauma to the eye caused by a human fingernail, contact lens, animal paws, makeup applicators, UV light burns from tanning beds or welding arcs, tree branches, wood or metal particles, children's toys, or sports injuries.

B. A history of surgical trauma, causing globe weakening.

C. A history of spontaneous corneal erosions.

COMMON COMPLAINTS

A. Sudden onset of eye pain.

B. Foreign-body sensation in the eye.

C. Watery eye.

D. Mild photophobia.

OTHER SIGNS AND SYMPTOMS

A. Change in vision/blurred vision.

B. Redness, swelling, inability to open the eye.

SUBJECTIVE DATA

A. Elicit the onset, duration, and course of symptoms; note any history of similar symptoms.

B. Question the client regarding visual changes (blurred, double, or lost vision, or loss of a portion of the visual field).

C. Question the client regarding the mechanism of injury and how much time has elapsed since the injury (minutes, hours, or days). Ask: What is their occupation, do they work with wood or metal, what sports are involved, or what hobbies predispose them to injury? Were goggles being worn and are they routinely worn during the sport or activity?

D. Review the client's history of exposure to herpetic outbreaks.

E. Determine the degree of pain, if any, and check for headache, photophobia, redness, itching, or tearing.

F. Ascertain whether the client wears contact lenses or glasses and for what length of time.

G. Ask if the client has tried any treatments before presentation to the office. If so, what?

H. Rule out the presence of any other infections, such as sinus infection. **Conjunctival discharge signifies an infectious etiology**.

I. Ask about foreign-body sensations.

J. Rule out farm injury due to risk of bacterial keratitis.

PHYSICAL EXAMINATION

A. Check vital signs: blood pressure, pulse, temperature.

B. Inspect:

1. Observe *both* eyes.

2. Test visual acuity and pupil reactivity and symmetry.

3. Observe cornea surface with direct illumination, noting shadow on surface of iris.

4. Perform funduscopic exam.

5. Evert eyelids for cornea inspection.

6. Inspect for foreign body and remove if indicated.

7. Perform fluorescein stain to visualize changes in epithelial lining. Cobalt blue light or Wood lamp should be used for visualization.

8. Consider slit-lamp examination to rule out traumatic hyphema or open globe injuries.

DIAGNOSTIC TEST

A. Perform fluorescein stain test: **An epithelial defect that stains with fluorescein is the hallmark symptom**.

DIFFERENTIAL DIAGNOSES

A. Corneal abrasion.

B. Corneal foreign body.

C. Acute angle glaucoma.

D. Herpetic infection (herpes simplex virus [HSV]): HSV is associated with decreased corneal sensation.

E. Recurrent corneal ulceration.

F. Ulcerative keratitis.

G. Spontaneous corneal erosion.

PLAN

A. General interventions:

1. Superficial or smaller corneal abrasions should not be patched. ▶

2. For deeper or larger abrasions and if a foreign body is ruled out, apply a patch that prevents lid motion for 24 hours. Patching is not recommended for pain control as it has the potential to delay healing.

3. Pressure patch is no longer recommended.

B. Client teaching: *See Client Teaching Guide for this chapter, "How to Administer Eye Medications."*

1. Discuss the use of protective eyewear and prevention of future ocular trauma in clients with a history of use of power tools or hammering.

2. Advise the client to not use/wear contact lenses until the eye is completely healed.

C. Pharmaceutical therapy:

1. Antibiotic drops or ointment: **Never instill antibiotic ointment if there is a possibility of a perforation. Patch the eye and refer the client to a physician or ophthalmologist:**

a. Erythromycin 0.5% ointment, 1/2-in. ribbon twice a day to four times a day for 7 days.

b. Sulfacetamide sodium ophthalmic solution 10% (Sulamyd), one to two drops instilled into the lower conjunctival sac every 2 to 3 hours during the day; may instill Q6H during the night for 5 to 7 days.

c. Sulfacetamide sodium (Sulamyd) ophthalmic solution or ointment interacts with gentamicin. Avoid using them together.

d. Para-aminobenzoic acid (PABA) derivatives reduce sulfacetamide's action. Wait for half to 1 hour before instilling sulfacetamide.

e. Sulfacetamide precipitates when used with silver preparations. Avoid using them together.

f. Polymyxin B sulfate (Polytrim) 10,000 U, bacitracin zinc 500 U/g ophthalmic ointment (Polysporin), a small ribbon of ointment applied into the conjunctival sac four to six times daily for 7 to 10 days.

g. Bacitracin 500 U/g ointment, 1/2-in. ribbon twice a day to four times a day for 7 to 10 days.

h. Contact lens wearers are often colonized with *Pseudomonas* and should be treated with either a fluoroquinolone or an aminoglycoside. Ciprofloxacin 0.3% solution one to two drops QID for 3 to 7 days; ofloxacin 0.03% one to two drops every 30 minutes on days 1 to 2, Q1H on days 3 to 7, and QID on days 7 to 9; gentamycin 0.3% solution one to two drops QID for 3 to 5 days; or tobramycin (Tobrex) ointment or drops QID for 3 to 5 days.

2. Analgesics: Oral nonsteroidal anti-inflammatory drugs (NSAIDs) or topical analgesics should be used sparingly for the first 24 hours. Diclofenac (Voltaren) 0.1% solution in the eye QID as needed or ketorolac (Acular) 0.5% solution in the eye QID as needed.

3. Avoid use of home prescriptions that will interfere with the healing process.

4. Avoid use of medications containing steroids. These products may increase the risk of superinfection and may slow down the healing process.

5. The length of therapy may vary but should be continued until the client is symptom-free for 24 hours.

FOLLOW-UP

A. Reevaluate the client **within 24 hours**. Cornea usually heals within 24 to 48 hours.

B. If the client is still symptomatic in 48 hours, a same-day referral to an ophthalmologist is warranted.

CONSULTATION/REFERRAL

A. Immediate referral to an ophthalmologist is required for large or central lesions or deep or penetrating wounds. If ophthalmology referral cannot be arranged, the client should be treated at the ED.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Retinal detachment should be considered as a source of eye pain and visual loss, especially in females with severe pregnancy-induced hypertension (HTN).

B. Adults:

1. Encourage protective eyewear for sports (including hockey, soccer, baseball, and basketball) and when using any power equipment or tools.

2. Clients with corneal abrasions secondary to contact lenses are at higher risk of bacterial keratitis. Injuries secondary to contact lenses should be evaluated to rule out a corneal infiltrate or ulcer. If this condition is observed, the client is at risk of *Pseudomonas* keratitis, an ocular emergency.

3. These clients should never receive an eye patch, should be covered with a topical ophthalmic agent that has *Pseudomonas* coverage, and should be evaluated by an ophthalmologist.

C. Geriatrics:

1. Research indicates that eye surgery is successful in the majority of older adult clients, secondary to advancement in technological microsurgery procedures. However, as demand for refractory surgery rises, corneal abrasions are also increasing as a complication of postsurgical ocular procedures.

2. Geriatric clients may not remember details of eye trauma event and should be evaluated for corneal abrasion if there are complaints of pain and photophobia.

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DACRYOCYSTITIS

DEFINITION

A. Infection or inflammation of the lacrimal sac, or dacryocystitis, can be acute or chronic.

B. Dacryocystitis is usually secondary to obstruction of the nasolacrimal duct.

INCIDENCE

A. The incidence is unknown in adults. Dacryocystitis is more common in children than in adults.

B. In adults, dacryocystitis is more common in females and after the age of 40. Females comprise 70% to 83% of cases, with a peak age of 60 to 70 years.

C. Caucasians tend to be more affected than other races because their lacrimal canal is longer and is inclined to deviate.

PATHOGENESIS
A. Bacterial infection of the lacrimal sac is most commonly caused by *Staphylococcus*, *Streptococcus*, *Haemophilus*, and *Pseudomonas*. Methicillin-resistant *Staphylococcus aureus* is more common in acute dacryocystitis.

PREDISPOSING FACTORS
A. Nasal trauma.
B. Deviated septum.
C. Nasal polyps.
D. Congenital dacryostenosis.
E. Inferior turbinate hypertrophy.

COMMON COMPLAINTS
A. Pain in the eye.
B. Redness.
C. Swelling.
D. Fever.
E. Tearing.
F. Visual change.

OTHER SIGNS AND SYMPTOMS
A. Purulent exudate may be expressed from the lacrimal duct.

SUBJECTIVE DATA
A. Elicit the onset, course, and duration of symptoms. Are symptoms bilateral or unilateral?
B. Review the client's activity when the symptoms began to determine if etiology is chemical, traumatic, or infectious.
C. Review other presenting symptoms, such as fever and discharge.
D. Review the client's history for previous episodes. Note treatments used in the past.
E. Review history for a recent herpes simplex virus (HSV) or fever blister.
F. Review ophthalmologic history.
G. Review medications.
H. Review blood glucose in diabetics.

PHYSICAL EXAMINATION
A. Check temperature, pulse, and blood pressure.
B. Inspect:
1. Assess both eyes.
2. Check peripheral fields of vision and sclera.
3. Evaluate conjunctiva for distribution of redness, ciliary flush, and foreign bodies.
4. Inspect lid margins: Evaluate for crusting, ulceration, and masses.
C. Palpate:
1. Palpate lacrimal duct and surrounding bony structures. Discharge can be expressed from the tear duct with application of pressure.

DIAGNOSTIC TESTS
A. Check visual acuity.
B. Culture any discharge.
C. Obtain plain radiographs if foreign body is suspected.
D. Obtain CT to evaluate trauma, suspected tumor, and orbital cellulitis.

DIFFERENTIAL DIAGNOSES
A. Dacryocystitis.
B. Chalazion.
C. Blepharitis.
D. Xanthoma.
E. Bacterial conjunctivitis.
F. Hordeolum.
G. Foreign body.
H. Orbital cellulitis.
I. Preseptal cellulitis.
J. Complications of sarcoidosis.

PLAN
A. General interventions:
1. Apply warm, moist compress at least four times per day.
2. Instruct the client to discard old makeup, including mascara, eyeliner, and eye shadow, used prior to infection.
3. Consider avoiding use of contacts and switch to their glasses.
B. Client teaching: *See Client Teaching Guide for this chapter, "How to Administer Eye Medications."*
1. Advise client to perform proper handwashing and cleaning.
C. Pharmaceutical therapy:
1. Coverage should be aimed at gram-positive organisms. Empiric coverage should include both gram-positive and Gram-negative coverage. Augmentin is a good choice.
2. Acute dacryocystitis:
a. Clindamycin 300 mg Q6H for 7 to 10 days depending on response.
b. Erythromycin 250 mg QID for 7 days.
c. Dicloxacillin 250 mg QID for 7 days.
3. Acute dacryocystitis with orbital cellulitis requires hospitalization with intravenous (IV) antibiotics.

FOLLOW-UP
A. Follow up in 3 days for reevaluation.

CONSULTATION/REFERRAL
A. Acute dacryocystitis: Abrupt onset with erythema, warmth, swelling, and pain of the lacrimal duct should be managed on the same day by an ophthalmologist.
B. Chronic dacryocystitis: Refer the client to an ophthalmologist for irrigation and probing if needed.
C. Lab studies are generally performed by an ophthalmologist.

INDIVIDUAL CONSIDERATIONS
A. Adults/Geriatrics:
1. Referral to an ophthalmologist for acute and chronic condition.

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DRY EYES

DEFINITION
A. Insufficient lubrication of the eye, or dry eyes, is due to a deficiency of any one of the major components of the tear film.
B. Defects in tear production are uncommon but may occur in conjunction with systemic disease. Presence of systemic disease should be evaluated.

INCIDENCE

A. Increased incidence of dry eyes in the elderly is due to decreased rate of lacrimal gland secretions. It has been estimated as a concern in as many as 30% of people 50 years of age and older.

B. The elderly population is vulnerable to dry eye disease due to their decreased rate of lacrimal gland secretions.

PATHOGENESIS

A. Decreased production of one or more components of the tear film results in dry eyes. The tear film comprises three layers:

1. An outermost lipid layer, excreted by the lid meibomian glands.

2. A middle aqueous layer, secreted by the main and accessory lacrimal glands.

3. An innermost mucinous layer, secreted by conjunctival goblet cells.

B. A defect in production of the aqueous phase by lacrimal glands causes dry eyes (keratoconjunctivitis sicca). The condition most often occurs as a physiologic consequence of aging, and it is commonly exacerbated by dry environmental factors. It may also develop in clients with connective tissue disorders.

C. In Sjögren syndrome, the lacrimal glands become involved in immune-mediated inflammation.

D. Mucin production may decline in the setting of vitamin A deficiency.

E. Loss of goblet cells can occur secondary to chemical burns.

PREDISPOSING FACTORS

A. History of severe conjunctivitis.

B. Eyelid defects such as fifth or seventh cranial nerve palsy, incomplete blinking, exophthalmos, and lid movement hindered by scar formation.

C. Drug-induced conditions, including use of anticholinergic agents:

1. Phenothiazine.

2. Tricyclic antidepressants.

3. Antihistamines.

4. Diuretics.

5. Isotretinoin (Accutane).

D. Systemic diseases such as rheumatoid disease, Sjögren syndrome, and neurologic disease.

E. Environmental factors such as heat (wood, coal, gas), air conditioners, winter air, and tobacco smoke.

F. Use of contacts.

G. Increasing age and associated loss of androgens.

H. Lipid abnormalities.

COMMON COMPLAINTS

A. Ocular fatigue.

B. Foreign-body sensation in the eye.

C. Itching, burning, irritation, or dryness sensation in the eye.

D. Redness.

E. Eye discharge.

F. Heavy eyelid sensation.

OTHER SIGNS AND SYMPTOMS

A. Photophobia.

B. Cloudy, blurred vision.

C. Rainbow of color around lights. Acute angle-closure glaucoma can present with a red, painful eye; cloudy, blurred vision and a rainbow of color around lights; dilatation of the pupil; and nausea and vomiting.

D. Bell palsy, signs of stroke, or other conditions that affect the blinking mechanism.

SUBJECTIVE DATA

A. Elicit onset, duration, and frequency of symptoms.

B. Note factors that worsen or alleviate symptoms.

C. Note medical history for systemic conditions and strokes.

D. List current medications, noting anticholinergic drugs and isotretinoin (Accutane) use.

E. Note whether the client wears contact lenses or glasses and ask for what length of time.

F. Review occupational and home exposure to irritants, allergens, and fans.

G. Assess whether the client produces tears. Note eye drainage amount, color, and frequency.

H. Review history of any ocular disease, surgeries, or autoimmune disorders.

I. Ask about dry mouth (as another symptom in association with Sjögren syndrome).

J. Confirm no foreign-body sensation or risk of exposures to foreign bodies.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.

B. Inspect:

1. Observe and evaluate *both* eyes.

2. Conduct a detailed eye exam. Check the eye, lid, and conjunctiva for masses, corneal abrasions, foreign bodies, and redness.

3. Check pupil reactivity and corneal clarity. The corneal reflex should be checked if there is concern for neuroparalytic keratitis or facial nerve palsy.

4. Complete a funduscopic exam. Check for completeness of lid closure as well as position of eyelashes.

5. Examine mouth for dryness.

6. Inspect skin for butterfly rash.

C. Palpate:

1. Palpate lacrimal ducts for drainage.

2. Invert upper lid and check for foreign body or chalazion.

3. Check sinuses for tenderness.

4. Palpate thyroid.

5. Palpate joints for warmth and redness or inflammation.

DIAGNOSTIC TESTS

A. There is no "gold standard" for the diagnosis of dry eye syndrome. Clinical diagnosis using a thorough history and physical exam should be considered. Validated questionnaires include the following:

1. Ocular Surface Disease Index (OSDI).

2. Impact of Dry Eye on Everyday Life (IDEEL).

3. Salisbury Eye Evaluation (SEE) Questionnaire.

B. A Schirmer test has variable results between examiners. To perform Schirmer test, use Whatman no. 41 filter paper, 5 mm by 35 mm. A folded end of filter paper is hooked over the lower lid nasally and the client is instructed to keep their eyes lightly closed during the test. Wetting is measured after 5 minutes; less than 5 mm is usually abnormal.

C. Slit-lamp exam.

DIFFERENTIAL DIAGNOSES

A. Dry eyes.

B. Stevens–Johnson syndrome.

C. Sjögren syndrome: The triad of chronic dry mouth, dry eyes, and arthritis suggests Sjögren syndrome. Facial telangiectasias, parotid enlargement, Raynaud syndrome, and dental caries are associated features. Clients complain first of burning ▶

and a sandy, gritty, foreign-body sensation, particularly later in the day.
D. Systemic lupus erythematosus.
E. Scleroderma.
F. Ocular pterygium.
G. Superficial pemphigoid.
H. Vitamin A deficiency.

PLAN

A. General interventions:
1. If no ocular disease is present, reduce environmental dryness by use of a room humidifier, eliminate fans, and reduce screen time, for a 2-week trial.
2. Apply artificial tear substitutes and nonprescription drops.
3. Consider stopping medications being used that may be contributing to dry eye symptoms.
4. Caution should be used when using over-the-counter (OTC) allergy medications, if allergy is a contributing cause. Topical antihistamines may exacerbate the condition over time or cause systemic side effects in the elderly.
B. Client teaching: *See Client Teaching Guide for this chapter, "How to Administer Eye Medications."*
C. Pharmaceutical therapy:
1. Topical artificial tears one or two drops QID, preferably one without preservatives (e.g., TheraTears, dry eye therapy, and Tears Naturale).
2. Drops may be instilled as often as desired.
3. Cyclosporine ophthalmic drops 0.05% or 0.01% one drop BID have been shown to reduce symptoms of dry eye. Six weeks of treatment may be needed before an effect is appreciated. OTC artificial tears may be used in addition.
4. Oral antioxidants and omega-3 fatty acids have shown some promise in reducing symptoms of dry eye.
5. Lifitegrast 5% one drop to each eye Q12H. This is usually ordered by ophthalmology.

FOLLOW-UP

A. Reevaluate the client in 2 weeks.

CONSULTATION/REFERRAL

A. Refer the client to an ophthalmologist if symptoms are unrelieved at 2-week follow-up.
B. Make an immediate referral for red eye, visual disturbance, or eye pain.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. The rate of lacrimal gland secretions diminishes with aging; therefore, the elderly are at an increased risk of developing dry eye.
2. Angiotensin-converting enzyme (ACE) inhibitors may reduce the risk of dry eye syndrome in some clients. Consider treatment with ACE inhibitors for hypertension (HTN) as appropriate in clients.
3. Assess use of OTC antihistamines and decongestants that can cause dry eye.
4. Evaluate for autoimmune disorders, including rheumatoid arthritis, scleroderma, and thyroid disorder, as well as possible vitamin D and vitamin A deficiencies.
5. Rule out neurogenerative disorders such Parkinson disease as symptoms of dry eyes.
6. Consider environmental adjustments, room humidifiers, and goggles/moisture chambers to fit eyewear (wear occasionally), and instruct older adults to frequently blink to increase natural eye lubrication. Keep eyes moistened during the day with artificial tear formulations and with ophthalmic gel solutions at night (e.g., Systane gel drop lubricant).

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EXCESSIVE TEARS

DEFINITION

A. Excessive tears disorder is an overproduction of tears. Complaints vary from watery eyes to overflowing tears that run down the cheeks, a condition known as epiphora.

INCIDENCE

A. The incidence is unknown. Previous research has reported the highest incidence of excessive tearing is in individuals between 50 and 70 years old and is more prevalent in females.

PATHOGENESIS

A. The most common cause is reflex overproduction of tears (as occurs in the elderly) due to a deficiency of the tear film.
B. Lacrimal pump failure and obstruction of the nasolacrimal outflow system are other causes of excessive tears.
C. Canalicular infections may be caused by *Actinomyces israelii* (*Streptothrix*) and *Candida*.

PREDISPOSING FACTORS

A. Blepharitis (inflammation of the eyelid).
B. Allergic conjunctivitis (infectious or foreign body).
C. Exposure to cold, air conditioning, or dry environment.
D. Lid problems: impaired pumping action of the lid motion due to seventh nerve palsy or conditions that stiffen the lids, such as scars or scleroderma.
E. Lid laxity from aging or ectropion (sagging of the lower lid).
F. Sinusitis.
G. Atopy.
H. Age: increased incidence in the elderly due to an overproduction of tears by the lacrimal gland.
I. Congenital obstruction.

COMMON COMPLAINTS

A. Watery eyes or tears running down cheeks are common complaints.

OTHER SIGNS AND SYMPTOMS

A. Unilateral tearing: obstructive etiology.
B. Bilateral tearing: environmental irritants.

SUBJECTIVE DATA

A. Inquire about onset, course, and duration of symptoms. Note frequency of excessive tearing. ▶

B. Ascertain whether this is a new symptom or whether the client has a history of similar complaints. Ask how it was treated and what the response was to treatment(s).
C. Determine severity. Do the tears run down the cheek?
D. Ascertain whether tearing is unilateral or bilateral.
E. Review common environmental predisposing factors.
F. Question the client regarding vision changes.
G. Review medical history.
H. Review recent history for sinus infections or drainage, facial fractures, and surgery.
I. Confirm no pain associated with condition.

PHYSICAL EXAMINATION

A. Inspect:
1. Evaluate *both* eyes.
2. Observe the lid structure and motion.
3. Conduct a dermal exam to rule out butterfly rash.
4. Confirm pupil reactivity.
5. Perform a funduscopic exam for vascular changes.
B. Palpate:
1. Apply gentle pressure over the lacrimal sac to check drainage.
2. Invert upper lid to check for foreign body.
3. Palpate face for sinus tenderness.

DIAGNOSTIC TEST

A. Culture any drainage expressed from the lacrimal sacs.

DIFFERENTIAL DIAGNOSES

A. Excessive tears.
B. Dendritic ulcer: Early symptoms are tears running down cheeks associated with a foreign-body sensation.
C. Congenital glaucoma.
D. Dacryocystitis (purulent discharge).
E. Reflex tearing caused by dry eye.
F. Blepharitis.

PLAN

A. General interventions:
1. Eliminate identifiable irritants.
2. Treatment is aimed mainly at the underlying condition (e.g., ocular infection).
3. Dacryocystitis is treated with hot compress at least four times a day and with systemic antibiotics.
B. Client teaching:
1. Instruct the client on the application of compress if dacryocystitis is suspected.
2. After pathology is ruled out, reassure the client excessive tearing is common and more frequent with aging.
C. Pharmaceutical therapy:
1. None required for diagnosis of excessive tears without infectious pathology.
2. Dacryocystitis:
a. Erythromycin 250 mg QID for 7 days.
b. Dicloxacillin 250 mg QID for 7 days.

FOLLOW-UP

A. See client in 48 to 72 hours to evaluate symptoms, especially if antibiotic therapy was needed for infection.

CONSULTATION/REFERRAL

A. Clients unresponsive to treatment should be promptly referred to an ophthalmologist.
B. Consider referral for lid malposition or nasolacrimal duct obstructions.

INDIVIDUAL CONSIDERATIONS

A. Adults:
1. Consider mental health support if condition alters self-image and limits socialization.
B. Geriatrics:
1. Encourage geriatric clients to protect eyes when traveling (airplanes, dry environments, and hot rooms during winter) and when facing exposure to windy areas, bright sunlight, and abrupt temperature changes. All these sources could easily cause older adults with dry eyes to excessively tear.
2. If shielding/protective measures do not resolve the symptom, further evaluate for infection or blocked tear duct. Common causes of increased tear production in geriatrics are dry eyes, allergic sinusitis, and upper respiratory infections.
3. How to determine dry eyes versus obstruction (excessive tears vs. nasolacrimal drainage) is as follows: If tears do not run down the cheeks, then it most likely is dry eyes. If tears run down the cheeks, then it most likely is nasolacrimal drainage. Unexplained excessive tears (epiphora) in geriatric clients will need to be referred to a specialist for further evaluation to rule out tumors or abscess.

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EYE PAIN

DEFINITION

A. Sensation of pain may affect the eyelid, conjunctiva, or cornea.

INCIDENCE

A. Unknown. Pain in the eye is most often produced by conditions that do not threaten vision.

PATHOGENESIS

A. The external ocular surfaces and the uveal tract are richly innervated with pain receptors. As a result, lesions or disease processes affecting these surfaces can be acutely painful.
B. Pathology confined to the vitreous, retina, or optic nerve is rarely a source of pain.

PREDISPOSING FACTORS

A. Eyelids: inflammation such as hordeolum (stye), trichiasis (in-turned lash), and tarsal foreign bodies.
B. Conjunctiva: viral and bacterial conjunctivitis or allergic conjunctivitis; or toxic, chemical, and mechanical injuries.
C. Cornea: keratitis (inflammation of the cornea) accompanying trauma, infection, exposure, vascular disease, or decreased lacrimation; microbial keratitis from contact use. If blood vessels invade the normally avascular corneal stroma, vision may become cloudy. Severe pain is a prominent symptom; movement of the lid typically exacerbates symptoms.

COMMON COMPLAINTS

A. Eye pain (sharp, dull, deep): The quality of the pain needs to be considered. Deep pain is suggestive of an intraocular problem. Inflammation and rapidly expanding mass lesions may cause deep pain. Displacement of the globe and diplopia may ensue. ▶

B. Eye movement may cause sharp pain due to meningeal inflammation (the extraocular rectus muscles insert along the dura of the nerve sheath at the orbital apex). Most cases are idiopathic, but 10% to 15% are associated with multiple sclerosis.

OTHER SIGNS AND SYMPTOMS

A. Symptoms may be unilateral or bilateral.
- **1.** Eyelids:
  - a. Tenderness.
  - b. Foreign-body sensation.
  - c. Redness.
  - d. Edema.
- **2.** Conjunctiva:
  - a. Mild burning.
  - b. Foreign-body sensation.
  - c. Itching (allergic).
- **3.** Cornea:
  - a. Burning.
  - b. Foreign-body sensation.
  - c. Considerable discomfort.
  - d. Reflex photophobic tearing.
  - e. Pain exacerbated by blinking.
  - f. Pain relieved with pressure holding the lid shut. With a foreign body or a corneal lesion, pain is exacerbated by lid movement and relieved by cessation of lid motion.
- **4.** Sclera:
  - a. Redness or vessel injection.
- **5.** Uveal tract (uveitis or iritis):
  - a. Dull, deep-seated ache and photophobia.
  - b. Profound ocular and orbital pain radiating to the frontal and temporal regions accompanying sudden elevation of pressure (acute angle-closure glaucoma).
  - c. Vagal stimulation with high pressure may result in nausea and vomiting.
  - d. Usual history of mild intermittent episodes of blurred vision preceding onset of throbbing pain, nausea, vomiting, and decreased visual acuity.
  - e. Halos around light.
- **6.** Orbit:
  - a. Deep pain with inflammation and rapidly expanding mass lesions.
  - b. Eye movement causing sharp pain due to meningeal inflammation.
- **7.** Sinusitis:
  - a. Secondary orbital inflammation and tenderness on extremes of eye movement.

SUBJECTIVE DATA

A. Review the onset, duration, and course of symptoms. Inquire regarding the quality of pain.
B. Review any predisposing factors such as trauma or a foreign object. Ask: Was the onset sudden or gradual?
C. Note reported changes in visual acuity or color vision.
D. Note aggravating or alleviating factors.
E. Determine whether the eye pain is bilateral or unilateral.
F. Review history for herpes, infections, and toxic or chemical irritants.
G. Review history for glaucoma and previous eye surgeries or treatments.
H. Assess the client for any other symptoms, such as migraine headache, sinusitis, or tooth abscess.
I. Inquire whether the client has lost a large amount of sleep or slept with contact lenses.
J. Inquire whether they have been exposed to a large amount of UV light or sunlight (vacation, tanning beds).
K. Review history for any other medical problems, such as lupus, sarcoidosis, or inflammatory bowel disease.

PHYSICAL EXAMINATION

A. Inspect:
- **1.** Evaluate *both* eyes.
- **2.** Test visual acuity and color vision.
- **3.** Observe for extraocular eye movements (EOMs) and visual fields.
- **4.** Check the eye, lid, and conjunctiva for masses, foreign bodies, and redness.
- **5.** Check pupil reactivity and corneal clarity.
- **6.** Conduct a funduscopic exam for disc abnormalities.
- **7.** Perform ear, nose, and throat exam.

B. Palpate:
- **1.** Palpate lacrimal ducts for drainage.
- **2.** Palpate sinuses for tenderness.
- **3.** Invert upper lid and check for foreign body or chalazion.
- **4.** Palpate the periorbital skin for tenderness.

DIAGNOSTIC TESTS

A. Fluorescein stain.
B. Measurement of intraocular pressure (IOP).

DIFFERENTIAL DIAGNOSES

A. Eye pain.
B. Hordeolum.
C. Chalazion.
D. Acute dacryocystitis.
E. Irritant exposure.
F. Conjunctival infection.
G. Corneal abrasion.
H. Foreign body or other trauma.
I. Ulcers.
J. Ingrown lashes.
K. Contact lens abuse.
L. Scleritis.
M. Acute angle-closure glaucoma: Fixed, midposition pupil, redness, and a hazy cornea may be present.
N. Uveitis.
O. Referred pain from extraocular sources such as sinusitis, tooth abscess, tension headache, temporal arteritis, and prodrome of herpes zoster.
P. Iritis.
Q. Bacterial keratitis.
R. Temporal arteritis.
S. Cluster headache.
T. Optic neuritis.
U. Orbital cellulitis.

PLAN

A. General interventions:
- **1.** The initial task is to be sure that there is no threat to vision.
- **2.** Treatment modality depends on the underlying cause of eye pain.

B. Client teaching: *See Client Teaching Guide for this chapter, "How to Administer Eye Medications."*

C. Pharmaceutical therapy:
  1. Medication depends on the underlying cause.

FOLLOW-UP
A. Follow-up depends on the underlying cause.

CONSULTATION/REFERRAL
A. Any change in visual acuity or color vision requires an urgent ophthalmologic consultation (same-day referral).

INDIVIDUAL CONSIDERATIONS
A. Geriatrics:
  1. Common pain medications for geriatric eye pain:
    a. Acular 0.5% one drop three to four times a day (maximum 2 weeks). Caution on risk of bleeding secondary to nonsteroidal anti-inflammatory drug (NSAID) classification.
    b. Lidocaine ophthalmic gel 3.5% (Akten) two drops to ocular surface or tetracaine 0.5% eye drops one drop to ocular surface. These anesthetic medications are usually implemented in-clinic for severe pain or applied prior to an ocular procedure/surgery.

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GLAUCOMA, ACUTE ANGLE CLOSURE

DEFINITION
A. Acute angle-closure glaucoma is an ocular emergency caused by elevations in intraocular pressure (IOP) that damage the optic nerve, leading to loss of peripheral fields of vision; this leads to loss of central vision, resulting in blindness.

INCIDENCE
A. Acute angle-closure glaucoma is the second leading cause of blindness in the United States. Ten percent of glaucoma cases are due to angle-closure glaucoma. Approximately 5% to 15% of the population develop some form of glaucoma.

PATHOGENESIS
A. The essential pathophysiologic feature of glaucoma is an IOP that is too high for the optic nerve. Increased IOP increases vascular resistance, causing decreased vascular perfusion of the optic nerve and ischemia. Light dilates the pupil, causing the iris to relax and bow forward. As the iris bows forward, it comes into contact with the trabecular meshwork and occludes the outflow of aqueous humor, resulting in increased IOP.

PREDISPOSING FACTORS
A. Narrow anterior ocular chamber.
B. Prolonged periods of darkness.
C. Drugs that dilate the pupils (e.g., anticholinergics).
D. Advancing age.
E. African American heritage.
F. Family history.
G. Trauma.
H. Neoplasm.
I. Corticosteroid therapy.
J. Neovascularization.
K. Female sex.
L. Hyperopia (farsightedness).

COMMON COMPLAINTS
A. Ocular pain.
B. Blurred vision, decreased visual acuity, "cloudiness" of vision.
C. "Halos" around lights at night.
D. Neurologic complaints (headache, nausea, or vomiting).

OTHER SIGNS AND SYMPTOMS
A. Red eye with ciliary flush. Ciliary flush is a red or purple ring that surrounds the cornea which expands beyond the ring.
B. "Silent blinder" is a nickname used for glaucoma. It causes extensive damage before the client is aware of visual loss.
C. Dilated pupil.
D. Hard orbital globe.
E. No pupillary response to light.
F. Increased IOP (normal IOP is 10–20 mmHg).

SUBJECTIVE DATA
A. Review the onset, course, and duration of symptoms; note visual changes in one or both eyes. Do symptoms get worse in early evening?
B. Review medical history and medications, including over-the-counter (OTC) products.
C. Review family history of glaucoma.
D. Determine whether there has been any difficulty with peripheral vision, any headache photophobia, or any visual blurring.
E. Rule out presence of any chemical, trauma, or foreign bodies in the eye.
F. Review any recent history of herpes outbreak.
G. Ask the client whether this has ever occurred before, and if so how it was treated.
H. Determine if IOP has been measured in the past.

PHYSICAL EXAMINATION
A. Check blood pressure.
B. Inspect:
  1. Examine both eyes.
  2. Rule out foreign bodies.
  3. Inspect for redness, inflammation, and discharge.
  4. Check pupillary response to light.
  5. Note redness around the iris, if the pupil is dilated, and if the cornea appears cloudy.
  6. Inspect anterior chamber of eye by holding penlight laterally and directing toward nasal area. Shallow chamber will cast a shadow on the nasal side of the iris.
  7. Facial symmetry.
  8. Cranial nerves.
  9. Strength of upper/lower extremities.

▶

10. Funduscopic exam: This may reveal notching of the cup and a difference in cup-to-disc ratio between the two eyes.

C. Palpate:

1. Palpate the globe of the eye, which will feel firm on palpation.

DIAGNOSTIC TESTS

A. Check visual acuity and peripheral fields of vision.

B. Measure IOP with a tonometer. Normal level is 10 to 21 mmHg; IOP in acute angle-closure glaucoma is often 30 to 50 mmHg. Tonometer exam is not recommended if external infection is present.

C. Slit-lamp exam: edematous and/or cloudy cornea.

D. Gonioscopy (gold standard) using a slit lamp. Exam typically performed by optometrist or ophthalmologist.

DIFFERENTIAL DIAGNOSES

A. Acute angle-closure glaucoma.

B. Acute iritis.

C. Acute bacterial conjunctivitis.

D. Iridocyclitis.

E. Corneal injury.

F. Foreign body.

G. Herpetic keratitis.

H. Cerebrovascular accident (CVA).

PLAN

A. General interventions:

1. Severe attacks can cause blindness in 2 to 3 days. Same-day referral is critical if angle-closure glaucoma is suspected. Vision loss is irreversible. Treatment is aimed at preventing further vision loss**. Seek medical attention immediately to prevent permanent vision loss**.

2. Frequency of attacks is unpredictable.

3. All pressure changes greater than 21 mmHg need further evaluation from an ophthalmologist.

B. Client teaching: *See Client Teaching Guide for this chapter, "How to Administer Eye Medications."*

C. Pharmaceutical therapy:

1. Management **must be instituted by an ophthalmologist**. If evaluation and treatment by an ophthalmologist are projected to be greater than 1 hour, initial treatment to lower pressures should be considered with the following:

a. Timolol maleate 0.5%.
b. Pilocarpine (Pilocar) 2%.
c. Apraclonidine 1%.

D. Surgical intervention:

1. Surgery is indicated if IOP is not maintained within normal limits with medications or if there is progressive visual field loss with optic nerve damage.

2. Surgical treatment of choice is peripheral iridectomy: excision of a small portion of the iris whereby the aqueous humor can bypass the pupil.

3. Laser peripheral iridotomy: creates on opening in the peripheral iris to reduce pressures against the optic nerve.

FOLLOW-UP

A. Annual eye exams by an ophthalmologist are necessary to monitor IOP and treatment efficacy.

CONSULTATION/REFERRAL

A. All clients should be referred to an ophthalmologist *immediately* for measurement of IOP, acute management, and possible surgical intervention (laser peripheral iridectomy).

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. Females normally have slightly higher IOPs than males.

2. Asians may have higher IOPs than African Americans and Caucasians.

3. Individuals older than age 40 should have their IOP measured periodically. Every 3 to 5 years is sufficient after a stable baseline is established for the client.

B. Geriatrics:

1. Incidence increases with age, usually in those older than 60.

2. Medications that have anticholinergic properties may adversely affect clients with narrow-angle glaucoma. These medications are used to treat diseases such as the following:

a. Asthma/chronic obstructive pulmonary disease (COPD): ipratropium bromide (Atrovent) or tiotropium bromide (Spiriva).
b. Incontinence/overactive bladder: tolterodine (Detrol), fesoterodine (Toviaz), darifenacin (Enablex), solifenacin (Vesicare), and oxybutynin (Ditropan).
c. Gastrointestinal problems: cimetidine (Tagamet).
d. Muscle spasms: orphenadrine and trihexyphenidyl.
e. Depression: selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine (Prozac) and paroxetine (Paxil), and tricyclic antidepressants such as amitriptyline (Elavil) and imipramine (Tofranil).
f. Allergies: antihistamines OTC that contain diphenhydramine.
g. Nausea: promethazine (Phenergan).
h. Anxiety: hydroxyzine (Vistaril).
i. Colds: medications containing ephedrine.

3. Use caution when using medications containing sulfonamide-containing drugs, such as topiramate (Topamax), acetazolamide (Diamox Sequels), quinine (Qualaquin), tetracycline (Sumycin), and trimethoprim-sulfamethoxazole (Bactrim).

4. Chronic use of oral steroids may affect open-angle glaucoma and should be avoided if possible.

5. Long-term use of steroids may also increase the risk of developing cataracts.

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HORDEOLUM (STYE)

DEFINITION

A. Hordeolum is an infection of the glands of the eyelids (follicle of an eyelash or the associated gland of Zeis [sebaceous] or Moll gland [apocrine sweat gland]), usually caused by *Staphylococcus aureus*.

1. If swelling is under the conjunctival side of eyelid, it is an internal hordeolum. ▶

2. If swelling is under the skin of the eyelid, it is an external hordeolum.

INCIDENCE

A. The incidence is unknown; it is more common in children and adolescents than in adults.

B. Clients with recurrent blepharitis, seborrheic dermatitis, rosacea, diabetes, and hyperlipidemia are at an increased risk of hordeola development.

PATHOGENESIS

A. Acute bacterial infection of the meibomian gland (internal hordeolum) or of the eyelash follicle (external hordeolum) is usually caused by *S. aureus*.

PREDISPOSING FACTORS

A. Age: commonly seen in adults.

B. Meibomian gland dysfunction.

C. Rosacea.

COMMON COMPLAINTS

A. Eye tenderness.

B. Sudden onset of a purulent discharge.

C. Painful bump on the edge of the eyelid.

OTHER SIGNS AND SYMPTOMS

A. Redness and swelling of the eye.

SUBJECTIVE DATA

A. Review the onset, course, and duration of symptoms.

B. Determine whether there is any visual disturbance.

C. Note whether this is the first occurrence. If not, ask how it was treated before.

D. Evaluate how much pain or discomfort the client is experiencing.

E. Review the client's history for chemical, foreign body, and/or trauma etiology.

F. Review the client's medical history and medications.

G. Does the client have a history of rosacea or frequent meibomian gland dysfunction?

PHYSICAL EXAMINATION

A. Inspect:

1. Examine both eyes; note redness, site of swelling, and amount and color of discharge.

2. Evert the lid and check for pointing.

3. Assess sclera and conjunctivae for abnormalities.

4. Inspect ears, nose, and throat.

B. Palpate:

1. Palpate eye for hardness and expression of discharge.

2. Evaluate for preauricular adenopathy.

DIAGNOSTIC TESTS

A. Test visual acuity.

B. Discharge can be cultured but is usually treated presumptively.

DIFFERENTIAL DIAGNOSES

A. Hordeolum.

B. Chalazion: the main differential diagnosis, which points to the conjunctival side of the eyelid and does not usually affect the margin of the eyelid.

C. Blepharitis.

D. Xanthoma.

E. Bacterial conjunctivitis.

F. Foreign body.

G. Basal cell carcinoma/sebaceous cell carcinoma.

PLAN

A. General interventions:

1. Most lesions will drain without any treatment. Warm compress and massage to the area may help facilitate drainage.

2. Lid scrubs with a mild baby shampoo may promote drainage.

3. Larger, persistent lesions may require antibiotic therapy. Contain the infecting pathogen. Crops occur when the infectious agent spreads from one hair follicle to another.

B. Client teaching: *See Client Teaching Guide for this chapter, "How to Administer Eye Medications."*

1. Apply warm compress to eye 10 to 15 minutes four times a day for comfort.

2. Instruct on proper eyelid hygiene and good handwashing.

3. Clients should discard all eye makeup, including mascara, eyeliner, and eye shadow.

C. Pharmaceutical therapy:

1. If hordeolum does not resolve with warm compress within 5 days or if multiple sites are infected:

a. Erythromycin ophthalmic 0.5% ointment 1 cm to affected eye up to six times daily.

b. If crops of sties occur, some clinicians recommend a course of tetracycline to stop recurrences (consult with a physician).

c. If cellulitis of the surrounding tissue is suspected, oral antibiotics that cover *Staphylococcus* are recommended. Oral antibiotics recommended include cephalexin and erythromycin.

FOLLOW-UP

A. Have client telephone or visit the office in 48 hours to check response.

B. If crops occur, diabetes mellitus must be excluded. Perform blood glucose evaluation.

C. Recommended follow-up appointment in 2 weeks.

CONSULTATION/REFERRAL

A. Hordeolum may produce a diffuse superficial lid infection, preseptal cellulitis, requiring referral to an ophthalmologist.

B. If hordeolum does not respond to topical antimicrobial treatment, refer the client to an ophthalmologist for possible incision and drainage if indicated.

C. Recurrent lesions should be referred to an ophthalmologist for further evaluation and treatment (biopsy) for possible basal cell carcinoma or sebaceous cell carcinoma.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. Recurrent lesions should be referred to a specialist for evaluation and workup for possible carcinoma.

2. Hordeolums are benign infections and usually respond in 1 to 2 weeks with proper treatment. If lesion has not resolved, further workup is recommended.

3. Address underlying conditions such as rosacea, frequent blepharitis, uncontrolled diabetes, and high cholesterol to prevent recurrent hordeolum in these clients.

B. Geriatrics:

1. Nonpharmacologic treatments include warm compress for 20 to 30 minutes (rewetting washcloth every 5–10 minutes), applying gentle massage, discontinuing wearing contact lens and eye makeup until healed, and encouraging clients to keep hands clean and to not rub their eyes. ▶

2. Pharmacologic treatments common in geriatrics include short-term nonsteroidal anti-inflammatory drugs (NSAIDs) for pain and inflammation and Tobradex ointment three to four times a day for 1 week (combination of an antibiotic and an anti-inflammatory), which would be contraindicated if coexistent with fungal or viral ocular infection.

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MACULAR DEGENERATION

DEFINITION

A. Macular degeneration (MD) is the result of age-related changes of the macula (central retina) that result in loss of central vision. MD is the leading cause of blindness in the United States.

INCIDENCE

A. The risk of MD increases with age, with risk starting as early as age 50. It is estimated that MD will affect 2.95 million people by 2025. Currently, 25% of people 75 years of age have some degree of MD and 14% of people older than the age of 84 years struggle with visual loss due to MD. With progressive MD in one eye, the opposite eye has greater than 40% risk of visual loss within 5 years.

PATHOGENESIS

A. There are two types of MD:

1. Dry type (nonneovascular) is secondary to retinal atrophy. The initial cause of the atrophic changes is unclear.

2. Wet type (neovascular) is secondary to abnormal vascular growth in the subretinal space. Immune-mediated inflammation is believed to play a major role in MD.

PREDISPOSING FACTORS

A. Age.
B. Smoking.
C. Family history.
D. Cardiovascular disease.
E. Dietary fat.
F. Cataract surgery.
G. Aspirin use.
H. Excessive alcohol intake (greater than three drinks per day).
I. White race.
J. Obesity.

COMMON COMPLAINTS

A. Gradual loss of vision in one or both eyes.
B. Scotoma (dark patch in the center of the visual field).
C. Distorted straight edges (appearing curved or irregular).

POTENTIAL COMPLICATIONS

A. Blindness resulting in functional impairments.
B. Depression secondary to visual loss.
C. Isolation.

SUBJECTIVE DATA

A. Review the onset, duration, and course of symptoms.
B. Was the change in vision sudden or gradual?
C. Ask if vision loss was unilateral or bilateral?
D. Was distance or near vision affected?
E. Determine family history of MD.
F. Review history of eye disorders and previous eye surgeries or treatments.
G. Review date of last eye exam, including dilation.
H. Review medical history, including current medications.
I. Determine the amount of change in activities of daily living (ADL) due to visual impairment.
J. Ask about perceived safety concerns and history of falls.

PHYSICAL EXAMINATION

A. Check vital signs, including blood pressure.
B. Inspect:

1. Symmetry of eyes and obvious deformities.

2. Pupil symmetry and response to light and accommodation.

3. Assessment of fields of vision and extraocular movements.

4. Funduscopic assessment observing for drusen bodies.

5. Snellen and Rosenbaum screening.

6. Observation of entropion or ectropion.

7. Observation of pterygium.

DIAGNOSTIC TESTS

A. Imaging of dry eye MD (performed by an ophthalmologist or an optometrist).

1. Fluorescein angiogram.

2. Optical coherence tomography.

3. Indocyanine green angiography.

4. Autofluorescence.

B. Imaging of wet eye MD (performed by an ophthalmologist or an optometrist).

1. Fluorescein angiography.

2. Indocyanine green angiography.

3. Optical coherence tomography.

DIFFERENTIAL DIAGNOSES

A. MD.
B. Presbyopia.
C. Cataracts.
D. Glaucoma.
E. Diabetic retinopathy.

PLAN

A. General interventions:

1. Referral to eye specialist for formal diagnosis and prescription for corrective lenses.

2. Discussion of eye health, including routine eye exams and prevention of injury.

B. Client teaching:

1. Prevention: Encourage a healthful diet of fruit, green leafy vegetables, fish, and nuts.

2. Discuss the importance of antioxidants, vitamin C 500 mg, vitamin E 400 IU, lutein 10 mg, zeaxanthin 1 mg, zinc oxide 80 mg, cupric oxide 2 mg, zeaxanthin 2 mg, and omega-3 fatty acids 1,000 mg. These supplements are thought to retard the progression of MD. Beta carotene has been removed from the list of recommendations due to the increased risk of lung cancer, particularly in smokers.

3. Stress the importance of routine eye exams. ▶

4. Emphasize the need to control blood sugar if diabetic and blood pressure if hypertensive.
5. Stress the importance of eye protection, including sunglasses.
6. Healthy lifestyle choices, including an active lifestyle and smoking avoidance, reduce the risk of MD.

C. Pharmaceutical therapy:
1. Ranibizumab, aflibercept, or bevacizumab intravitreal injections may be used by a specialist for treatment.

CONSULTATION/REFERRAL

A. Referral to an optometrist or ophthalmologist is required for accurate diagnosis and treatment.

INDIVIDUAL CONSIDERATIONS

A. Adults:
1. Corrective lenses, additional visual aids, good lighting, and visual rehab should all be considered to reduce functional impairments and isolation from MD.
2. Eye exams are covered, under certain circumstances, by Medicare. Clients can receive up-to-date coverage information at Medicare.gov.

B. Geriatrics:
1. The following link is a web-based age-related macular degeneration (AMD) update page which emphasizes the geriatric's point of view regarding AMD and how practitioners may help meet clients' needs and create shared-decision care plans: www.webrn-maculardegeneration.com/.
2. Current research posits that use of anticholinergic medication for at least 3 months is associated with an increase of AMD (as evidenced by a high Anticholinergic Burden Score). The suggested component contributing to primary lesions of AMD was increase of amyloid-beta deposits from anticholinergic use.
3. Geriatric clients who suffer from AMD have double the rate of depression compared with those without the disease. Evaluate for anxiety, depression, and quality of life issues every annual visit and treat appropriately.
4. Review safety precautions with driving, ambulating, and within the home environment, and examine the ability to perform ADLs at home/work or activities such as shopping, sports, and so forth.
5. Promote dietary modifications that research has suggested to prevent/improve AMD: water-rich foods, vitamin C, zinc oxide, lutein, and fish oil.
6. Vascular endothelial growth factor (VEGF) inhibitors (Avastin and Lucentis) reduce neovascularization, are monthly treatments, and might maintain vision for up to 2 years. Complications include uveitis, increased intraocular pressures, cataracts, and retinal detachment.

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REFRACTIVE DISORDERS

DEFINITION

A. Clear vision occurs when the cornea and the crystalline lens bend (refract) light appropriately onto the retina. Refractive errors occur when any component of the optical system malfunctions. Refraction errors are common and correctable. Refractive errors include ametropia (including myopia, hyperopia, astigmatism) and presbyopia.

INCIDENCE

A. Refractive disorders affect more than one-third of people 40 years or older. Myopia and astigmatism are the two most prevalent refractive disorders. Myopia (nearsightedness) is more common in Caucasians and Asians. Astigmatism and presbyopia increase with aging.

PATHOGENESIS

A. Emmetropia (normal refraction) begins in childhood and continues into early adulthood. The etiology of poorly developed emmetropization is unknown.

PREDISPOSING FACTORS

A. Myopia:
1. Genetic predisposition.
2. Reading for long periods.
3. Medications (sulfonamides, diuretics, and cholinergics), the adverse effects of which are reversible with discontinuing medication.
4. Diabetes.
5. Trauma.
6. Excessive accommodation.
7. Cataracts.
8. Older maternal age at birth or maternal smoking.

B. Hyperopia:
1. Trauma.
2. Anticholinergic medications.

COMMON COMPLAINTS

A. Fear of driving or performing usual, common activities due to decreased visual acuity.

POTENTIAL COMPLICATIONS

A. Motor vehicle accidents.
B. Increased risk of falls and hip fractures.
C. Isolation due to fear of leaving familiar environment.

SUBJECTIVE DATA

A. Review the onset, duration, and course of symptoms.
B. Was the change in vision sudden or gradual?
C. Determine family history of refractive disorders.
D. Review history of eye disorders and previous eye surgeries or treatments.
E. Review date of last eye exam, including dilation.
F. Review medical history, including current medications and history of diabetes.
G. Determine if client has used over-the-counter (OTC) corrective lenses and duration of use.
H. Has there been a change in vision associated with headaches?
I. Ask about the amount of up-close work, including reading, computer use, factory assembly, and sewing.
J. Determine the amount of change in activities of daily living (ADLs) due to visual impairment.
K. Ask about perceived safety concerns and history of falls.

PHYSICAL EXAMINATION

A. Check vital signs, including blood pressure.
B. Inspect:
1. Symmetry of eyes and obvious deformities.
2. Pupil symmetry and response to light and accommodation.
3. Assess fields of vision and extraocular movements.
4. Funduscopic assessment.
5. Snellen and Rosenbaum screening.
6. Observe for entropion or ectropion.
7. Observe for pterygium.

DIAGNOSTIC TESTS

A. Phoropter (performed by an optometrist or ophthalmologist).

DIFFERENTIAL DIAGNOSES

A. Cranial nerve dysfunction (cranial nerves II, III, and VI).
B. Transient ischemic attack (TIA)/cerebrovascular accident (CVA).
C. Trauma (if sudden unilateral reduction in acuity).
D. Glaucoma.
E. Space-occupying intracranial lesion.
F. Age-related macular degeneration.
G. Diabetic retinopathy.
H. Cataracts.

PLAN

A. General interventions:
1. Refer to an eye specialist for formal diagnosis and prescription for corrective lenses.
2. Discuss eye health, including routine eye exams and prevention of injury.
B. Client teaching:
1. Importance of routine eye exam.
2. Importance of control of blood sugars if diabetic and blood pressure if hypertensive.
3. Importance of eye protection, including quality sunglasses.

CONSULTATION/REFERRAL

A. Referral to an eye specialist is required for accurate diagnosis following initial eye screening.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Increase in estrogen and progesterone in pregnancy is associated with vision change of myopia or hyperopia.
2. Advanced maternal age, higher parity, regular television watching, and computer use contribute to refractory changes seen.
B. Adults:
1. Refraction errors are more common with aging, especially myopia, presbyopia, and astigmatism. At 40 years of age, clients should expect presbyopic changes.
2. Diabetic retinopathy, although not a refractive error, is a leading cause of blindness. Clients should receive an annual ophthalmic exam.
3. Eye exams are covered under certain circumstances by Medicare. Clients can receive up-to-date coverage information at Medicare.gov.
C. Geriatrics:
1. Safe driving: age-related vision changes to evaluate and discuss with older adults ≥60 years old:
a. Ability to see road sign clearly decreases.
b. The vehicle's instrument panel becomes blurry and it is difficult to quickly adjust from road to panel.
c. Challenges in judging distances.
d. Color perception changes.
e. Night vision ability decreases.
f. Pain and difficulty adjusting to sunlight/headlights.
g. Peripheral vision loss.
2. Avoid wearing eyeglasses or sunglasses with wide frames or side-arm temples that could restrict vision.
3. Suggest handheld or stand magnifiers when viewing labels or doing tasks such as sewing, puzzles, nail hygiene, and so forth. Consider video magnification for computers/monitors. Avoid overuse of eyes, causing "tired eyes." Consider using talking timepieces, audio books, and apps that read aloud internet news and social media comments.
4. Allow geriatric clients to express feelings of vision loss and assist with eyesight solutions to assure safety and quality of life.

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RETINOPATHY

DEFINITION

A. Retinopathy is damage of retinal blood vessels most commonly due to pathology secondary to hyperglycemia (diabetic retinopathy). There are two major categories of retinopathy: nonproliferative and proliferative. Retinopathy is further classified into nonproliferative stages—mild, moderate, severe, and very severe—and proliferative stages—early, high risk, and severe. Symptoms do not present until late in the disease process. Retinopathy is a risk marker of higher rates of morbidity and mortality from cardiovascular disease.

INCIDENCE

A. Diabetic retinopathy is the leading cause of blindness of adults between the ages of 25 and 74 years, occurring in 77.3% of those with type 1 diabetes and 25.1% of those with type 2 diabetes.

PATHOGENESIS

A. Nonproliferative retinopathy is due to nerve fiber damage, retinal hemorrhages, microaneurysms, or tortuous/dilated retinal vessels.
B. Proliferative retinopathy is due to neovascularization vessel growth that produces harmful changes in the retinal bed, including preretinal or vitreous hemorrhages, fibrosis, and retinal detachment.
C. Macular edema may occur during any stage of retinopathy.

PREDISPOSING FACTORS
A. Diabetes, including gestational diabetes.
B. Uncontrolled hypertension (HTN).
C. Smoking.
D. Chronic kidney disease.
E. Dyslipidemia.

COMMON COMPLAINTS
A. Visual impairments are not common until pathologic changes are well established.
B. "Curtain falling" or floaters may be mentioned in vitreous hemorrhage.

POTENTIAL COMPLICATIONS
A. Loss of independence due to visual impairments.
B. Loss of employment.
C. Motor vehicle accidents.
D. Isolation due to fear of leaving familiar environment.
E. Depression.

SUBJECTIVE DATA
A. Review the onset, duration, and course of symptoms.
B. Was the change in vision sudden or gradual?
C. Discuss home glucose monitoring and results.
D. Review history of eye disorders and previous eye surgeries or treatments.
E. Review date of last eye exam, including dilation.
F. Review medical history, including current medications and year of onset of diabetes or HTN.
G. Determine if client has used over-the-counter (OTC) corrective lenses for self-treatment and determine duration of use.
H. Determine the level of change in activities of daily living (ADLs) due to visual impairment.

PHYSICAL EXAMINATION
A. Check vital signs, including blood pressure.
B. Inspect:
1. Symmetry of eyes and obvious deformities.
2. Pupil symmetry and response to light and accommodation.
3. Assessment of fields of vision and extraocular movements.
4. Funduscopic assessment.
5. Snellen and Rosenbaum screening.

DIAGNOSTIC TESTS
A. Funduscopic exam, dilated.
B. Fluorescein angiography (performed by an optometrist or ophthalmologist).
C. Hemoglobin A1c.

DIFFERENTIAL DIAGNOSES
A. Diabetic retinopathy.
B. Hypertensive retinopathy.

PLAN
A. General interventions:
1. Identify the possible diagnosis of retinopathy and refer to an eye specialist for formal diagnosis.
2. Panretinal laser photocoagulation for proliferative retinopathy (ophthalmology).
3. Vitreous surgery (ophthalmology).
4. Clients diagnosed with diabetes are encouraged to improve their glucose levels.
B. Client teaching:
1. Advise the client on the importance of routine eye exams.
2. Discuss the importance of good control of blood sugar if diabetic and blood pressure if hypertensive.
3. Lifestyle modifications: smoking cessation, weight loss, dietary changes, reduction of alcohol.
4. Proliferative diabetic retinopathy clients need to avoid any exercise that may raise blood pressure due to increased risk of retinal hemorrhage or detachment.
C. Pharmaceutical therapy:
1. None. The client is followed by an ophthalmologist.

CONSULTATION/REFERRAL
A. Referral is required for accurate diagnosis following initial funduscopic screening.

INDIVIDUAL CONSIDERATIONS
A. Adults:
1. Retinopathy is the leading cause of blindness between the ages of 25 and 74. Clients should receive an annual ophthalmic exam.
2. Retinopathy can be slowed based on the stage of progression. Early diagnosis is key to sight-saving intervention.
B. Geriatrics:
1. The highest risk factors for older adult retinopathy are uncontrolled diabetes, uncontrolled HTN, and smoking. Retinopathy changes can start when client is in the prediabetic phase of Type 2 diabetes
2. Vascular endothelial growth factor (VEGF) inhibitors are becoming first-line treatment for retinopathy. These medications (Avastin and Lucentis) can cause macular edema (swelling of the central part of the eye), which is associated with retinopathy (ischemia and vitreous hemorrhage).
3. Manage glycemic levels specifically related to comorbidities and the client's life expectancy. (Individuals in hospice have less stringent glycemic control.) Keep client's blood pressure ≤140/80.

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SUBCONJUNCTIVAL HEMORRHAGE

DEFINITION
A. Subconjunctival hemorrhage presents as blood patches in the bulbar conjunctiva.

INCIDENCE
A. Subconjunctival hemorrhage may be seen in adults after forceful exertion (coughing, sneezing, childbirth, strenuous lifting). Subconjunctival hemorrhages account for approximately 3% emergent visits in the United States, and males have a higher prevalence than females. The true incidence is unknown secondary to many cases not being reported.

PATHOGENESIS
A. Subconjunctival hemorrhage occurs when the small blood vessels in the conjunctiva rupture from force or injury. When this occurs, the blood leaks out from the vessel, causing a red staining of the sclera. This may appear as a dark red spot on ▶

the white part of the eye. This disorder is believed to be secondary to increased intrathoracic pressure that may occur from forces such as pushing during labor and delivery, vomiting, excessive coughing, or with physical exertion.

PREDISPOSING FACTORS

A. Local trauma.
B. Systemic hypertension (HTN).
C. Acute conjunctivitis.
D. Vaginal delivery.
E. Severe coughing.
F. Severe vomiting.
G. Long-term hemodialysis.

COMMON COMPLAINT

A. Red-eyed appearance without pain.

OTHER SIGNS AND SYMPTOMS

A. Bright red blood in plane between the conjunctiva and sclera.
B. Usually unilateral.
C. Normal vision.

SUBJECTIVE DATA

A. Identify onset and duration of symptoms.
B. Elicit information about trauma to the eye; is it due to severe coughing or vomiting?
C. Identify history of conjunctivitis or HTN.
D. Review medications for anticoagulant or antiplatelet use, including over-the-counter (OTC) herbal products.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure (rule out HTN).
B. Inspect:
  1. Observe eyes.
  2. Inspect ears, nose, and mouth.
  3. Inspect skin for bruises, petechiae, or other trauma.
  4. Assess for signs of trauma or abuse. **Blood in the anterior chamber (hyphema) can result from injury or abuse**.
C. Percuss:
  1. Percuss abdomen for hepatosplenomegaly.
E. Other physical examination components are dependent on etiology.

DIAGNOSTIC TESTS

A. Perform visual screening.
B. Test extraocular movements (EOMs) and peripheral vision.

DIFFERENTIAL DIAGNOSES

A. Subconjunctival hemorrhage.
B. Systemic HTN.
C. Blood dyscrasia.
D. Trauma to eye.
E. Conjunctivitis.
F. Hyphema.
G. Abuse.

PLAN

A. General interventions:
  1. Reassure the client that the hemorrhage is not damaging to the eye or vision and that the blood reabsorbs on its own over several weeks.
  2. A slit-lamp exam may be performed to rule out any ocular trauma.
B. Client teaching:
  1. Educate the client regarding safety to prevent trauma to the eye.
C. Pharmaceutical therapy:
  1. None.

FOLLOW-UP

A. If subconjunctival hemorrhage recurs, evaluate the client further for systemic HTN or blood dyscrasia.

CONSULTATION/REFERRAL

A. Consult or refer the client to a physician if hyphema is noted, if glaucoma is suspected, or if the client has additional eye injuries.

INDIVIDUAL CONSIDERATIONS

A. Adults:
  1. Always measure blood pressure to rule out systemic HTN.
B. Geriatrics:
  1. Research indicates that the common causes of elderly subconjunctival hemorrhage are HTN and use of aspirin. It occurs most often with geriatric males and primarily observed in the nasal region of the eye.
  2. Always measure blood pressure to rule out systemic HTN.
  3. Consider evaluation for blood dyscrasia.
  4. Assess clotting times if client is taking warfarin (Coumadin) or other products interfering with coagulation.

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UVEITIS

DEFINITION

A. Uveitis, also known as iritis, is inflammation of the uveal tract (iris, ciliary body, and choroid) and is usually accompanied by a dull ache and photophobia due to irritative spasm of the pupillary sphincter.

INCIDENCE

A. The true incidence is unknown. Approximately 15% of clients with sarcoidosis present with uveitis. Of all cases 48% to 70% are considered idiopathic.
B. Older adults, females, smokers, and those with gastrointestinal (GI) inflammatory diseases have a higher prevalence of uveitis.

PATHOGENESIS

A. The cause is unknown. Underlying causes include infections, viruses, and arthritis.

PREDISPOSING FACTORS

A. Collagen disorders.
B. Autoimmune disorders.
C. Ankylosing spondylitis.
D. Sarcoidosis.
E. Juvenile rheumatoid arthritis.

F. Lupus.
G. Reiter syndrome.
H. Behcet syndrome.
I. Syphilis.
J. Tuberculosis.
K. AIDS.
L. Crohn disease.
M. Lyme disease.
N. Cerebral vasculitis.

COMMON COMPLAINTS

A. Eye pain: painless to deep-seated ache.
B. Photophobia.
C. Blurred vision with decreased visual acuity.
D. Black spots.
E. Eye redness.

OTHER SIGNS AND SYMPTOMS

A. Unilateral or bilateral symptoms:
1. Unilateral: The pupil is smaller than that of the other eye due to spasm.
B. Ciliary flush.
C. Pupillary contraction.
D. Nausea and vomiting with vagal stimulation.
E. Halos around lights.
F. Hypopyon (pus in anterior chamber).
G. Limbal flush with small pupil.

SUBJECTIVE DATA

A. Elicit the onset, course, duration, and frequency of symptoms. Are symptoms bilateral or unilateral?
B. Identify the possible causal activity or agent (chemical, traumatic, or infectious etiologies).
C. Review the client's history of uveitis and other ophthalmologic disorders.
D. Review any associated fever, rash, headache, weight loss, joint pain, back pain, oral ulcers, or genital ulcers.
E. Review full medical history for comorbid conditions.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.
B. Inspect:
1. Assess both eyes for visual acuity and peripheral fields of vision.
2. Check sclera and conjunctiva.
C. Other physical and mental health assessments need to be completed related to comorbid conditions.

DIAGNOSTIC TESTS

A. Slit-lamp test: Slit-lamp examination reveals cells in the anterior chamber and "flare," representing increased aqueous humor protein. Inflammatory cells, called keratic precipitates, can collect in clusters on the posterior cornea.
B. Penlight examination: Flashlight examination shows a slightly cloudy anterior chamber in the uveitic eye.

DIFFERENTIAL DIAGNOSES

A. Uveitis: usually idiopathic, but may be associated with many systemic and ocular diseases.
B. Acute angle-closure glaucoma.
C. Retinal detachment.
D. Central retinal artery occlusion.
E. Endophthalmitis.

PLAN

A. General interventions:
1. Treat underlying cause as indicated.
2. Provide immediate referral to an ophthalmologist due to possible complications of cataracts and blindness.
B. Client teaching:
1. Inform the client that recurrent attacks are common and also require immediate attention.
2. Advise the client to notify the provider if eye pain, change of vision, or other new symptoms occur.
C. Pharmaceutical therapy:
1. Medications are given by an ophthalmologist.
2. Uveitis and colitis often flare simultaneously; oral steroids are effective for both.

FOLLOW-UP

A. A client with uveitis needs a follow-up with an ophthalmologist.

CONSULTATION/REFERRAL

A. The client should be referred *immediately* to an ophthalmologist for evaluation and intervention.

INDIVIDUAL CONSIDERATIONS

A. Adults:
1. Recurrent uveitis may be a sign of another systemic condition. Other conditions include infections (bacterial, spirochetal, viral, fungal, and parasitic infections), inflammatory diseases including spondyloarthropathies (ankylosing spondylitis, psoriatic arthritis, reactive arthritis), inflammatory bowel disease, multiple sclerosis, and the use of new medications. Further workup should be performed for recurrent uveitis.
B. Geriatrics:
1. Research has indicated that uveitis is strongly associated with immunosuppression and autoimmunity disorders such as ankylosing spondylitis, gout, psoriasis, and rheumatic diseases, which are prevalent among the geriatric population.
2. With noninfectious/treatment-resistant uveitis, refer to an ophthalmologist/rheumatologist for collaborative management. Monoclonal tumor necrosis factor inhibitors (TNFi) such as adalimumab (Humira) and infliximab (Remicade) are demonstrating safety and efficacy with no statistically significant differences.
3. A natural possible treatment for uveitis is taking a multivitamin.
4. Difficult-to-treat and nonresponding uveitis can be treated by ophthalmology with slow-release implantable steroid.

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CLIENT TEACHING GUIDE

CONJUNCTIVITIS

PROBLEM You have an infection of the eye, or conjunctivitis, which causes redness, itching, drainage from the eye, and crusting on the eyelids.

CAUSE Bacteria, viruses, or allergies can cause eye infections.

PREVENTION

A. Wash your hands frequently.
B. Avoid persons with conjunctivitis, such as pinkeye.
C. Avoid known allergens.

TREATMENT PLAN

A. All types:
  1. Wash your hands frequently, especially after touching the eyes, to avoid spread.
  2. Use cool compress on the eyes as needed.
  3. Wash crusting eyelids with baby shampoo daily.
  4. Wipe the eyes from inner to outer corners.
B. Bacterial: Bacterial conjunctivitis is contagious up to 24 hours after starting medication.
C. Viral: Viral conjunctivitis is contagious for 48 to 72 hours, but may last up to 2 weeks.

Activity: As tolerated.

Diet: As tolerated.

Medications: No medications are prescribed for viral infections. You will be given instructions on how to use eye drops or eye ointment.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. A reaction to your medication.
B. Trouble seeing.
C. New symptoms, including pain in your eye or increased redness.
D. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

HOW TO ADMINISTER EYE MEDICATIONS

PROBLEM You have been prescribed a medication for your eye(s). It is very important that you know the correct way to use your eye medication.

CAUSE You have been diagnosed with_____.

PREVENTION The health of your eyes is important.

A. Use good handwashing techniques and try not to rub your eyes with your fingers.
B. Clean your contact lenses regularly with contact cleaning solution. Do not put your contacts in your mouth to moisten. Change and remove your contacts as recommended by the manufacturer.
C. Wear sunglasses in bright sunshine.
D. Use eye goggles when working and playing sports to ensure extra protection.
E. Change your eye makeup often. Mascara, eye shadow, and eyeliner grow bacteria. Do not share makeup.

TREATMENT PLAN

A. Correct use of your medication is important.
B. You may or may not require an eye patch or shield.

How to Apply Eye Ointment

A. Always wash your hands before placing medication in your eyes.
B. Gently pull down the lower eyelid.
C. Make a small pocket between the eyeball and the eyelid.
D. If you have someone helping to put your eye ointment in the lower lid pocket, look up and away while they put in the medicine.
E. Do not let the tube of medicine touch the eye or eyelid.
F. Squeeze a thin ribbon of the medication into the pocket of the eyelid.
G. Start at the inner fold of the eye going from the nose to the outer eye.
H. Let go of the eyelid and blink to spread the medication.

How to Instill Eye Drops

A. Always wash your hands before placing medication in your eyes.
B. Gently pull down the lower eyelid.



FIGURE 1 How to instill eye drops into the eye.

(*continued*)

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CLIENT TEACHING GUIDE

HOW TO ADMINISTER EYE MEDICATIONS (*CONTINUED*)

C. Make a small pocket between the eyelid and the eyeball.
D. If you have someone helping to put eye drops in the lower lid pocket, look up and away while they put in the medicine.
E. Do not let the bottle of medication touch the eye or eyelid.
F. Squeeze the prescribed number of drops of the medicine into the pocket of the eyelid.
G. Let go of the eyelid and blink (or tell the client to blink) to spread the medication (Figure 1).

Activity: No restrictions are required unless you require eye surgery; then you will be given specific instructions about the amount of activity allowed.

Diet: No restrictions.

Medications: _____

You Have Been Prescribed: _____

You Need to Use the Medicine: _____

You Need to Notify the Office If:
A. You are unable to put in the medication yourself or get help from others.
B. You are not better 24 to 48 hours after starting the medication.
C. Your vision is worse after using the medication.
D. You have an allergic reaction to the medicine.
E. Other: _____

Phone: _____

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CHAPTER 9

EAR GUIDELINES

Jill C. Cash and Emily Y. Brignola

ACUTE OTITIS MEDIA

DEFINITION

A. Acute otitis media (AOM) is inflammation of the middle ear associated with an acute bacterial infection of the middle ear. There is usually a rapid onset of symptoms.

INCIDENCE

A. AOM commonly occurs in children ages 12 months or less, but it may also be seen in adults and geriatric clients.

PATHOGENESIS

A. Obstruction of the eustachian tube can lead to middle ear effusion and infection. Contamination of this middle ear fluid often results from a backup of nasopharyngeal secretions. The most common bacterial pathogens in adults are *Streptococcus pneumoniae* and *Haemophilus influenzae*.

PREDISPOSING FACTORS

A. Recurrent otitis media (three or more episodes in the past 6 months).
B. Previous episode of otitis media or upper respiratory infection (URI) within the past month.
C. Medical condition that predisposes to otitis media (e.g., Down syndrome, AIDS, cystic fibrosis, cleft palate, immune disorders, and craniofacial abnormalities).
D. Native American/Inuit heritage.
E. Exposure to tobacco smoke and air pollution.
F. History of allergies.

COMMON COMPLAINTS

A. Otalgia.
B. Decreased or muffled hearing.
C. Ear fullness.
D. Fever may or may not be present.

OTHER SIGNS AND SYMPTOMS

A. Sleeplessness within past 48 hours.
B. Decreased appetite.
C. Acute hearing loss.
D. URI symptoms.
E. Mastoiditis presenting with a swollen and red mastoid.
F. Perforated tympanic membrane (TM; sudden severe pain followed by immediate relief of pain with fluid drainage from the ear).
G. Cholesteatoma (saclike structure in the middle ear accompanied by white, shiny, greasy debris).

SUBJECTIVE DATA

A. Elicit the onset and duration of symptoms.
B. Inquire whether the client recently had (or has concurrently) a URI.
C. Determine whether the client has any change in hearing.
D. Assess the client for any otorrhea (ear drainage) from the ear(s).
E. Question the client or their caregiver regarding risk factors.
F. Identify the client's history of otitis media.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.
B. Inspect:
  1. Observe the canal and auricle for redness, deformity, drainage, or foreign body.
  2. Inspect the TM position to determine if it is neutral and whether landmarks are visible, retracted, full, or bulging.
  3. Observe ears for decreased or absent TM mobility (with pneumatic otoscope).
  4. Inspect nose, mouth, and throat.
C. Auscultate:
  1. Auscultate heart and lungs.

DIAGNOSTIC TESTS

A. Tympanometry is a test that measures how easily the TM vibrates. Tympanogram shows flat or type B curve.
B. A diagnostic audiogram should be done in clients with persistent otitis media (≥3 months' duration).
C. Consider complete blood count if the client appears toxic with a high fever.

DIFFERENTIAL DIAGNOSES

A. Chronic otitis media.
B. Otitis media with effusion (OME).
C. URI.
D. Mastoiditis.
E. Otitis externa.
F. Eustachian tube dysfunction (ETD).
G. Cholesteatoma (saclike structure in the middle ear usually accompanied by chronic foul-smelling ears, otorrhea, and progressive hearing loss).
H. Spontaneous TM rupture.
I. Herpes zoster infection.
J. Other deep space head/neck infections.

The contributions of Kathleen Bradbury-Golas to this chapter in prior editions are acknowledged here.

PLAN

A. General interventions:

1. Pain relief with acetaminophen or ibuprofen. Auralgan may be used for a topical pain relief.

B. Client teaching:

1. Stress the importance of taking antibiotics as prescribed and finishing the prescription as ordered. If pain, fever, or other symptoms do not improve in 48 to 72 hours, then the client should notify the provider for further evaluation.

C. Pharmaceutical therapy:

1. Drug of choice: First-line therapy for adults with mild infection is amoxicillin-clavulanate (Augmentin) 875 to 125 mg orally BID. For severe infection (fever, severe pain, hearing loss, etc.) or those with infections resistant to *S. pneumoniae,* amoxicillin 1,000 mg with clavulanate 62.5 mg, extended-release, orally BID (for lower weight clients and/or those with milder infections), OR amoxicillin 2,000 mg with clavulanate 125 mg, extended-release, orally BID (for higher weight clients and/or those with more severe infections), is recommended.

2. If amoxicillin-clavulanate cannot be used, amoxicillin 500 mg orally TID or 875 mg orally BID may be used as an alternative first-line therapy.

3. For penicillin allergy, cephalosporins are used.

a. Cefdinir 300 mg orally Q12H or 600 mg orally daily for 5 to 7 days.

b. Cefpodoxime 200 mg orally Q12H for 5 to 7 days.

c. Cefuroxime 500 mg orally Q12H for 5 to 7 days.

d. Ceftriaxone 1 to 2 g intravenously or 1 g intramuscularly once daily for 3 days.

4. Other alternatives for clients known to have severe allergy to beta-lactam antibiotics include doxycycline or a macrolide.

a. Doxycycline 100 mg Q12H.

b. Clarithromycin 500 mg orally Q12H.

c. Azithromycin 500 mg orally as a single dose on day 1, then 250 mg orally on days 2 to 5 or clarithromycin 500 mg orally BID for 5 to 7 days.

5. Trimethoprim-sulfamethoxazole is not used due to high rate of resistance among *H. influenzae* and *S. pneumoniae* and because it is not effective against group A *Streptococcus*. Similarly, clindamycin is not used because it has no activity against *H. influenzae.*

6. Antibiotic treatment is recommended for all adults and geriatric clients diagnosed with AOM.

FOLLOW-UP

A. Recheck the client in 2 weeks to evaluate for resolution of the infection. Documentation of the resolution of the ear infection is valuable information if infections are recurrent.

B. If fever, pain, hearing loss, or other new symptoms present or if the client did not improve in 48 to 72 hours, then a follow-up appointment is recommended.

CONSULTATION/REFERRAL

A. Consult or refer the client to a specialist (otolaryngologist) if they appear septic or are diagnosed with mastoiditis.

B. A client with persistent otitis media with a hearing loss of 20 dB or more should be referred to an otolaryngologist.

C. A client with a chronic TM perforation after recurrent or severe episodes of AOM should be referred to an otolaryngologist.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Do not use sulfa medications (sulfonamides) in pregnant clients.

B. Geriatrics:

1. Elderly clients may present with OME and/or AOM secondary to blocked eustachian tube and/or an URI.

2. Bullous myringitis is a condition in which blisters are visible on the TM, which can cause significant pain. Bullous myringitis may be caused by a viral, bacterial, or mycoplasmal infection.

3. Otitis media may cause inflammation of petrous bone adjacent to middle ear (mastoiditis), which could become critical for elderly in terms of further hearing loss, uncontrolled pain, and increased confusion.

4. Geriatrics are particularly vulnerable to recurrent otitis media (minimal of three relapses in 6 months or minimal of four annual infections).

5. Prevention strategies for the elderly are (a) to control modifiable risk factors such as smoking cessation and diabetes control and (b) sufficiently treat each ear infection as soon as possible.

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CERUMEN IMPACTION (EARWAX)

DEFINITION

A. Cerumen is defined as a mixture of ear secretions and dead epithelial cells which migrate laterally and often mix with hair. Cerumen occurs naturally in the external auditory canal (EAC) and is a protective mechanism of the ear that maintains homeostasis by lubricating the ear canal, removing debris, and providing bactericidal protection. Excessive accumulation of cerumen can cause a partial or complete obstruction of the EAC, leading to discomfort and associated conductive hearing loss (CHL).

INCIDENCE

A. Certain individuals are at increased risk of excessive earwax due to overproduction or inability of the ear to clear itself. The populations most likely to be diagnosed with cerumen impaction include the elderly, young children, those with mental disabilities, and institutionalized clients. The incidence in nursing home clients may be as high as 60%.

B. Approximately 12 million office visits per year occur for cerumen extraction in the United States.

PATHOGENESIS

A. With age, the normal self-cleaning mechanisms of the ear fail. Cilia, which have become stiff, cannot remove cerumen and dirt from the ear canal. Placing cotton swabs, paper clips,▶

bobby pins, and other objects into the EAC may also impact cerumen.

PREDISPOSING FACTORS

A. Age (decreased function of ear cilia).
B. Use of hearing aids or earbuds.
C. Recurrent otitis externa.
D. Dermatologic conditions of the external canal.
E. Previous audiologic surgeries.
F. Keratosis obturans.
G. Use of cotton swabs or other objects to clean the EAC.

COMMON COMPLAINTS

A. Dryness and itching of ear canal.
B. Dizziness/unsteadiness.
C. Otalgia.
D. Hearing loss.
E. Fullness in the ear.
F. Chronic cough.
G. Tinnitus.

SUBJECTIVE DATA

A. Elicit onset, duration, and laterality of symptoms.
B. Elicit history of cerumen impaction.
C. Question the client regarding the method used to clean the ears.
D. Determine personal or family history of anatomic deformity.
E. Assess for any physical barriers to wax extrusion, such as cotton swabs, hearing aids, or devices utilized to protect hearing (earplugs).

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.
B. Inspect:
  1. Observe ears for thick, light to dark brown wax occluding the auditory canal.
  2. Observe tympanic membrane (TM) if possible. Perforated TM is associated with otitis media.
  3. Inspect nose and throat.
C. Auscultate:
  1. Auscultate heart and lungs.

DIAGNOSTIC TESTS

A. Cerumen impaction is diagnosed with direct visualization of an otoscope.
B. When hearing loss is the primary subjective complaint, you may consider a hearing assessment. Expected outcomes associated with cerumen impaction include CHL of 35 to 40 dB and positive Rinne (tuning fork test reveals bone conduction greater than air conduction in affected ear).

DIFFERENTIAL DIAGNOSES

A. Cerumen impaction.
B. Foreign body in the ear canal.
C. Otitis externa: White, mucus-like ear discharge is associated with otitis externa.

PLAN

A. General interventions:
  1. If experienced in removal of cerumen impaction and chooses to proceed with cerumenectomy, the clinician may elect to obtain written informed consent prior to performing this procedure. Remove impaction by means of lavage or curettage. Be sure to inspect the canal and TM after removal of the cerumen. Document the client's hearing before and after removal of cerumen.
  2. If initial management is unsuccessful or for severe cerumen impaction, consider otolaryngology consult for microscopic cerumenectomy.
B. Client teaching: *See Client Teaching Guide for this chapter, "Cerumen Impaction (Earwax)."*
  1. Instruct the client to avoid cleaning their own ears. Advise not to clean ears with cotton swabs, bobby pins, and so forth. Using these devices pushes the wax further into the ear canal and can worsen symptoms.
  2. Instruct clients to avoid ear candling.
C. Pharmaceutical therapy:
  1. None indicated.

FOLLOW-UP

A. If cerumen is removed, no immediate follow-up is required. Consider having the client return in 4 to 6 weeks to recheck their ears for cerumen.

CONSULTATION/REFERRAL

A. Consult or refer the client to a specialist (otolaryngologist) when cerumen cannot be safely removed.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
  1. Cerumen impaction is common in older adult and geriatric clients secondary to the atrophic cilia and dry epithelium of the ear canal.
  2. The use of hearing aids and earbuds can also contribute to wax buildup and cause wax to be pushed further into the canal. Persons using hearing aids or earbuds should be evaluated for wax buildup as indicated.
  3. To prevent uncertainty and fears, the elderly with hearing loss need to be reminded that as a liquid treatment/water is held for 15 minutes in the canal, cerumen expands and might temporarily cause further hearing loss. Continue to encourage the client during any treatment and, if necessary, have the nurse stay with client during the wait time.

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HEARING LOSS

DEFINITION

A. Impaired hearing (complete or partial hearing loss) results from interference with the conduction of sound, its conversion to electrical impulses, or its transmission through the nervous system. There are three types of hearing loss: ▶

1. Conductive hearing loss (CHL; defect in the conductive pathway of the ear).
2. Sensorineural hearing loss (SNHL; defect in the inner ear, cochlea, or auditory nerve (cranial nerve VIII).
3. Mixed hearing loss (MHL; combined conductive and sensorineural hearing loss).

INCIDENCE

A. Hearing loss is the most prevalent sensory deficit in the United States. Approximately 30 million (12.7%) individuals in the United States have bilateral hearing loss and 48 million (20.3%) have hearing loss in at least one ear.
B. Approximately 43% of persons aged 65 to 84 years have hearing loss.
C. Globally, approximately 538 million individuals experience hearing loss that is considered to be disabling.

PATHOGENESIS

A. *CHL (air–bone gap)* occurs when sound is not conducted effectively and presents with a diminution of volume, particularly low tones and vowels. It may be caused by one of the following:
1. Otologic processes such as chronic or serous otitis media, otitis externa, foreign body, cerumen impaction, or trauma to the middle ear.
2. Otosclerosis disorder of the architecture of the bony labyrinth, which fixes the footplate of the stapes in the oval window.
3. Exostoses: bony excrescences of the external auditory canal.
4. Tympanic membrane (TM) perforation.
5. Cholesteatoma: growth of squamous epithelium in the middle ear space.
6. Glomus tumors: benign, highly vascular tumors derived from normally occurring glomera of the middle ear and jugular bulb.
7. Congenital malformations such as microtia and/or atresia.

B. *SNHL* occurs after damage to the inner ear or vestibulocochlear nerve and characteristically produces impairment of the high-tone perception. It is usually bilateral and symmetrical.
1. Presbycusis is hearing loss associated with aging and is the most common cause of diminished hearing in the elderly; onset is bilateral, symmetric, and gradual.
2. Noise-induced hearing loss is due to chronic exposure to sound levels in excess of 85 to 90 dB.
3. Drug-induced hearing loss can be caused by aminoglycoside antibiotics, furosemide, ethacrynic acid, quinidine, chemotherapeutic agents such as cisplatin, and high-dose aspirin.
4. Ménière disease produces a fluctuating, unilateral, low-frequency impairment usually associated with tinnitus, a sensation of fullness in the ear, and intermittent episodes of vertigo.
5. Acoustic neuroma is a benign tumor of cranial nerve VIII (rare).
6. Sudden deafness can derive from head trauma, skull fracture, meningitis, otitis media, scarlet fever, mumps, congenital syphilis, multiple sclerosis, and perilymph leaks or fistulas.
7. Infectious, autoimmune, or postinflammatory processes.

PREDISPOSING FACTORS

A. Acoustic or physical trauma.
B. Ototoxic medications (e.g., aminoglycosides, aspirin, radiation therapy, chemotherapy).
C. Changes in barometric pressures/barotrauma.
D. Recent upper respiratory infection (URI).
E. Pregnancy.
F. Otosclerosis.
G. Nasopharyngeal cancer.
H. Serous otitis media/middle ear effusion/eustachian tube dysfunction (ETD).
I. Cerumen impaction.
J. Foreign body in the ear.

COMMON COMPLAINTS

A. Partial or total hearing loss.
B. Associated tinnitus.
C. Difficulty understanding the television, phone conversations, and people talking.
D. Aural fullness, ear pressure.

SUBJECTIVE DATA

A. Elicit the onset, duration, progression, and severity of the symptoms. Note whether symptoms are bilateral or unilateral.
B. Obtain the client's history of past or recent trauma.
C. Review the client's occupational and recreational exposure to risk factors.
D. Review the client's medical history and medications, including over-the-counter (OTC) drugs and prescriptions.
E. Review the client's family medical history.
F. Review the client's history for recent URI or ear infections, especially for chronic ear infections.
G. Elicit data about any previous hearing loss, how it was treated, and how it affected daily activities. There is often a history of ear disease with CHL.
H. Review the client's other symptoms, such as dizziness, fullness or pressure in the ears, and noises.
I. Review what causes difficulty with hearing, high tones versus low frequencies. Can the client hear people talking, the television at normal volume, doorbells ringing, telephone ringing, and watch ticking?

PHYSICAL EXAMINATION

A. Inspect:
1. Examine both ears for comparison.
2. Externally inspect ears for discharge; note color and odor. Obstruction of the auditory canal by impacted cerumen, a foreign body, exostoses, external otitis, otitis media with effusion (OME), or scarring or perforation of the eardrum due to chronic otitis may be present.
3. Conduct otoscopic examination to observe the auditory canal for cerumen impaction or foreign body.
4. Inspect TM for color, landmarks, contour, perforation, and acute otitis media (AOM). A reddish mass visible through the intact TM may indicate a high-riding jugular bulb, an aberrant internal carotid artery, or a glomus tumor.

B. Palpate:
1. Palpate auricle and mastoid area for tenderness, swelling, or nodules.
2. Check lymph nodes if infection is suspected.

C. Perform neurologic exam:
1. Perform Weber and Rinne tests.
   a. In the Weber test, the tuning fork is perceived more loudly in the ear with CHL.
   b. The Rinne test shows that bone conduction is better than air conduction (it is normal when air conduction is greater than bone conduction).

DIAGNOSTIC TESTS

A. Audiogram screening in primary setting.
B. Air insufflation for TM mobility.
C. Diagnostic pure-tone audiometry (hearing test) with tympanometry (used to evaluate the TM and the middle ear status) performed by an audiologist.
D. CT scan of temporal bones or MRI of the brain and internal auditory canals after consultation with an otolaryngologist.

DIFFERENTIAL DIAGNOSES

A. Congenital hearing loss.
B. Traumatic hearing loss.
C. Ototoxicity.
D. Presbycusis.
E. Ménière syndrome.
F. Acoustic neuroma.
G. Cholesteatoma.
H. Infection.
I. Cerumen impaction.
J. Otitis externa.
K. Foreign body in the ear.
L. Tumors.
M. Otosclerosis.
N. Perforation of TM.
O. Serous otitis media.
P. Hypothyroidism.
Q. Paget disease.

PLAN

A. General interventions:
  1. Treat any primary cause (e.g., remove impacted cerumen, ETD).
B. Client teaching:
  1. Harmful noise levels should be avoided.
  2. Individual hearing protection devices should be utilized in all clients exposed to occupational noise.
C. Pharmaceutical therapy:
  1. Treat primary condition if applicable.

FOLLOW-UP

A. If hearing loss is identified and cause is not immediately treatable, refer the client to otolaryngology for further evaluation.

CONSULTATION/REFERRAL

A. Any sudden onset of hearing loss requires immediate referral.
B. The client should be referred to an otolaryngologist for an extensive workup when the primary cause cannot be identified.
C. Referral should be made to a hearing aid specialist for hearing evaluation and treatment as indicated (e.g., hearing aids).

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
  1. Impaired hearing among the elderly is common and can lower the quality of life. Up to 80% of those over 85 years have hearing loss.
  2. People with seriously impaired hearing often become withdrawn or appear confused.
  3. Subtle hearing loss may go unrecognized.
  4. Impacted cerumen is very common in the elderly and can be a source of hearing loss.
  5. Hearing loss in the elderly affects both peripheral and central auditory systems. The highest frequencies are predominantly impacted, which is the area vital to understanding spoken words.
  6. Geriatrics with depression and dementia significantly benefit from cochlear implants. Studies have suggested that the enhanced amplification improved behavioral health issues and quality of life. There is a 1% failure rate. Client selection criteria are the following:
    a. Moderate to severe bilateral SNHL.
    b. Unilateral deafness with/without ipsilateral tinnitus.
    c. Benefit from hearing aids less than expected.
    d. No medical contraindication with anesthesia and no active ear infections.
    e. Family/surrogate support, motivation, and appropriate expectations.
  7. Communicating with the elderly who have hearing loss:
    a. Ask what is their preference in communication.
    b. Stand/sit 2 to 3 feet away with client sitting in front of a wall to reflect sound.
    c. Speak slowly toward the better ear using a lower pitched voice (do not shout).
    d. Rephrase rather than repeat sentences, and pause between each phrase/idea as needed.
  8. Elderly clients with dexterity challenges may benefit with user-friendly hearing aid models such as behind-the-ear or in-the-ear types.
  9. Clients with advanced cognitive deficits might need specific aids attached to the device and fastened to clothing to prevent loss of hearing aid. Audiologists would assist families/surrogates with safety measures for these attachments.

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OTITIS EXTERNA

DEFINITION

A. Otitis externa is an inflammation of the external ear canal that commonly produces pruritus, pain, and purulent discharge. It may be acute (rapid onset generally within 48 hours of symptoms) or chronic depending on the underlying pathogenesis. It may or may not involve the tympanic membrane (TM).

INCIDENCE

A. Otitis externa is seen in persons of all ages (lifetime incidence is up to 10%).
B. Both sexes are equally affected.
C. The incidence is higher during summer months. Of otitis externa visits, 44% occur between the months of June and August.
D. Clients with asthma, allergic rhinitis, atopic dermatitis, or other chronic skin disorders are more likely to experience recurrent otitis externa.
E. All varieties of otitis externa, with the exception of necrotizing otitis externa, are common.

PATHOGENESIS

A. Acute diffuse otitis externa (swimmer's ear): Characterized by rapid onset (1–2 days) of symptoms including otalgia (often severe), external auditory canal swelling, possible otorrhea, and/or cellulitis of the pinna/adjacent skin. Bacteria causes 90% of cases. *Pseudomonas* is the most common bacterial infection (67%), followed by *Staphylococcus* and *Streptococcus*. Infection can also be fungal *(Aspergillus*, 90%). Bacterial or fungal invasion is usually preceded by trauma to the ear canal, aggressive cleaning of the naturally bactericidal cerumen, or frequent submersion in water (swimming).

B. Chronic otitis externa: Condition generally results from a persistent, low-grade infection and inflammation with *Pseudomonas*.

C. Eczematous otitis externa: Otitis externa is associated with primary coexistent skin disorder such as atopic dermatitis, seborrheic dermatitis, and psoriasis.

D. Necrotizing or malignant otitis externa: Invasive *Pseudomonas* infection results in skull base osteomyelitis. Cranial nerve palsies (of cranial nerves VII, IX, and XII) and periostitis of the skull base have been associated with necrotizing otitis externa. Necrotizing or malignant otitis externa is a life-threatening condition that occurs in diabetic or immunocompromised clients. It is most commonly seen in the immunocompromised or diabetic geriatric client.

PREDISPOSING FACTORS

A. Exposure to a moist or humid environment (frequent swimming).

B. Ear trauma from scratching, foreign object, or vigorous cleaning.

C. Use of a hearing aid or earplugs.

D. Primary skin conditions, such as eczema.

E. Immunocompromised, malnourished, or comorbid conditions.

COMMON COMPLAINTS

A. Otalgia.

B. Itching.

C. Erythematous and swollen external canal.

D. Purulent discharge.

E. Hearing loss from edema and obstruction of canal with drainage.

OTHER SIGNS AND SYMPTOMS

A. Plugged ear sensation (aural fullness).

B. Tenderness to palpation (tragus, pinna, or both) that is disproportionate to what may be expected based on visual inspection.

SUBJECTIVE DATA

A. Elicit the onset, duration, and intensity of ear discomfort.

B. Inquire into the client's history of ear infections.

C. Determine whether the client notes any degree of hearing loss.

D. Question the client about recent exposure to immersion in water (swimming).

E. Determine if the client wears hearing aids, earplugs, or other devices that may impact the ear canal.

F. Question the client as to ear canal cleaning practices and any recent trauma to canal.

PHYSICAL EXAMINATION

A. Temperature.

B. Inspect:

1. Carefully examine the ear with an otoscope due to its extreme tenderness.

2. Observe the ear for erythematous and edematous external canal; look for otorrhea and debris.

3. Observe the TM, which may appear normal.

4. Inspect the nose and throat.

C. Auscultate:

1. Auscultate the heart and lungs.

D. Palpate:

1. Apply gentle pressure to tragus and manipulate pinna to assess for tenderness.

2. Palpate preauricular, postauricular, and lateral cervical lymph nodes.

DIAGNOSTIC TESTS

A. Examine ear canal scrapings and drainage under microscope for hyphae (if fungal infection is suspected from history or ineffective topical therapy).

DIFFERENTIAL DIAGNOSES

A. Otitis externa.

B. Acute otitis media (AOM).

C. Chronic suppurative otitis media.

D. Cholesteatoma.

E. Mastoiditis.

F. Referred pain (wisdom tooth eruption).

G. Herpes zoster oticus (vesicular eruptions in the ear canal are associated with herpetic otitis externa).

PLAN

A. General interventions:

1. The primary treatment goals are to reduce pain and inflammation and to treat the underlying organism.

2. In extreme cases, the client's ear canal may be blocked by edema or drainage, preventing passage of ear drops. Cautiously irrigate the canal and insert a cotton ear wick (approximately 1 in. long for adults) by gently rotating it to allow passage of drops. Consider referral for abnormal or extensive cases.

B. Client teaching: *See Client Teaching Guide for this chapter, "Otitis Externa."*

1. The client should be advised to keep water out of the ear during treatment, which typically lasts 1 week. During this time, bathing or showering is permitted with earplugs or a cotton ball coated with petroleum jelly inserted into the ear to block water passage into the ear canal.

2. Clients with appropriately treated uncomplicated acute otitis externa generally show signs of improvement within 48 to 72 hours.

C. Pharmaceutical therapy:

1. Oral antibiotics are indicated only if there is an associated otitis media and should not be used alone for otitis externa.

2. For early, mild cases associated with swimming in which the primary symptom is pruritus, homemade preparations of 50% isopropyl alcohol and 50% vinegar or acetic acid/aluminum acetate can be used as a drying agent and to create an unsatisfactory environment for *Pseudomonas* growth.

3. For mild infection, use topical therapy with acidifying agent such as Vosol or Vosol HC, which includes a glucocorticoid therapy. Instill five drops in the ear canal three to four times daily. (Vosol and Vosol HC are contraindicated with perforated eardrum; Vosol HC is contraindicated with viral otic infections.)

4. For moderate infection, use of an acidifying agent, antibiotic, and glucocorticoid therapy (CiproDex) is suggested. Other alternatives include ciprofloxacin (Cipro HC) and ofloxacin (Floxin). Potentially ototoxic ototopical aminoglycosides (Neomycin and Polymyxin) should be avoided if the integrity of the TM is in question.

a. If fungal infection is suspected, clotrimazole and betamethasone dipropionate lotion (Lotrisone) can be used for fungal infections if TM is intact. Nystatin 100,000 units/mL or clotrimazole topical solutions may also be used for *Candida* or yeast infections.

b. Acyclovir is the treatment of choice for herpes simplex or herpes zoster.

5. Severe or resistant infections in healthy clients may require additional management with oral antibiotics and antifungals. In addition, oral antibiotics should be considered as an adjunct to topical therapy in clients who are immunocompromised, malnourished, or have comorbidities, including associated otitis media and/or cellulitis of the outer ear:

a. Ciprofloxacin for pseudomonal infections; dicloxacillin or cephalexin for staphylococcal infections.

b. Itraconazole (Sporanox) for treatment of otomycosis (fungal otitis externa).

6. Use of acetaminophen or ibuprofen should be adequate for pain control. Pain uncontrolled by these medications should be a red flag for serious infection and warrants immediate referral.

FOLLOW-UP

A. Usual follow-up is within 3 to 5 days to assess improvement. Recheck in 1 to 2 weeks.

B. If wicking is required, the wick should be removed within 3 to 5 days, once the swelling is reduced. It is common for the wick to spontaneously fall out as the swelling improves. Upon reduction of swelling, clients can then instill ototopical drops directly into the ear canal.

CONSULTATION/REFERRAL

A. Any client suspected of necrotizing otitis externa requires immediate referral due to the potential life-threatening complications of osteomyelitis.

B. Consult or refer the client to an otolaryngologist or ED if osteomyelitis is suspected.

C. For complications (e.g., abscess, mastoiditis, and meningitis), refer to the ED for emergent treatment.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Ramsay Hunt syndrome, also known as herpes zoster oticus, occurs when the shingles virus breaks out near the ear, affecting cranial nerves V, IX, and X, causing painful blisters/rash near the ear and/or the external ear canal. Facial paralysis, change in taste perception, and/or hearing loss in the affected ear may occur. Symptoms may include shingles rash near the ear and/or inside the external ear canal, ear pain, change in taste, hearing loss, ringing in the ear, dizziness, and/or facial paralysis near the ear/mouth. Prompt treatment with antiviral medications is recommended to prevent permanent damage to the nerve. Hearing loss and facial paralysis are commonly temporary but may also be permanent. Nerve damage may occur. Referral to an otolaryngologist for evaluation is recommended.

2. Malignant otitis externa is found mostly in the elderly and/or diabetics. Treatments include surgical removal of dead tissue, antibiotics, and hyperbaric oxygen treatment (HBOT), if the client is determined a candidate.

3. There is an increased risk of otitis externa with diabetic geriatrics. The primary goal is to keep blood glucose controlled and to monitor ear canals frequently.

4. The elderly population are apt to clean their ears more often than younger adults secondary to dry itchy skin. Frequent cleansing may alter the immune response and pH balance of the external canal, predisposing to risk of otitis externa infection.

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OTITIS MEDIA WITH EFFUSION

DEFINITION

A. Otitis media with effusion (OME) is a noninfectious asymptomatic middle ear fluid without signs of bacterial infection.

INCIDENCE

A. OME is seen in persons of all ages.

B. OME often follows an acute upper respiratory infection (URI) or recent diagnosis of acute otitis media (AOM).

C. The middle ear effusion typically resolves without treatment in more than 80% of all clients. Although symptoms can sometimes continue for up to 12 weeks, the majority of clients will have complete symptom resolution by 4 weeks.

D. Approximately 2.2 million new cases of OME are diagnosed in the United States each year.

PATHOGENESIS

A. The effusion may be sterile fluid secondary to URI and eustachian tube dysfunction. It may be residual fluid after an episode of AOM.

PREDISPOSING FACTORS

A. Recent AOM.
B. Concurrent or recent URI.
C. Chronic sinusitis.
D. Allergies.
E. Craniofacial predisposition.
F. Smoking or exposure to secondhand smoke.
G. History of middle ear infections.
H. Barotrauma.

COMMON COMPLAINTS

A. Ear pain.
B. Increased pressure sensation in the ears.
C. Recent hearing loss/muffled hearing.

OTHER SIGNS AND SYMPTOMS

A. A sense of fullness in the ears.
B. Dizziness/unsteadiness.
C. Tinnitus.

SUBJECTIVE DATA

A. Elicit onset, laterality, and duration of symptoms.
B. Inquire about recent history of otitis media or URI.
C. Inquire about subjective hearing loss.
D. Determine if the client has a history of frequent otitis media.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.
B. Inspect:
  1. Inspect ears, noting fluid level, serous middle fluid, and a translucent, amber, gray membrane with decreased mobility.
  2. Inspect nose, mouth, and throat.
C. Auscultate:
  1. Auscultate heart and lungs.
D. Palpate:
  1. Palpate head, neck, and lymph nodes in the head, chin, and neck.
E. Perform neurologic examination:
  1. Perform Weber and Rinne tests with tuning fork.

DIAGNOSTIC TESTS

A. Pneumatic otoscopy reveals decreased mobility. **Assessment with pneumatic otoscopy is strongly recommended**.
B. Tympanometry is a test that measures how easily the tympanic membrane (TM) vibrates. Type A tympanograms are normal; type B tympanograms indicate a hypercompliant TM (e.g., middle ear effusion); type C tympanograms indicate negative middle ear pressure.

DIFFERENTIAL DIAGNOSES

A. OME.
B. AOM.
C. Cerumen impaction or foreign body.
D. Otitis externa.
E. Cholesteatoma.
F. Myringitis.
G. Tympanosclerosis.

PLAN

A. General interventions:
  1. Client should be monitored closely for resolution of effusion without treatment within several weeks.
  2. Clients with persistent OME (>12 weeks) should be referred to an otolaryngologist for a hearing evaluation and possible tympanostomy tubes as indicated.
  3. Maneuvers such as intermittent autoinsufflation may be helpful for more symptomatic clients (persistent aural fullness, minimal to moderate discomfort) in helping relieve the symptoms until the effusion resolves.
B. Client teaching: *See Client Teaching Guide for this chapter, "Otitis Media With Effusion."*
C. Pharmaceutical therapy:
  1. Use of nasal corticosteroids and nasal antihistamines may be helpful in clients with OME.
  2. Systemic decongestants can be used.

FOLLOW-UP

A. Recheck the client's ears after 4 weeks to monitor for spontaneous resolution. If no resolution is noted, schedule a follow-up appointment.

CONSULTATION/REFERRAL

A. Consult or refer the client to an otolaryngologist if treatment is not effective or if the client has a persistent effusion (at least 3 months) along with a hearing loss of 20 dB or more.
B. Consider referring the client to an otolaryngologist prior to 12 weeks if they do not have the usual underlying risk factors associated with OME.
C. Consider earlier referral to an otolaryngologist when the OME is bilateral due to potential increased hearing loss.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
  1. OME in geriatrics is an ominous sign that needs thorough investigation. A pharyngeal congestion could spread to the eustachian tubes, causing a pressure increase in the middle ear that forms serous effusion. Often these infections stem from a virus and most erupt from a URI.
  2. The prognosis tends to be poor for the elderly if their immune system is compromised. In this population, it could lead to permanent hearing loss, endocarditis, or septicemia. Prevention and immediate effective treatment are paramount in avoiding complications.

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PRESBYCUSIS

DEFINITION

A. A sensorineural hearing loss (SNHL) associated with aging.
B. Symmetrical progressive hearing loss over many years; gradual.
C. Affects the high frequencies of hearing first.

INCIDENCE

A. Hearing loss is common in the elderly and increases with age. Presbycusis affects more than half of adults by age 75 years, most adults over the age of 80, and nearly all adults who are 90 or older.
B. Males tend to be affected slightly earlier and experience more impairment, possibly related to higher levels of noise exposures.
C. The World Health Organization estimates there will be 1.2 billion people over 60 years old worldwide who will suffer significantly from presbycusis by 2025.

PATHOGENESIS

A. Multiple factors of heredity, cumulative noise exposure, and age-related physiologic changes to the ear are thought to be factors in the degree of hearing loss. Although presbycusis is multifactorial, physiologic changes in the cochlea have been identified as the primary underlying mechanism of decreased hearing.

PREDISPOSING FACTORS

A. Age.
B. Family history (genetics).
C. Ototoxic medications (e.g., gentamicin and aspirin).
D. Cumulative noise exposure.
E. Cardiovascular disease (hypertension [HTN] and hyperlipidemia).
F. Smoking.
G. Immunologic disorders.
H. Hormonal factors (estrogen).

COMMON COMPLAINTS

A. Partial hearing loss.
B. Difficulty understanding the television, phone conversations, and people talking.
C. Difficulty in background noises.
D. Tinnitus.

SUBJECTIVE DATA

A. Elicit the onset, duration, progression, and severity of the symptoms. Note whether symptoms are bilateral or unilateral.
B. Review the client's family history.
C. Review the client's occupational and recreational exposure to noise.
D. Review the client's medical history for cardiovascular disease, HTN, and hyperlipidemia.
E. Ask specifically about all medications, including over-the-counter (OTC) drugs and prescriptions.
F. Review the client's history for recent upper respiratory infection (URI), or ear infections, especially for chronic ear infections.
G. Elicit data about any previous hearing loss, how it was treated, and how it affected daily activities. There is often a history of ear disease with conductive hearing loss.
H. Review the client's other symptoms, such as dizziness, fullness or pressure in the ears, and noises.
I. Review what causes difficulty with hearing, high tones versus low frequencies. Can client hear people talking, the television at normal volume, doorbells ringing, telephone ringing, and watch ticking?

PHYSICAL EXAMINATION

A. Inspect:
  1. Examine head, neck, and ears for signs of trauma, asymmetry, masses, or edema.
  2. Externally inspect ears for discharge; note color and odor. Obstruction of the auditory canal by impacted cerumen, a foreign body, exostoses, external otitis, otitis media with effusion (OME), or scarring or perforation of the eardrum due to chronic otitis may be present.
  3. Conduct otoscopic examination to observe the auditory canal for cerumen impaction or foreign body.
  4. Inspect tympanic membrane (TM) for color, landmarks, contour, perforation, and acute otitis media (AOM). A TM that is opaque rather than translucent can be a variation of normal in elders and is not related to hearing loss.
B. Common screening tests:
  1. Whispered voice: From 2 feet behind the client, the examiner whispers words or short phases while occluding one ear at a time.
  2. Client report: Ask the client, "Do you have difficulty hearing?"
  3. Screening version of the Hearing Handicap Inventory for the elderly.
  4. Weber and Rinne tests to differentiate conductive from SNHL: The Weber test reveals conductive hearing loss when sound travels toward the poor ear. SNHL is present when sound travels toward the good ear. This screening test is performed by striking a tuning fork and then placing it on the middle of the head. The client is then asked where sound is being heard from—the left ear, the right ear, or equal in both ears. Normal results are reflected by sound being heard equally in both ears. The Rinne tuning fork test reveals bone conduction greater than air conduction in the affected ear (abnormal). The Rinne test is performed by placing the struck tuning fork against the mastoid bone. Begin counting or timing the interval from the start to when the client can no longer hear. Continue counting or timing the interval to determine the length of time sound is heard by air conduction. Air-conducted sound should be heard twice as long as bone-conducted sound after bone conduction stops.

DIAGNOSTIC TEST

A. Audiogram in primary setting.

DIFFERENTIAL DIAGNOSES

A. Presbycusis.
B. Sudden SNHL.
C. Ototoxicity.
D. Ménière syndrome.
E. Acoustic neuroma.
F. Cholesteatoma.
G. Chronic otitis media.
H. Cerumen impaction or foreign body.

PLAN

A. General interventions:
  1. All clients with suspected hearing loss should undergo formal testing.
  2. Goals are to prevent further hearing loss and assist the client in lifestyle changes that increase quality of life.
B. Client teaching:
  1. Let clients know that a follow-up with a specialist is important to identify the correct diagnosis.
  2. Summarize treatment options of aural rehabilitation, hearing aids and, when applicable, cochlear implants.
  3. Assess for and teach clients the consequences of excessive environmental noise; consider protective mechanisms such as earplugs.
C. Nonpharmaceutical therapy:
  1. Aggressive management of comorbidities such as HTN and hyperlipidemia may slow progression of hearing loss.
  2. Evaluate for polypharmacy and the role it may play in hearing loss. Specifically assess for ototoxic medications. Eliminate when possible.

FOLLOW-UP

A. Stress the importance of follow-up with the audiologist and otologist. Often the client does not pursue viable options that would improve quality of life.

CONSULTATION/REFERRAL

A. The client should be referred to an otolaryngologist immediately for sudden, severe, or rapidly progressing hearing loss and hearing loss that is unilateral; associated with pain, bleeding, or drainage; or involves other neurologic deficits.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Studies have strongly suggested that music training and education among the elderly positively affected presbycusis and were important factors in slowing deterioration of music perception and perhaps speech intelligibility.

2. The elderly may "try out" OTC hearing aids. Studies have strongly suggested that these OTC devices are not efficient secondary to ineffective volume control, high equivalent input noise, and irregular frequency response. Encourage clients to talk about their feelings regarding hearing loss and their motivation to improve it with appropriate auditory devices. This will help build a foundational willingness from the client for a consultation/referral.

3. Impaired hearing among the elderly is common and can lower quality of life. Individuals with significantly impaired hearing may become withdrawn or appear confused.

4. Impacted cerumen is common in the elderly.

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TINNITUS

DEFINITION

A. The word "tinnitus" comes from the Latin *tinnire*, which means "to ring." Tinnitus is the perception of an auditory sensation such as hissing, sizzling, or ringing in the absence of external stimuli. Sounds can be constant or intermittent and vary in intensity from client to client. The tinnitus can also be categorized as rhythmical or pulsatile and corresponding with a heartbeat (this may suggest a vascular origin).

B. Primary tinnitus is defined as tinnitus that is idiopathic and may or may not be associated with sensorineural hearing loss (SNHL). Secondary tinnitus is defined as tinnitus that is associated with an underlying cause other than SNHL (e.g., cerumen impaction, middle ear disease, eustachian tube dysfunction, Meniere disease, and vestibular schwannoma).

INCIDENCE

A. Tinnitus is a very common medical problem that affects an estimated 50 million people in the United States.

B. Prevalence is similar between males and females and is known to increase with age.

PATHOGENESIS

A. Tinnitus is poorly understood and there is wide disagreement on the pathophysiology. It is best described as a nonspecific manifestation of pathology of the inner ear, cranial nerve VIII, or the central auditory mechanism. There is a strong association between tinnitus, stress, anxiety, and depression.

PREDISPOSING FACTORS

A. High levels of noise exposure, either recreational or occupational.

B. Otologic conditions:

1. Neoplasms.
2. Hearing loss.
3. Cerumen impaction.
4. Otitis media/middle ear effusion.
5. Mastoiditis.
6. Labyrinthitis.
7. Tympanic membrane (TM) perforations.
8. Acoustic trauma.

C. Neurologic conditions:

1. Vertigo.
2. Migraines.
3. Epilepsy.
4. Meningitis.
5. Multiple sclerosis.
6. Jugular bulb anomaly: Compression of the ipsilateral jugular vein abolishes the objective tinnitus of a jugular megabulb anomaly.

D. Trauma to head or neck or issues with temporomandibular joint (TMJ) disorder.

E. Immune system disorders such as rheumatoid arthritis, systemic lupus erythematosus, or systemic sclerosis.

F. Endocrine influences such as diabetes, hypothyroidism, and pregnancy.

G. Psychological disorders:

1. Anxiety.
2. Depression.
3. Posttraumatic stress disorder.
4. High stress.

H. Hypertension.

I. Ototoxic drugs: More than 130 medications are known to cause tinnitus or hearing loss. Clients should be assessed for concurrent use of multiple ototoxic medications, especially when other risk factors are present. The most common broad categories include the following:

1. Analgesics.
2. Antibiotics.
3. Antineoplastics and immunosuppressives.
4. Diuretics.
5. Corticosteroids.

COMMON COMPLAINTS

A. Ringing.
B. Roaring.
C. Buzzing.
D. Clicking.
E. Hissing.
F. Hearing loss.
G. Fluttering.

OTHER SIGNS AND SYMPTOMS

A. "Muffled" hearing.
B. Change in own voice, lower pitch.

SUBJECTIVE DATA

A. Review the onset, duration, course, and type of symptoms; note whether they are bilateral, unilateral, or centralized.

B. Determine the frequency and quality of sound; is the ringing constant, intermittent, or pulsating?

C. Review all medications, including over-the-counter (OTC) drugs and prescriptions.

D. Determine if there is a family history of tinnitus, hearing loss, or neurofibromatosis.

E. Ask whether the client has experienced recent physical or emotional trauma. Inquire about recent/current stressors the client has experienced.

F. Inquire about any history of ontologic disorders, either acute or chronic.

G. Ask about associated symptoms:

1. Headache.
2. Hearing loss.
3. Noise intolerance.
4. TMJ.
5. Vertigo.

H. Review work, hobbies, and music habits for noise levels (potential damage).

I. Assess the date of last hearing exam and determine whether there was any known hearing loss.

J. Review whether client uses cotton-tipped swabs or other small objects for ear cleaning.

PHYSICAL EXAMINATION

A. General: The goal of the physical exam is to evaluate for the most common known causes of tinnitus.

B. Inspect:

1. Observe the external ear for discharge; note color and odor.
2. Conduct otoscopic exam of the auditory canal for cerumen impaction or foreign body.
3. Inspect the TM for color, landmarks, contour, perforation, and acute otitis media (AOM).

a. The landmarks (umbo, handle of malleus, and the light reflex) should be visible on a normal exam.
b. The TM should be pearly gray in color and translucent.
c. A bulging TM is more conical, usually with a loss of bony landmarks and a distorted light reflex.
d. A retracted TM is more concave, usually with accentuated bony landmarks and a distorted light reflex (pathologic conditions in the middle ear may be reflected by the characteristics of the TM).

C. Auscultate:

1. Assess for bruits or murmurs over the ear canal, auricular areas, neck, and chest.

D. Palpate:

1. Palpate auricle and mastoid area for tenderness, swelling, or nodules.
2. Palpate the TMJ for tenderness or crepitus.

E. Perform visual exam:

1. Inspect for papilledema.
2. Assess for visual field changes and for nystagmus.

F. Perform neurologic exam:

1. Assess all cranial nerves.
2. First evaluate how the client responds to your questions.
3. Clients who speak in a monotone or with erratic volume may have hearing loss.
4. Check the client's response to a soft whisper (should respond at least 50% of the time).
5. Perform Rinne and Weber tests.

DIAGNOSTIC TESTS

A. When tinnitus is acute (<3 weeks) and has no associated neurologic deficits or hearing loss, consider audiometry. Any focal neurologic findings warrant an MRI and/or prompt physician referral. Tinnitus lasting longer than 3 weeks should be referred to an otolaryngologist. Appropriate diagnostics are influenced by intermittent hearing loss, vertigo, abnormal physical exam findings, pulsatile quality, and unilateral presentation.

DIFFERENTIAL DIAGNOSES

A. Tinnitus.

B. Hearing loss that could be related to multiple otologic conditions:

1. Cerumen impaction.
2. Foreign body.
3. AOM.
4. Ménière disease.
5. Presbycusis.
6. Cholesteatoma.

C. TMJ syndrome.

D. Ototoxicity.

E. Central nervous system lesion.

PLAN

A. General interventions:

1. Assess for underlying cause of tinnitus.
2. Refer to an otolaryngologist for tinnitus lasting longer than 3 weeks.

B. Client teaching: *See Client Teaching Guide for this chapter, "Tinnitus."*

1. Educate the client regarding techniques/therapies to improve symptoms of tinnitus, such as sound therapy, interventions to reduce distress such as counseling, and cognitive behavioral therapy (mindfulness meditation, biofeedback). Consider hearing aids for clients with hearing loss and tinnitus.
2. Encourage the client to attend therapy sessions as indicated.

C. Pharmaceutical therapy:

1. Medications do not have a beneficial role in the treatment of tinnitus. There are no medications to "cure" tinnitus.

FOLLOW-UP

A. Length of follow-up depends on whether the client has an identified and/or treatable cause of tinnitus. Typically no specific follow-up is required for tinnitus unless a treatable problem is identified.

CONSULTATION/REFERRAL

A. Consult with an otolaryngologist as indicated.

B. Referral of an anxious client to the otolaryngologist may be necessary to assure the client that everything has been explored and that there is no serious or correctable underlying condition.

C. Any client with a history of head trauma should be referred to an otolaryngologist because in rare cases tinnitus may be associated with an acoustic neuroma, arteriovenous fistula, or an aneurysm of the intrapetrous portion of the internal carotid artery.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Tinnitus onset later in life tends to be more abrupt and distressing than with younger adults. It is important to monitor this population closely for comorbid anxiety/depression, psychosocial changes, and sleep pattern disruptions.

2. Ringing in the ears can be a severe distraction that could increase the risk of unexpected accidents and falls.

3. Geriatrics with arterial bruits, venous hums, shunts, and high cardiac output are particularly vulnerable to exacerbative tinnitus. Monitor for social isolation and conversational withdrawal. Encourage referral for cochlear implants to those who have long-term tinnitus with profound hearing loss.

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CLIENT TEACHING GUIDE

CERUMEN IMPACTION (EARWAX)

PROBLEM A buildup of earwax in the external ear canal that may cause itching, pain, dizziness, ear ringing, aural fullness, and/or temporary hearing loss.

CAUSE Earwax production is a normal, healthy process of the gland of the ear. Earwax is produced to protect the ear from infection and trauma. The wax is continuously being produced and removed from the ear on its own mechanism. Ears are self-cleaning organs. However, at times, an overproduction of wax may build up and remain in the external ear canal. With age, the normal mechanisms of the ear for removing earwax are decreased. This is called cerumen impaction. Use of cotton swabs to remove earwax can push the wax further into the ear and cause problems deeper into the ear canal.

PREVENTION Do not use cotton swabs, paper clips, bobby pins, or other objects to clean your ears. These can damage the ear canal and lead to an external ear infection and/or cause further impaction.

TREATMENT PLAN

A. It is generally safe to clean the external ears with a wet washcloth. The external ear that is visible to the eye is the only part of the ear that should be cleaned with a wet washcloth.

B. In general, it is preferable to allow your healthcare provider or your otolaryngologist to clean your ears. Do not try to remove the earwax on your own because of the chance of damaging your eardrum or creating an environment for bacteria to grow. Never stick any kind of tool into your ear. This will usually push the wax further into your ear canal, making removal more difficult for your healthcare provider.

Activity: As tolerated.

Diet: As tolerated.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:

A. You are unable to hear.
B. You have colored drainage or fluid draining from your ears.
C. You run a fever.
D. You have dizziness.
E. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

OTITIS EXTERNA

PROBLEM Your practitioner has diagnosed you with a condition known as otitis externa, sometimes also referred to as "swimmer's ear." This is a common condition characterized by itching in the ear, sometimes followed by ear pain, swelling, and drainage of the ear canal. Difficulty hearing may also occur. The eardrum is rarely affected.

CAUSE Otitis externa occurs from irritation to the external canal of the ear. The most common causes of otitis externa are long exposure to water in the ear canal after frequent swimming and too vigorous cleaning of the wax from your ears. It may involve either a bacterial or a fungal infection.

PREVENTION You may prevent future problems with otitis externa by following these measures:

A. Clean the outer ear only as needed. Do not use cotton-tipped swabs or any other device to clean down into the ear canal. Usually, wax is just pushed deeper into the canal with this method and the canal may be traumatized by the instrument used.

B. For swimmers or others susceptible to frequent recurrences of otitis externa, it may be helpful to dry the ear canals with a blow dryer on a low setting after exposure to water. You may also instill a solution of isopropyl alcohol (rubbing alcohol) in the ear twice daily and after every submersion in water. Do not instill rubbing alcohol into the ear if the ear is infected or if there is a hole in the eardrum.

C. Earplugs may be beneficial to swimmers with recurrences of otitis externa.

TREATMENT PLAN For the most common bacterial infections associated with otitis externa, antibiotic/steroid ear drops are usually prescribed. For fungal infections associated with otitis externa, antifungal/steroid ear drops are usually prescribed. Frequent cleanings by an otolaryngologist may be required to remove infection. For both bacterial and fungal infections associated with otitis externa, the ear should be kept dry until it is healed (including no swimming). To bathe or shower, first coat cotton balls with petroleum jelly and use them to plug the ears.

Activity: Avoiding moisture accumulation in the ear is important. Bathing and hair washing are permitted as previously described. Continue with dry ear precautions until cleared by your healthcare provider.

Diet: No changes are required in your diet.

Medications:

A. Ear drops are used to treat otitis externa.

B. The drops should be applied down the ear canal's opening, moving the earlobe back and forth to help the ear drops pass downward.

C. In severe cases, oral antibiotics may be given.

You Have Been Prescribed: _____

You Need to Instill _____ **Drops Into the Affected Ear** _____ **Times Per Day.**

You Need to Notify the Office If You:

A. Have symptoms that worsen or do not seem to be improving.

B. Have a fever over 100.8°F.

C. Have increased pain, redness, or swelling in the ear or around the ear or face.

D. Are unable to instill ear drops into the ear due to swelling of the ear canal.

E. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

OTITIS MEDIA WITH EFFUSION

PROBLEM You have inflammation of the middle ear with effusion, which is the presence of fluid in the middle ear without infection.

CAUSE The middle ear fluid can remain behind the tympanic membrane after you have been treated for an ear infection (otitis media). The eustachian tube is blocked and the fluid behind the ear is not draining out properly. Symptoms may include difficulty hearing and a feeling of fullness in the ear.

TREATMENT PLAN

A. Determine if you are having increased difficulty hearing.
B. Make accommodations for the hearing loss, such as reducing or eliminating external noises while having a conversation clearly and loudly.
C. If you have a buildup of fluid in the middle ear for longer than 12 weeks or you notice a significant or increasing hearing deficit, you should receive a hearing evaluation.
D. Follow up with a healthcare provider as instructed to avoid complications and/or permanent hearing loss.

Activity: There is no activity restriction.

Diet: There is no special diet.

Medications: Middle ear fluid often resolves without treatment within 3 months. Nasal spray steroids and nasal spray antihistamines (Astelin) are sometimes prescribed and can improve the time that is taken for the fluid to resolve. These medications should be taken exactly as directed. Antibiotics are likely not needed.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. Fever.
B. Decreased appetite.
C. Decreased activity level.
D. Ear pain.
E. Noticed a change in hearing loss or speech development.
F. Any other new symptoms that occur.
G. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

TINNITUS

PROBLEM Tinnitus is an irritating noise or sound that is heard in one or both ears, commonly referred to as a buzzing, humming, or ringing noise.

CAUSE Tinnitus is poorly understood and the cause is not known. It probably originates in the brain and not the ear. There is a strong association between tinnitus, stress, anxiety, and depression. Stressors include emotional, physical (comorbidities), environmental, and so forth. You do not have to have hearing loss to have tinnitus.

TREATMENT PLAN

A. Hearing evaluation will be performed to determine if there is hearing loss.
B. A CT or MRI may be performed to evaluate for the cause of the tinnitus.
C. Rest, exercise, and mindfulness meditation are encouraged to reduce stress, anxiety, and depression, which can worsen symptoms of tinnitus.

Activity: There is no activity restriction.

Diet: Some foods may make the tinnitus worsen. Therefore, reducing the amount of sodium, nicotine, alcohol, and caffeine (tea, coffee, soft drinks) is encouraged.

Medications: Avoid aspirin. Aspirin has been known to aggravate the symptoms of tinnitus.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. Fever.
B. Ear pain.
C. A change in hearing loss or speech development.
D. Dizziness.
E. Any other new symptoms that occur.
F. Other: _____

Phone: _____

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CHAPTER 10

NASAL GUIDELINES

Jill C. Cash and Sarah Hendershott Taylor

ALLERGIC RHINITIS

DEFINITION

A. Allergic rhinitis is a chronic or recurrent condition characterized by nasal congestion, clear nasal discharge, sneezing, nasal itching, conjunctival itching, and periorbital edema. It usually occurs seasonally after exposure to allergens (same time every year, associated with pollen count) or may be perennial (year-round, related to indoor inhalants, animal dander, and mold). Although these classifications can occur distinctly, clients may also have combined seasonal and perennial allergic rhinitis. "Allergic" suggests that a specific immunoglobulin E (IgE) antibody mediates the condition.

INCIDENCE

A. Prevalence varies according to geographic region; 10% to 30% of adults have allergic rhinitis. In 70% of cases, allergic rhinitis develops before age 20.

B. Intranasal corticosteroid sprays are recommended for adolescents 12 years of age or older as initial treatment, instead of combination therapy with antihistamines and/or leukotriene antagonists.

C. Sleep-disturbed breathing is one of the most significant sequelae of allergic rhinitis.

PATHOGENESIS

A. This is an IgE-mediated inflammatory disease involving nasal mucosa; IgE antibodies bind to mast cells in the respiratory epithelium, and histamine is released. This results in naso-ocular inflammation, leading to congestion, rhinorrhea, sneezing, and pruritus. The nasal allergic response has an immediate and late phase. The immediate phase peaks 15 to 30 minutes after allergen exposure and the late phase peaks 6 to 12 hours after exposure.

PREDISPOSING FACTORS

A. Genetic predisposition to allergy (especially for individuals with eczema or asthma).

B. Exposure to allergic stimuli: pollens, molds, animal dander, dust mites, and indoor inhalants.

COMMON COMPLAINTS

A. Nasal congestion/obstruction.
B. Sneezing.
C. Clear rhinorrhea.
D. Cough from postnasal drip.
E. Sore throat.
F. Itchy, puffy eyes with tearing.

OTHER SIGNS AND SYMPTOMS

A. Dry mouth from mouth-breathing, snoring.
B. Sleep disturbance due to difficulty with breathing, leading to malaise/fatigue.
C. Itchy nose.
D. Decrease/loss of smell and taste.
E. Eczema.
F. Shortness of breath, difficulty breathing, and wheezing.
G. Headache.
H. Halitosis.

SUBJECTIVE DATA

A. Ask about onset, course, and duration of symptoms.
B. Inquire about the characteristics of nasal discharge.
C. Inquire about exposure to people with similar symptoms.
D. Ask about seasonal impact on symptoms.
E. Inquire about other diseases caused by allergens, such as asthma, eczema, and urticaria.
F. Rule out pregnancy.
G. Ask female clients about their birth control method, specifically birth control pills.
H. Review exposure to irritants.
I. Ask about any past or recent nasal trauma.

PHYSICAL EXAMINATION

A. Check vital signs: temperature, blood pressure, pulse, and respirations.
B. Inspect:
1. Examine face. Note Dennie lines (skin folds under the eyes) and allergic salute (transverse crease on the nose from chronic rubbing).
2. Examine eyes and conjunctivae.
   a. Tearing, red and swollen eyelids, and allergic shiners (dark circles under eyes from venous congestion in maxillary sinuses) are seen with allergies.
   b. Palpebral conjunctiva pale and swollen, bulbar conjunctiva is injected.
3. Examine ears, nose, and throat.
   a. Red, dull, bulging, perforated tympanic membrane is seen with otitis media.
   b. Nasal redness, swelling, polyps, and enlarged turbinates are seen with upper respiratory infection (URI). Mucosa appears pale blue, boggy with clear discharge in chronic allergies.
   c. Cobblestone appearance in pharynx, tonsils, adenoids, and turbinates is seen in chronic allergies.
   d. Use otoscope light to transilluminate under superior orbital ridge of frontal sinus cavity and also maxillary ▶

The contributions of Kathleen Bradbury-Golas to this chapter in prior editions are acknowledged here.

sinus cavity to assess for fluid in sinus cavity. Healthy sinuses contain air and light up symmetrically.

C. Palpate:

1. Palpate face and frontal maxillary sinuses for tenderness.

2. Examine head and neck for enlarged lymph nodes.

D. Percuss:

1. Percuss sinus cavities and mastoid bone.

2. Percuss chest for consolidation.

E. Auscultate:

1. Auscultate heart and lungs.

DIAGNOSTIC TESTS

A. Diagnosis may be made from history and physical. Other diagnostic tests include the following:

1. Best test: skin prick–puncture method to detect allergen sensitization.

2. Serum tests for allergen-specific IgE antibodies (e.g., IgE immunoassay).

DIFFERENTIAL DIAGNOSES

A. Allergic rhinitis.

B. URI.

C. Medication-induced rhinitis (consider systemic medications, nasal decongestants, or cocaine abuse).

D. Sinusitis.

E. Nasal polyps.

F. Work-related (e.g., irritant, corrosive).

G. Endocrine conditions such as hypothyroidism.

H. Influenza.

PLAN

A. General interventions:

1. Avoid allergens (most effective treatment).

2. Keep bedroom as allergen-free as possible. Implement changes in work and home environments; consider removing upholstery and carpets to reduce reservoirs for dust mites.

B. Client teaching: *See Client Teaching Guide for this chapter, "Allergic Rhinitis."*

C. Pharmaceutical therapy:

1. Topical therapy:

a. Topical corticosteroids are the single most effective maintenance therapy for allergic rhinitis. They have been proven more effective than oral or topical antihistamines. They are first-line agents for older adults.

i. Fluticasone propionate (Flonase), 50 mcg/spray, two sprays each nare daily.

ii. Mometasone (Nasonex), 50 mcg/spray, two sprays each nare daily.

iii. Beclomethasone (Qnasl), 80 mcg/spray, two sprays each nare daily.

iv. Budesonide (Rhinocort), 32 mcg/spray, two sprays each nare daily.

v. Triamcinolone (Nasacort), 55 mcg/spray, two sprays each nare daily.

vi. Fluticasone furoate (Flonase Sensimist), 27.5 mcg/spray, two sprays each nare daily.

b. Topical antihistamine: *Consider these options in place of oral antihistamine for older adults.*

i. Azelastine hydrochloride (Astelin), 0.1% or 0.15% spray, one to two sprays each nare, one to two times daily.

ii. Olopatadine (Patanase), 665 mcg/spray, two sprays each nare, BID.

c. Other agents:

i. Nasal saline:

1) Nasal spray or irrigations help lavage irritants from the mucosa.

2) Saline gel can help lubricate nasal mucosa to help prevent dryness.

ii. Ipratropium bromide (Atrovent nasal), 0.03% spray, two sprays each nare, two to three times daily.

1) Useful in decreasing rhinorrhea.

iii. Oxymetazoline (Afrin), 0.05% spray, one to two sprays each nare, one to two times daily.

1) **Must not use longer than 3 days due to rebound congestion.**

2) Decongestants are NOT recommended as monotherapy in chronic treatment but can help with severe nasal congestion.

iv. Phenylephrine (Neo-Synephrine), 1% spray or drops, two sprays/drops in each nare Q4H.

1) Therapy should not last longer than 3 days.

2) Decongestants are NOT recommended as monotherapy in chronic treatment but can help with severe nasal congestion.

2. Oral therapy:

a. Antihistamines (H_1 receptor antagonists) are the preferred agent. Several may need to be tried before an effective one is found. Drugs may also need to be switched occasionally to prevent tolerance. *Avoid first-generation, sedating antihistamines in older adults.*

i. Cetirizine (Zyrtec), 10 mg daily.

ii. Levocetirizine (Xyzal), 5 mg daily.

iii. Loratadine (Claritin), 10 mg daily.

iv. Fexofenadine (Allegra), 180 mg daily.

FOLLOW-UP

A. Client should return for follow-up visit in 2 to 3 weeks if necessary, earlier if symptoms worsen after 3 days of treatment.

CONSULTATION/REFERRAL

A. Refer the client to an allergist if symptoms continue and interfere with daily activities.

B. Allergist may prescribe immunotherapy following identification of offending allergens.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Over-the-counter (OTC) antihistamines such as diphenhydramine HCl (Benadryl) may be used for up to 5 days.

2. OTC decongestants such as oxymetazoline HCl (Afrin) may be used up to 3 days.

B. Geriatrics:

1. Beers considerations:

a. Antihistamines are effective for this population; however, there are more side effects and drug interactions in this group. Consider age and possible drug–drug interactions when prescribing medications for allergic rhinitis.

b. Anticholinergic effects in older adults are sedation, bladder/bowel retention, confusion, delirium, high risk of falls, and being prone to accidents.

c. Acetylcholinesterase inhibitors often prescribed for cognitively impaired geriatrics will increase anterior rhinorrhea.

d. Best tolerated medications for the elderly population that offer fewer side effects include the following:
i. Intranasal steroid sprays.
ii. Nasal saline rinse and spray.
iii. Most second-generation antihistamines, such as cetirizine, fexofenadine, and loratadine.
iv. Topical (nasal) antihistamine sprays.
v. Limited Benadryl might be appropriate for an acute, severe allergic reaction.

2. Studies show that sleep disordered breathing is more prevalent in the elderly with allergic rhinitis. Sleep efficiency was lower and waking after sleep onset was longer. It is imperative that clinicians obtain a thorough sleep history and assess for any signs/symptoms of sleep deprivation and decreased cognitive alertness with this population to maintain safety and quality of life.

3. The elderly population tends to be undertreated. Nasal steroid sprays are first-line agents and minimally sedating antihistamines can be used. Note that some may experience adverse anticholinergic side effects and starting with lower doses should be considered. Topical antihistamines are a good option and may be initiated prior to trial of oral antihistamines.

4. Common allergens for geriatrics with persistent allergic rhinitis are house dust mites, pet dander, and cockroach allergens. Assessment of elderly home environments and living conditions could initiate improved avoidance measures and reduce the risk of allergic outbreaks.

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EPISTAXIS

DEFINITION

A. Epistaxis is a nosebleed or hemorrhage from the nose. This can be classified as anterior or posterior, depending on the source of bleeding.

INCIDENCE

A. Nosebleeds occur in approximately 60% of Americans at least once in their lifetime. However, fewer than 10% will seek medical care for a nosebleed. In the adult population, most cases occur between the ages of 45 and 65.

PATHOGENESIS

A. Epistaxis is caused by disruption of the nasal mucosa. More than 90% of nosebleeds are related to local irritation rather than underlying anatomic lesions and are self-limiting. Most start in the anterior nasal cavity (Kiesselbach plexus).

B. Posterior bleeds arise most commonly from the sphenopalatine artery and result in significant hemorrhage. Posterior nosebleeds require prompt referral to ED for care by an otolaryngologist and possible nasal packing, cautery, and/or hospital admission.

C. Nosebleeds more commonly occur during the winter months. They may also be associated with other conditions such as upper respiratory infections (URIs) or allergic rhinitis.

PREDISPOSING FACTORS

A. Local trauma, usually from nose picking.
B. Acute inflammation from a URI (e.g., common cold, acute sinusitis, and allergic rhinitis).
C. Vigorous nose-blowing.
D. Inhalation of chemical irritants.
E. Drying and crusting of nasal septum.
F. Trauma.
G. Cocaine use.
H. Pregnancy.
I. Neoplasm.
J. Systemic causes:
1. Bleeding disorders (most common).
2. Hypertension.
3. Heart failure.
4. Anticoagulation.

COMMON COMPLAINT

A. Common complaint is unusually severe or recurrent nosebleeds.

OTHER SIGNS AND SYMPTOMS

A. Anterior epistaxis:
1. Unilateral.
2. Continuous, mild/moderate bleeding from nasal septum.
B. Posterior epistaxis:
1. Brisk (arterial) bleeding.
2. Blood flowing into pharynx (indicates a more serious problem).

SUBJECTIVE DATA

A. Inquire about amount, duration, and frequency of bleeding. Ask about what side the bleeding started.
B. Ask about use of oral anticoagulants, aspirin, or aspirin-containing compounds (e.g., Pepto-Bismol, aspirin, Excedrin).
C. Ask about recent or current URIs, family history of abnormal bleeding, recent surgery, or trauma.
D. Rule out pregnancy.
E. Ask about possible foreign body in the nose.
F. Ask about cocaine use or occupational exposure to irritants or chemicals.
G. If the client has a history of nosebleeds, how did the client treat previous nosebleeds?
H. Has the client ever been evaluated for a blood clotting abnormality, such as thrombocytopenia or platelet dysfunction?
I. Does the client complain of bruising easily, melena, or heavy menstrual periods?
J. Ask about family history of bleeding disorders, such as hemophilia or von Willebrand disease.

PHYSICAL EXAMINATION

A. Check temperature, blood pressure (check for orthostatic hypertension), pulse, and respirations. **If nasal packing is required, take precaution and monitor client closely for vasovagal episode during insertion of nasal packing.**
B. Inspect:
1. Check airway patency with client sitting and leaning forward. ▶

2. Observe skin, mucous membranes, and conjunctiva for rash, pallor, purpura, petechiae, and telangiectasias.
3. Perform full eye exam, noting pupillary response.
4. Examine nose for septal perforations and ulcerations, which indicate cocaine use. Collagen diseases (such as lupus) are occasionally responsible for ulceration. Epistaxis is rare in hemophiliacs without trauma but is characteristic of von Willebrand disease.
5. Examine nasal discharge: A unilateral foul discharge with blood indicates a foreign body in the nose.
6. After bleeding has stopped:
a. Inspect nasal mucosa for color, discharge, masses, lesions, and swelling of turbinates.
b. Inspect the nasal septum for alignment, septal perforation, and crusting.

C. Auscultate:
1. Auscultate heart and lungs.

D. Palpate:
1. Check for enlarged lymph nodes in the neck to rule out sarcoidosis, tuberculosis, or malignancy.

E. Percuss:
1. Percuss sinuses.

DIAGNOSTIC TESTS

A. None required unless the client has recurrent or severe blood loss.
B. Drug screen, if indicated.
C. Hematocrit and hemoglobin if bleeding is severe.
D. Complete blood count (CBC) with differential.
E. Platelets, prothrombin time (PT), and partial thromboplastin time (PTT) if bleeding disorder is suspected.
F. Sinus films if recurrent sinus pain, tenderness, bleeding, or if nasal mass is suspected.

DIFFERENTIAL DIAGNOSES

A. Epistaxis.
B. Foreign body.
C. Septal deformity or perforation.
D. Nasal tumors or mass.
E. Coagulation disorder (von Willebrands disease).
F. Drug-induced coagulopathy.
G. Hypertension.
H. Pregnancy.

PLAN

A. General interventions:
1. The main goal is to stop bleeding or reduction of recurrence.

B. Client teaching: *See Client Teaching Guide for this chapter, "Nosebleeds."*

C. Treatment/management:
1. To control *anterior* bleeding:
a. Have client sit and lean forward to prevent swallowing of blood.
b. Initial tamponade:
i. Soak a cotton pledget in phenylephrine (Neo-Synephrine) or oxymetazoline (Afrin); place this plug into bleeding nostril and hold *bilateral* pressure over nasal alae for 10 to 15 minutes without releasing pressure.
ii. Remove and check for bleeding.
iii. If this fails, chemical cautery may be needed.
c. Cautery:
i. Only do cautery if bleeding site is visualized.
ii. Warn client that this may be a little painful. Anesthetize mucous membrane by applying cotton soaked with 2% lidocaine, lidocaine with epinephrine, or 4% cocaine.
iii. Apply silver nitrate by applying applicator tip to the site of bleeding for no longer than 10 seconds until a white precipitate forms. While silver nitrate needs moisture to work, it will only work on a relatively bloodless surface.
iv. Avoid overzealous cautery, which can lead to ulceration and perforation.
v. Avoid cautery of both sides of the septum, which can lead to tissue necrosis.
vi. If bleeding continues, repeat initial tamponade and cautery, then place a small amount of oxidized regenerated cellulose (Surgicel) against the bleeding artery. **If client continues to have high-flow epistaxis, nasal packing may need to be performed in emergency setting.**
vii. If bleeding stops, give client antibiotic ointment to apply gently with fingertip or cotton swab TID for 3 days. Saline gel or Vaseline can also be used.
2. To control *posterior* bleeding:
a. Have client sit and lean forward to prevent swallowing of blood.
b. As it can be difficult to determine the source of epistaxis, try to control bleeding first with the above steps of topical vasoconstrictor and anesthetic, then apply pressure.
c. **Consult a physician. The client needs ED care immediately due to rapid blood loss.**
d. Monitor vital signs (blood pressure and pulse) and order hematocrit; blood type and crossmatch may be needed.

FOLLOW-UP

A. Anterior epistaxis: as needed.
B. Posterior epistaxis: needs admission to hospital and referral to an otolaryngologist.

CONSULTATION/REFERRAL

A. Anterior epistaxis: Referral to an otolaryngologist is recommended for unsuccessful cessation of hemorrhage or persistent, recurrent epistaxis despite interventions.
B. Posterior epistaxis: Refer to a physician and/or otolaryngologist immediately.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Nosebleeds are common.
2. Suggest use of saline spray to keep mucous membranes moist and use humidifier at bedtime.
3. Follow use of saline spray with Vaseline applied with Q-tip daily to prevent recurrent nosebleeds.

B. Geriatrics:
1. In the geriatric population, epistaxis is classically associated with hypertension, arteriosclerosis, coagulation disorders, chronic anemia, angina pectoris, and cerebral hemorrhages. Be sure to ask if the client has been prescribed daily aspirin or other anticoagulation and review education with client/family regarding the benefits as well as the risks.
2. Applying lipid-based lubricant on rims of nostrils to maintain mucosal moisture may cause lipoid pneumonia in the elderly. Use saline/water-based lubricant instead.
3. Although epistaxis with geriatrics may indicate an underlying serious condition, it might also be benign, related to natural aging: inflammation of mucosa, injury ▶

(scratching too hard), dryness, or side effects from medications.

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NONALLERGIC RHINITIS

DEFINITION

A. Nonallergic rhinitis is rhinitis without an identifiable etiology and without an immunoglobulin E dependent component. It is characterized by nasal congestion, postnasal drainage, rhinorrhea, and sneezing, and is associated with some degree of autonomic dysregulation. It is classified in various ways: idiopathic, vasomotor, atrophic, geriatric, drug-induced, gustatory, occupational, or rhinitis of pregnancy.

INCIDENCE

A. Nonallergic rhinitis may be responsible for 17% to 52% of all rhinitis cases in adults.

B. Of nonallergic rhinitis clients, 70% present with symptoms after 20 years of age (whereas a majority of allergic rhinitis clients present before the age of 20).

PATHOGENESIS

A. No single theory of pathogenesis for chronic nonallergic rhinitis has been determined, but it can be divided into noninflammatory and inflammatory. It is a diagnosis of exclusion.

B. Clients exhibit clinical hypersensitivity of nasal mucosa to certain odors and irritants.

C. Atrophic and geriatric rhinitis result from progressive atrophy of nasal mucosa.

D. Overuse of topical nasal decongestants or cocaine and some systemic medications can result in nasal dysfunction.

E. Rhinitis of pregnancy results from hormonal changes; symptoms abate with delivery.

PREDISPOSING FACTORS

A. Adulthood.

B. Abrupt changes in temperature, odors, and emotional stress.

C. Other predisposing factors depend on type (e.g., overuse of medications, side effects of systemic medications, and occupational environment).

COMMON COMPLAINTS

A. Nasal congestion.

B. Sneezing.

C. Clear rhinorrhea.

D. Coughing.

E. Symptoms are often exacerbated by weather conditions.

SUBJECTIVE DATA

A. Ask about the onset, duration, and course of symptoms. Clinical history is the **most important** tool used to make distinction between allergic and nonallergic rhinitis.

B. Inquire about the color and other characteristics of nasal discharge.

C. Ask about other discomforts and exposure to people with similar symptoms.

D. Inquire about seasonal impact on symptoms, effect of weather changes on symptoms, previous treatments, and results.

E. Rule out pregnancy. Ask female clients about birth control method, specifically contraceptives.

F. Ask about use of prescription drugs, over-the-counter (OTC) drugs (especially aspirin), and illicit drugs (cocaine).

G. Review medical history for other respiratory problems, such as asthma, emphysema, or chronic bronchitis.

PHYSICAL EXAMINATION

A. Check temperature and blood pressure.

B. Inspect:

1. Observe general appearance.
2. Inspect conjunctivae for "allergic shiners" (dark circles under eyes), tearing, and eyelid swelling.
3. Examine ears for signs of otitis media (red, bulging, perforated tympanic membrane, and purulent drainage).
4. Examine nose for redness, swelling, polyps (soft, pedunculated, nontender, pale-gray smooth structures), enlarged turbinates, foreign objects, septal deviation, septal perforation (sign of cocaine abuse), ischemia, mucosal injury, atrophy, and "cobblestoned" pharyngeal mucosa (sign of allergy). **However, none of these findings are diagnostic and the mucosa may appear relatively normal in nonallergic rhinitis.**

C. Auscultate:

1. Auscultate heart and lungs.

D. Percuss:

1. Percuss sinus cavities and mastoid process.
2. Percuss chest for consolidation.

E. Palpate:

1. Palpate face for sinus tenderness.
2. Palpate head and neck for enlarged lymph nodes.

DIAGNOSTIC TESTS

A. Nonallergic rhinitis is a diagnosis of exclusion. Diagnosis is based on characteristic history and physical exam, as well as absence of clinical allergy to aeroallergens.

B. Skin testing for allergies may be done to exclude allergic rhinitis.

DIFFERENTIAL DIAGNOSES

A. Nonallergic rhinitis.

B. Allergic rhinitis.

C. Upper respiratory infection (URI).

D. Foreign body.

E. Sinusitis.

F. Deviated septum.

G. Tumor.

H. Nasal polyps.

I. Endocrine conditions such as hypothyroidism and pregnancy.

J. Drug use: oral contraceptives, aspirin, alpha-adrenergic blockers, cocaine, and nasal decongestant overuse.

PLAN

A. General interventions:

1. Identify triggers and address alleviating triggers with avoidance/minimization.

B. Client teaching:

1. Teach the client the significance of individual triggers for nonallergic rhinitis. Encourage use of a journal to learn personal triggers.

2. Avoid triggers such as smoking, smoke-filled rooms, wood-burning stoves/fireplaces, sprays, and perfumes.

3. Other triggers may include weather changes, hormonal changes, and medications.

4. Teach methods of treatment and identify treatments that work best for the client.

5. Encourage use of neti pot or nasal saline flush with warm saline water daily to cleanse sinus cavity. Cleansing the sinus cavity daily will help remove foreign materials inhaled and will also help with tissue edema. Clean pot after each use and allow to air-dry.

C. Pharmaceutical therapy:

1. Monotherapy versus combination therapy:

a. Clients with mild disease may be adequately treated with intranasal corticosteroid or intranasal antihistamine spray. For clients who are more symptomatic, clinical evidence has shown combination therapy to be more effective, with both daily intranasal corticosteroid and antihistamine spray.

b. Topical corticosteroids:

i. Fluticasone propionate (Flonase), 50 mcg/spray, two sprays each nare daily.

ii. Mometasone (Nasonex), 50 mcg/spray, two sprays each nare daily.

iii. Beclomethasone (Qnasl) 80 mcg/spray, two sprays each nare daily.

iv. Budesonide (Rhinocort), 32 mcg/spray, two sprays each nare daily.

v. Triamcinolone (Nasacort), 55 mcg/spray, two sprays each nare daily.

vi. Flonase furoate (Flonase Sensimist), 27.5 mcg/spray, two sprays each nare daily.

c. Topical antihistamine:

i. Azelastine hydrochloride (Astelin), 0.1% or 0.15% spray, two sprays each nare one to two times daily.

ii. Olopatadine (Patanase), 665 mcg/spray, two sprays each nare BID.

2. If rhinorrhea is the dominant or only symptom (such as in gustatory rhinitis or rhinitis in older adults), recommend anticholinergic nasal spray:

a. Ipratropium (Atrovent) 0.03% or 0.06%, two sprays each nare, two to three times daily.

3. Decongestants: Oral and nasal decongestants are NOT recommended unless antihistamines and nasal steroids have failed. Examples include oral pseudoephedrine and nasal oxymetazoline (Afrin).

a. **Therapy should not continue longer than 3 days to prevent effects of rebound congestion.**

FOLLOW-UP

A. Clients receiving chronic therapy for rhinitis should be seen every 3 to 12 months, depending on severity of symptoms.

CONSULTATION/REFERRAL

A. Consult with a physician if symptoms continue despite treatment.

B. If treatment fails, refer the client to an allergist for testing and/or ear, nose, and throat specialist to consider surgical approaches for clients with severe, chronic nonallergic rhinitis.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Reassure pregnant clients that rhinitis is a common hormonal response. Nonallergic rhinitis is not contagious and cannot cross the placenta.

B. Geriatrics:

1. Obstructive sleep apnea is associated with allergic and nonallergic rhinitis. Obtain a thorough sleep history and look for any signs/symptoms of sleep deprivation and/or decreased cognitive alertness.

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SINUSITIS

DEFINITION

A. Sinusitis (often referred to as rhinosinusitis) is inflammation of the mucous membranes and the paranasal sinuses. It may be acute, subacute, or chronic.

1. Acute sinusitis: abrupt onset of inflammation and/or infection with symptom resolution after therapy, lasting less than 4 weeks.

2. Subacute sinusitis: persistent symptoms despite therapy, lasting 4 to 12 weeks.

3. Chronic sinusitis: episodes of prolonged (>12 weeks) inflammation and/or infection.

INCIDENCE

A. Sinusitis is very prevalent, affecting millions of people in the United States each year. It is estimated that one in seven individuals in the United States will experience an episode of acute sinusitis each year. Incidence is higher among females, and among all adults it is highest in those aged 45 to 64. Chronic sinusitis affects between 5% and 12% of the general population.

PATHOGENESIS

A. Etiology of acute rhinosinusitis involves factors such as environmental, anatomic, and systemic. These factors can occur at the same time and predispose clients not only to acute sinusitis but also to the development of chronic sinusitis.

B. The most common cause of acute sinusitis is viral infection, with the most common causes being rhinovirus, influenza virus, and parainfluenza virus. Acute bacterial rhinosinusitis occurs in 0.5% to 2% of all sinusitis episodes. The most common bacterial organisms are *Streptococcus pneumoniae, Haemophilus influenzae*, and *Moraxella catarrhalis*. If acute sinusitis infections are not resolved or become recurrent, *Staphylococcus aureus* or *Pseudomonas aeruginosa* can become dominant.

C. The transition from viral to bacterial has been studied extensively and is thought to be secondary to decreased mucociliary clearance and consequent colonization of bacteria on retained mucus secretion from the obstruction of outflow. The sinonasal mucosa inflammation from the triggering agent stuns the cilia and mucus remains trapped in the sinuses, and ▶

bacteria proliferate. Anatomic abnormalities can also interfere with normal mucociliary clearance and can prolong illness.

D. Other causes of acute sinusitis are fungi, dental abscess, and autoimmune reactions. Fungal sinusitis tends to occur in immunosuppressed clients.

PREDISPOSING FACTORS

A. Recent upper respiratory infection (URI).
B. Allergic rhinitis.
C. Cigarette smoke (firsthand or secondhand).
D. Air pollutant exposure.
E. Sinonasal anatomy (septal deviation, septal spurs, turbinate hypertrophy, Haller cells, adenoidal hypertrophy, or obstructive mass).
F. Ciliary dysfunction (as in Kartagener syndrome and cystic fibrosis).
G. Dental anatomy/abscess.
H. Asthma.
I. Neoplasms.
J. Immunodeficiency (often associated with recurrent acute or chronic sinusitis).
K. Trauma.
L. Diving and swimming.
M. Air travel or rapid changes in altitude.

COMMON COMPLAINTS

A. Mucopurulent rhinorrhea.
B. Headache and/or facial pain.
C. Dental or ear pain.
D. Loss of smell (anosmia).
E. Nasal congestion.

OTHER SIGNS AND SYMPTOMS

A. Fever (mostly associated with acute sinusitis).
B. Malaise or fatigue.
C. Cough.
D. Periorbital edema.
E. Halitosis.
F. Snoring, mouth-breathing.
G. Hyponasal speech.

POTENTIAL COMPLICATIONS

A. Consider immediate ear, nose, and throat referral.
  1. Meningitis (symptoms are increased fever, stiff neck).
  2. Preseptal or orbital cellulitis.
  3. Subperiosteal or intracranial abscess.
  4. Epidural abscess.
  5. Cavernous sinus thrombosis (acute thrombophlebitis due to infection in the area where veins drain into the cavernous sinus).
  6. Osteomyelitis of the sinus bones.

SUBJECTIVE DATA

A. Elicit onset, duration, and course of symptoms. Have symptoms lasted longer than 10 days or longer than 12 weeks?
B. Inquire whether seasons affect symptoms.
C. Ask the client about recent URI and how it was treated. Did the client receive antibiotics? Did the client finish the full course of antibiotics?
D. Ask about allergies.
E. Inquire about recent dental problems, especially dental abscesses.
F. Find out what home therapies and over-the-counter (OTC) medications the client tried before the office visit.
G. Ask if the client took a trip recently, especially by airplane.
H. Inquire whether the client was swimming or diving recently.
I. Review the client's medical history for cystic fibrosis, asthma, nasal abnormalities (e.g., deviated septum), and other respiratory problems.

PHYSICAL EXAMINATION

A. Check temperature, blood pressure, pulse, and respirations.
B. Inspect:
  1. Observe eyes for periorbital swelling, "allergic shiners" (dark circles under eyes), tearing, and signs of orbital cellulitis (conjunctival edema, drooping lid, decreased extraocular motion, and vision loss).
  2. Examine ears.
  3. Inspect the nose for erythema, edema, discharge, lack of nostril patency, septal deviation and polyps, and presence of a foreign body.
  4. Transilluminate maxillary and frontal sinuses in a darkened room. Absence of light reflection is not definitive.
  5. Examine the mouth and pharynx for erythema and tonsillar enlargement, check teeth for uneven surfaces (sign of grinding), and check retropharynx for evidence of postnasal drip.
C. Auscultate:
  1. Auscultate heart and lungs.
D. Palpate:
  1. Palpate all sinuses for pain and tenderness.
    a. Frontal sinusitis: pain on palpation of forehead.
    b. Maxillary sinusitis: the cheeks are tender, or the client may present with jaw and tooth pain.
    c. Ethmoid sinusitis: swelling and tenderness in the eyelids and surrounding tissue.
  2. Palpate neck for lymphadenopathy.
E. Perform neurologic exam:
  1. Evaluate for signs of meningeal irritation, assessing for Brudzinski sign, Kernig sign, and nuchal rigidity.

DIAGNOSTIC TESTS

A. Diagnosis is usually made through history and physical.
B. If the disease is refractory to initial treatment or becomes recurrent or chronic, further investigation may be necessary.
  1. Rule out immunodeficiencies (cystic fibrosis, Wegener granulomatosis, sarcoidosis, Churg–Strauss syndrome).
  2. Skin testing for allergies.
  3. Nasal endoscopy to detect abnormalities of the nasal passage (polyps, pus). Culture can be obtained for culture-directed antimicrobial treatment.
  4. Sinus CT is the best imaging method for viewing paranasal sinuses and reveals extent of inflammation and disease. CT is not recommended for routine investigation for acute rhinosinusitis. It is recommended for chronic and recurrent sinusitis, for clients in immunocompromised state, or if there is concern for complications (e.g., fungal sinusitis, disease extending outside of the sinus cavities).

DIFFERENTIAL DIAGNOSES

A. Sinusitis.
B. Headache (cluster, migraine).
C. Rhinitis (allergic or nonallergic).
D. Temporomandibular joint disease.
E. Trigeminal neuralgia.
F. Cerebral spinal fluid rhinorrhea.
G. Tumor/neoplasm.
H. Autoimmune disease.
I. Odontogenic disease.

PLAN

A. General interventions:

1. Teach client to avoid smoking and secondhand smoke.

2. Drinking extra fluids helps loosen secretions and hydrate the body.

3. Encourage the client to use medications as prescribed. OTC medications such as antihistamines and decongestants should be used with caution.

4. Application of warm, moist compress to the face several times a day will help with discomfort.

5. Humidifiers should be used daily. Advise client to clean humidifiers on a regular basis.

6. Nasal saline to the nares three times a day will help keep nasal passages moist.

B. Client teaching: *See Client Teaching Guide for this chapter, "Sinusitis."*

C. Pharmaceutical therapy:

1. Initial treatment with antibiotics should be 5 to 7 days, and if not improving on initial therapy can extend to 10 to 14 days.

a. First-line treatment: Augmentin 875 mg BID or 500 mg TID.

b. At risk of pneumococcal resistance or failed initial therapy (older than 65 years, recent hospitalization, recent use of antibiotics in the past month, or immunocompromised client): Augmentin 2,000 mg BID.

c. Beta-lactam allergy: doxycycline 100 mg BID or 200 mg daily.

d. Respiratory fluoroquinolone can be considered as alternative for penicillin-allergic clients but should be reserved for those who have no alternative treatment options: levofloxacin 750 mg or 500 mg daily or moxifloxacin 400 mg daily.

e. The same antibiotics can be used for chronic sinusitis, but treatment should last 3 to 4 weeks.

2. Oral and topical decongestants may be used to correct the underlying edematous mucosa (use cautiously with hypertension).

a. Oxymetazoline (Afrin) 0.05% spray, one to two sprays each nare, one to two times daily. DO NOT use longer than 3 days.

b. Phenylephrine (Neo-Synephrine) 1% spray or drops, one to two sprays/drops each nare Q4H.

c. Pseudoephedrine HCl 30 to 60 mg up to TID.

d. Nasal saline spray throughout the day and saline irrigation BID.

3. Intranasal corticosteroid sprays may be used to decrease nasal inflammation.

a. Fluticasone propionate (Flonase), 50 mcg/spray, two sprays each nare daily.

b. Mometasone (Nasonex), 50 mcg/spray, two sprays each nare daily.

c. Beclomethasone (Qnasl) 80 mcg/spray, two sprays each nare daily.

d. Budesonide (Rhinocort), 32 mcg/spray, two sprays each nare daily.

e. Triamcinolone (Nasacort), 55 mcg/spray, two sprays each nare daily.

f. Flonase furoate (Flonase Sensimist), 27.5 mcg/spray, two sprays each nare daily.

4. Antihistamines are recommended to block histamine production in response to the allergy triggers and prevent allergy symptoms.

a. Cetirizine (Zyrtec), 10 mg daily.

b. Levocetirizine (Xyzal), 5 mg daily.

c. Loratadine (Claritin), 10 mg daily.

d. Fexofenadine (Allegra), 180 mg daily.

FOLLOW-UP

A. Recheck the client in 5 to 7 days after initial therapy, and if no improvement consider extending therapy or alternative therapy.

B. If the client is not improving and may be resistant to antibiotics, they may be switched to a different antibiotic for 14 days.

CONSULTATION/REFERRAL

A. Admit the client to the hospital if they have signs and symptoms that the disease has spread intracranially or intraorbitally.

B. Refer the client to an otolaryngologist if they do not improve in 4 weeks or after failed medical therapy.

C. Refer the client to an otolaryngologist for suspected neoplasm, abscess, osteomyelitis, meningitis, or sinus thrombosis.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Amoxicillin/Augmentin are the desired antibiotics for use during pregnancy.

2. Clients who are allergic to penicillin should be treated with azithromycin.

3. Doxycycline and fluoroquinolones are NOT recommended during pregnancy.

B. Geriatrics:

1. Nose-blowing precautions should be used for clients currently prescribed warfarin (Coumadin) and/or aspirin and other anticoagulants.

2. Avoid use of trimethoprim-sulfamethoxazole (TMP-SMX) with warfarin because the medication can cause a significant increase in prothrombin time (PT)/international normalized ratio (INR). Also avoid TMP-SMX in clients taking angiotensin-converting enzyme inhibitor or angiotensin receptor blocker or clients with decreased creatinine clearance to avoid hyperkalemia.

3. Inappropriate antibiotic therapy with geriatrics can cause significant risks and adverse effects that include drug interactions and contraindications, side effects related to polypharmacy, age-related metabolisms, risks of multidrug-resistant organisms, and *Clostridium difficile*:

4. Home treatment suggestions for geriatrics:

a. Safe (reliable brand) home humidifier with automatic shutoff and adequate weight balance to prevent hot steam spillage and avoid burns.

b. Drinking plenty of water or hot/cold herbal teas—staying well hydrated.

c. Placing a warm, moist washcloth over nose and sinuses.

d. Using gentle saline rinses; or while showering, breathing in steam an extra couple of minutes.

e. Minimizing exposure to allergens (dust and dander).

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CLIENT TEACHING GUIDE

ALLERGIC RHINITIS

PROBLEM Allergic rhinitis is a chronic or recurrent condition. Common symptoms are nasal congestion, sneezing, and clear nasal discharge. It is not contagious, so you cannot catch it from anyone and you cannot spread it to others.

CAUSE You are having an allergic response after being exposed to an allergen.

PREVENTION

A. The best prevention is to avoid things you know you are allergic to, for example, smoke (cigarette, cigar, wood smoke), pollens and molds, animal dander, dust mites, and indoor inhalants such as hair spray and other aerosol spray products.
B. Target your bedroom as "allergy free" by removing carpets, damp mopping the floors weekly, hanging washable curtains instead of blinds, removing books and stuffed animals, using foam pillows, and encasing the pillows and mattress in plastic.
C. Do not blow your nose too frequently or too hard. It may cause your eardrum to perforate (tear). Blow through both nostrils at the same time to equalize the pressure.
D. Use tissues when you blow your nose. Dispose of them and then wash your hands. If no tissue is available, do the "elbow sneeze" into the bend of your arm (away from your open hands). Always wash your hands.

TREATMENT PLAN

A. Use the air conditioner in your house and car to reduce exposure to pollens.
B. Use an air filtration system in your house or buy a small one for your bedroom.
C. Dust your house often, using a cloth and cleaner or polish to keep dust from flying.
D. Allergy testing may need to be done if you have had allergies for a long time. Ask your healthcare provider about a consultation with an allergist.

Activity: There are no activity restrictions. However, you may want to exercise indoors during the spring, summer, and fall when pollen counts are high.

Diet: Eat well-balanced meals. Drink at least six to eight glasses of liquid a day.

Medications: Common medications used include antihistamines, decongestants, and nasal sprays.
A. Antihistamines: Some antihistamines may cause drowsiness; use with caution. You may consider using a different antihistamine during the day that does not cause drowsiness.
B. Decongestants: Decongestants may increase blood pressure and may also interact with other medications. Please consult with your provider before using these medications.
C. Nasal sprays: Nasal saline spray is safe to use several times a day. Nasal decongestant sprays may be used for a short period of time. Do NOT use longer than 3 days to prevent rebound side effects from this medication. Consult with your provider if using a nasal decongestant spray.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. You experience trouble breathing or catching your breath.
B. You have asthma and your symptoms worsen.
C. Your symptoms are not any better after using the medications for 3 complete days.
D. Your nasal discharge changes to a greenish color.
E. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

NOSEBLEEDS

PROBLEM Most nosebleeds stop within 10 minutes. **If you have trouble breathing with a nosebleed, *call 911***.

CAUSE Nosebleeds may be caused by several problems:
A. Trauma from nose picking or forcefully blowing the nose.
B. Chronic sinus infections.
C. Allergies.
D. Drugs, including over-the-counter medications such as aspirin and Pepto-Bismol, or street drugs such as snorted cocaine.
E. Exposure to irritants.

PREVENTION
A. Avoid picking your nose. Keep fingernails trimmed short.
B. Do not blow your nose too frequently or too hard (it may also cause eardrum tearing).
C. Blow your nose through both nostrils at the same time to equalize pressure.
D. Use a humidifier in your home, or place a container of water near the radiator.
E. Keep the inside of your nose moist and promote healing with a nasal saline spray or gel.
F. Avoid smoking and secondhand smoke.

TREATMENT PLAN
A. If you experience a nosebleed, take these steps:
  1. Sit up and lean forward.
  2. Apply pressure to the bridge of your nose for 10 to 15 minutes to stop the blood flow.
  3. If the bleeding continues, spray Afrin into your nostril.
  4. If the bleeding still continues, lightly soak a cotton ball with the nasal spray, insert it into your nose, and press.
  5. If the bleeding stops, use nasal saline spray and gel to keep the area moist and promote healing. Avoid picking your nose or hard nose-blowing.
B. If the bleeding continues, seek medical care.

Activity: Avoid or limit the following activities for 3 to 5 days after a nosebleed:
A. Heavy lifting.
B. Straining.
C. Bending over from the waist.
D. Very hot showers.

Diet: Avoid hot, spicy foods for 3 to 5 days after a nosebleed.

Medications: Avoid medications that increase bleeding, such as aspirin and Pepto-Bismol.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. Bleeding does not stop with pressure or nasal spray applied to the bleeding site.
B. You keep having nosebleeds (more than two in a week or four in a month).
C. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

SINUSITIS

PROBLEM Sinusitis (sinus infection) is classified as an acute, subacute, or chronic condition. In acute sinusitis, the infection is resolved after treatment. In subacute sinusitis, there is a persistent, yellow to green nasal discharge despite treatment. In chronic sinusitis, episodes of prolonged inflammation continue longer than 3 months despite treatment.

CAUSE Sinusitis occurs when the mucous lining in your sinus cavities becomes inflamed and infected with viruses, bacteria, or allergen. This can occur after a cold or tooth abscess.

PREVENTION

A. If you have a tooth abscess, see your dentist and finish all your antibiotics.
B. Do not blow your nose too frequently or too hard. It may cause your eardrum to perforate (tear). Blow through both nostrils at the same time to equalize pressure.
C. To prevent spreading germs to others, cover your mouth when you cough.
  1. Use tissues when you blow your nose. Dispose of them and then wash your hands.
  2. If no tissue is available, do the "elbow sneeze" into the bend of your arm (away from your open hands).
D. Always wash your hands after coughing or using tissues.

TREATMENT PLAN

A. Avoid smoking and secondhand smoke.
B. Use steam inhalation to liquefy secretions.
C. Use a room humidifier. Keep your humidifier clean—it can grow bacteria.

Activity: There are no activity restrictions; however, diving, swimming, and flying may increase the occurrence of symptoms or make them worse. Make sure to get plenty of rest each day.

Diet: Eat a healthy diet. Drink at least 8 to 10 glasses of liquid every day.

Medications: Take all of your prescribed antibiotics (if indicated), even if you feel better.

Over-the-counter medications:
A. Pain relievers: ibuprofen (Advil) or acetaminophen (Tylenol) as needed for facial pain.
B. Antihistamines: Some antihistamines may cause drowsiness. Use with caution. You may consider using an antihistamine during the day that does not cause drowsiness.
C. Decongestants: Decongestants may increase blood pressure and may also interact with other medications. Please consult with your provider before using these medications.
D. Nasal sprays: Nasal saline spray is safe to use in the nose several times a day. Nasal decongestant sprays may be used for a short period of time. Do NOT use longer than 3 days to prevent rebound side effects from this medication. Consult with your provider if you are using a nasal decongestant spray.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Call the Office If:
A. Your eyelids begin to swell or droop, or you experience decreased vision.
B. You have stiffness in your neck or increased fever.
C. You have asthma and you are getting worse.
D. You begin vomiting and are unable to keep down your antibiotic.
E. You are a diabetic and your blood sugars are elevated, or you notice ketones in your urine.
F. Other: _____

Phone: _____

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CHAPTER 11

THROAT AND MOUTH GUIDELINES

Jill C. Cash and Kathleen Bradbury-Golas

DENTAL ABSCESS

DEFINITION

A. A dental abscess is a space infection of the gingival or periodontal tissues.

INCIDENCE

A. The incidence is unknown.

PATHOGENESIS

A. An abscess occurs when bacteria gain access into the gingiva or periodontal tissues. Dental infections are often polymicrobial and usually caused by anaerobic gram-negative rods and gram-positive cocci.

PREDISPOSING FACTORS

A. Poor dental hygiene.
B. Dental caries.
C. Low socioeconomic status.
D. Poor nutrition.
E. Physical and mental disabilities.

COMMON COMPLAINTS

A. Throbbing pain at infected site that may radiate to jaw, ears, cheek, neck, head.
B. Swelling (gingival edema).
C. Difficulty chewing with tooth due to pain.

OTHER SIGNS AND SYMPTOMS

A. Fever.
B. Warmth, redness.
C. Loss of appetite.
D. Heat and cold sensitivity.
E. Halitosis.
F. Trismus (unable to open mouth).

POTENTIAL COMPLICATIONS

A. The risk of complications increases with valvular disease. The following are complications:
1. Sepsis.
2. Leukocytosis associated with facial cellulitis.
3. Hematogenous dissemination to native or prosthetic heart valves, joints, or other devices.
4. Osteomyelitis of the jaw.

SUBJECTIVE DATA

A. Elicit information from the client regarding onset, duration, location, and quality of pain.
B. Note radiation of pain as well as alleviating or aggravating factors.
C. Note if pain is brought on by contact with hot, cold, or sweet substances; this may indicate periapical abscess or dental caries.
D. Ask if the client has a fever. If so, ask how high and for how long.
E. Inquire about history of mitral valve prolapse or rheumatic fever.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.
B. Inspect:
1. Inspect teeth for caries, mobility of teeth or protrusion from sockets, and gum disease.
2. Examine teeth for erosion, enamel decalcification, diminished tooth size, discoloration, and sensitivity to temperature changes.
C. Palpate:
1. Palpate neck and submental area for enlarged, tender lymph nodes.
D. Percuss:
1. Percuss all teeth. Tenderness is diagnostic of an abscess.
E. Auscultate:
1. Auscultate heart and lungs.

DIAGNOSTIC TESTS

A. CT.
B. X-ray (consider panoramic).
C. Serum labs: complete blood count (CBC) with differential.

DIFFERENTIAL DIAGNOSES

A. Dental abscess.
B. Periodontal disease.
C. Cellulitis.
D. Peritonsillar abscess.

PLAN

A. General interventions:
1. Treat immediate infection.
2. Refer to a dentist for immediate evaluation and treatment.
B. Client teaching:
1. Advise client to apply a heating pad to painful facial area for comfort.
2. Advise soft diet until pain resolves.

3. Review daily dental care and hygiene with client. Recommend regular brushing and dental flossing after meals, use of fluoride and/or antimicrobial mouth rinse, smoking cessation, reducing sugar intake, and regular dental appointments.

4. Chlorhexidine 0.12% oral mouth rinse may be used for gingivitis.

C. Pharmaceutical therapy:

1. Drug of choice: amoxicillin-clavulanic acid (Augmentin) 875 mg BID for 7 to 14 days. Dental consultation required.

2. Other medications (penicillin-allergic clients):

a. Clindamycin (Cleocin) 300 to 450 mg orally Q8H until dental consultation.

b. Cephalexin (Keflex) 500 mg Q6H until dental consultation.

c. **Levofloxacin plus metronidazole if clindamycin-resistant.**

3. For discomfort and fever: ibuprofen (Advil) 400 to 600 mg orally every 6 to 8 hours, not to exceed 1,200 mg/d. Tylenol 500 mg orally every 4 to 6 hours may be used for clients with renal impairment. Consider renal function when prescribing nonsteroidal anti-inflammatory drugs (NSAIDs) to older adults and geriatric clients.

FOLLOW-UP

A. Follow up 2 to 3 days after dental exam to evaluate results, as needed.

CONSULTATION/REFERRAL

A. Advise the client to see a dentist promptly, even if pain resolves.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Clients may safely have dental procedures during pregnancy.

2. X-ray films may be taken with lead shield over the client's abdomen.

3. Epinephrine and nitrous oxide should not be used during dental procedures.

4. Tetracycline should not be used; it causes staining of fetal bones and teeth.

B. Geriatrics:

1. Two primary oral factors with older adults' propensity for dental abscess are the following:

a. Xerostomia: hypofunction of salivary glands, medications (antihypertensives, antidepressants, antipsychotics), and diabetes.

b. Facial pain: root caries and reduced masticatory ability (could lead to malnutrition), which lowers the body's immune system.

2. Observational studies indicate that the elderly population with remaining teeth tend to have less dental abscesses or periodontal problems. They performed appropriate physical functioning, were less frail, and maintained better oral healthcare. These factors decrease the risk of health problems and infections that arise during the aging process.

3. When oral healthcare educational programs are implemented to geriatrics (in the community setting or long-term care facility), plaque levels improve and there is decreasing trend in gingival bleeding/infections. Chlorhexidine 0.12% mouth rinse may be used for gingivitis. Encourage elderly clients and their families to maintain oral healthcare and visit a dentist regularly.

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DYSPHAGIA

DEFINITION

A. Dysphagia is defined as difficulty swallowing.

1. Oropharyngeal dysphagia is defined as difficulty with the initial task of swallowing, which can be accompanied by choking, coughing, or a food block in the pharynx.

2. Esophageal dysphagia is defined as difficulty swallowing after initiation of swallowing, feeling food lodged in the esophagus. A motor disorder occurs when there is difficulty swallowing solids and/or liquids. This can be intermittent or progressive. A mechanical obstruction occurs when there is difficulty swallowing solids, which may be nonprogressive or progressive:

a. Nonprogressive disorders include an esophageal ring or eosinophilic esophagitis.

b. Examples of progressive disorders include peptic stricture (chronic heartburn is present) and esophageal/gastric cardia cancer.

3. Odynophagia is defined as pain with swallowing.

INCIDENCE

A. Dysphagia affects approximately 300,000 to 600,000 persons annually.

B. Approximately 15% of the elderly population are affected by dysphagia.

PATHOGENESIS

A. In advancing age, muscle strength and the range of motion of swallowing decline, which may impact the effectiveness of swallowing. A decrease in oral secretions, taste changes, and smell acuity may also impact the effectiveness of swallowing.

PREDISPOSING FACTORS

A. Age (elderly).

B. Neurologic conditions such as stroke, myasthenia, multiple sclerosis, Parkinson disease, and so forth.

C. Dementia.

D. Trauma or radiation to neck and head.

COMMON COMPLAINTS

A. Difficulty with swallowing.

B. Reflux of food/liquid into the nose.

OTHER SIGNS AND SYMPTOMS

A. Weight loss.

B. Nutritional deficiency.

C. Refusal to eat.

POTENTIAL COMPLICATIONS

A. Malnutrition.

B. Pneumonia.

SUBJECTIVE DATA

A. Ask the client if they have difficulty swallowing when initiating the swallow or a few seconds after trying to swallow. ▶

B. Does difficulty swallowing occur at every meal or snack? If so, is it getting worse?
C. Is it difficult to swallow solid foods, liquids, or both? Difficulty swallowing solids and liquids occurs when a motility disorder of the esophagus is present. Difficulty swallowing solids that generally progresses to liquids is commonly caused by a mechanical obstruction.
D. When swallowing occurs, does the client experience coughing or reflux, or does food go up through the nose?
E. Can the client identify a site where food commonly gets lodged?
F. Has the client noticed any change in appetite? Weight loss or weight gain? Nausea, vomiting, diarrhea, constipation, change in stools, heartburn, or chest pain?
G. Has the client started any new medications that have caused indigestion or reflux? Common medications that may cause reflux include bisphosphonates, ferrous sulfate, potassium chloride, ascorbic acid, tetracycline, and NSAIDs.
A. Does the client have a history of dry mouth/dry eyes indicating salivary production change (e.g., medications causing dry mouth/eyes include anticholinergics, antihistamines, and antihypertensive medications; radiation to head/neck; new diagnosis of Sjögren syndrome)?

PHYSICAL EXAMINATION

A. Check vital signs: temperature, pulse, respirations, and blood pressure.
B. Inspect:
  1. Inspect the oral cavity for lesions, ulcers, and poor dentition. Note muscle weakness or paralysis in movement of tongue.
  2. Inspect head and neck for any masses.
  3. Inspect face for muscle weakness and neck for swallowing difficulty.
  4. Assess all cranial nerves, focusing on cranial nerves V, VII, X, XI, and XII.
C. Palpate:
  1. Palpate the neck and supraclavicular lymph nodes for adenopathy or palpable masses.
D. Auscultate:
  1. Heart.
  2. Lungs.

DIAGNOSTIC TESTS

A. Upper endoscopy.
B. Barium swallow.
C. Motility testing.

DIFFERENTIAL DIAGNOSES

A. Oropharyngeal dysphagia.
B. Esophageal dysphagia.
C. Odynophagia.
D. Achalasia (decreased peristalsis in the distal esophagus and loss of relaxation of the lower esophageal sphincter while swallowing).

PLAN

A. General interventions:
  1. Identify the cause of dysphagia and treat accordingly.
B. Client teaching:
  1. Educate the client regarding the possible causes of dysphagia.
  2. Stress the importance of nutrition and educate the client and their family about dietary alternatives to maintain nutrition and prevent weight loss.
  3. Discuss potential diagnostic testing necessary to identify the cause of dysphagia.
C. Dietary management:
  1. Depending on the diagnosis and treatment, dietary management—solids versus liquids—will need to be discussed with the client and their family.
  2. A modified food diet should be provided to the client and their family, prepared with a nutritionist and dietitian.
D. Pharmaceutical therapy:
  1. H2 blockers and proton pump inhibitors may be prescribed to help control acid reflux.
  2. If infection of the esophagus is suspected, antibiotics may be considered.
  3. Calcium channel blockers may help with esophageal spasms.

CONSULTATION/REFERRAL

A. Refer to gastroenterology for screening tests required for diagnosis.
B. Refer to a speech or swallowing specialist for assessment and education regarding swallowing exercises. Swallow rehabilitation may be suggested.
C. Refer to a dietitian/nutritionist for alternative food options and education on proper nutrition and prevention of weight loss.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
  1. Dysphagia may occur in conditions such as systemic sclerosis and Sjögren syndrome.
  2. Dysphagia is commonly seen after long-term intubation.
  3. For the older adult population, consider cancer in the differential diagnosis if the client is noted to have anemia and/or weight loss.
  4. Neurologic origin is the most common cause of oropharyngeal dysphagia and can be efficiently managed by the client in collaboration with a clinical swallow specialist.
  5. Screen all geriatric clients for dysphagia because the swallowing reflex is regarded as a normal age-related occurrence. Any difficulties would render further studies as well as initiate education for diet and mechanical exercises to prevent aspiration and other complications.
  6. Recommend safe swallowing techniques to geriatric clients, which could include multiple small swallows, chin-tuck when swallowing, dietary modifications, avoiding rushed/forced eating, sitting upright 90°, and a review of safe administration of medications and practices that are unsafe (such as crushing enteric-coated tablets).
  7. A speech therapist or a clinical swallow specialist would manage any food consistency changes unique to the client's needs and degree of swallowing challenges.

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EPIGLOTTITIS

DEFINITION

A. Epiglottitis is inflammation and swelling of the epiglottis and is a medical emergency.

INCIDENCE

A. Epiglottitis usually occurs in children between ages 2 and 8 years, but may also occur in adults. The incidence of epiglottitis has decreased since the *Haemophilus influenzae* vaccine was introduced.

PATHOGENESIS

A. Epiglottitis is almost always caused by *H. influenzae* (25%), although *Streptococcus pneumoniae* and *S. pyogenes* have also been implicated. Other noninfectious causes include physical trauma, thermal injury to the epiglottitis, and/or ingesting caustic agents.

PREDISPOSING FACTORS

A. Upper respiratory infection.
B. Trauma to the mucosa in the nasopharynx tissue from a viral infection or from injury from food being swallowed.
C. Hypertension.
D. Diabetes mellitus.
E. Substance abuse.
F. Immune deficiency.

COMMON COMPLAINTS

A. Sudden onset of fever.
B. Sudden onset of dysphagia.
C. Sudden onset of drooling.
D. Sudden onset of muffled voice.

OTHER SIGNS AND SYMPTOMS

A. Difficulty with breathing and/or respiratory distress.
B. Stridor.
C. Very ill appearance.
D. Sore throat.
E. Change in voice (muffled voice commonly called a "hot potato voice," as if the client is struggling with a mouthful of hot food).

SUBJECTIVE DATA

A. Determine onset, duration, and course of illness.
B. Has breathing been labored?
C. Is breathing interfering with the client's ability to eat or drink?
D. Has the client had a fever?
E. Has the client had trouble swallowing or talking?

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.
B. Inspect:
1. Observe overall appearance.
2. Check nail beds and lips for cyanosis.
3. Note drooling or difficulty swallowing.
4. Note breathing pattern and rhythm.
5. Note cough if present.
6. Direct exam of the throat can be attempted if there is no concern about compromising the airway. If airway compromise is of concern, do not examine the throat; airway occlusion may result.
C. Auscultate:
1. Auscultate heart and lungs.

DIAGNOSTIC TESTS

A. Lateral neck radiograph confirms diagnosis. However, this test may delay establishment of airway.

DIFFERENTIAL DIAGNOSES

A. Epiglottitis.
B. Foreign-body aspiration.
C. Retropharyngeal abscess.

PLAN

A. General interventions:
1. Immediately transfer to the ED for evaluation and hospitalization.
2. While awaiting transport to hospital, establish patent airway, start oxygen, and assemble airway equipment.
3. Insert intravenous (IV) access for fluids and antibiotic administration.
4. If respiratory arrest occurs, you may not be able to see the airway to intubate. An Ambu bag and mask may work temporarily, but nasogastric (NG) tube insertion may be necessary to prevent gastric distension.
5. Prompt recognition and appropriate treatment usually result in rapid resolution of swelling and inflammation.
B. Client teaching:
1. Educate client and family that epiglottitis is a medical emergency.
2. If client has drooling and no cough, diagnosis is most likely epiglottitis.
C. Pharmaceutical therapy (in-hospital treatment):
1. IV fluids.
2. Antibiotics; IV antibiotics after physician consultation.
3. Blood and epiglottis cultures obtained prior to starting antibiotics.
4. Drug of choice:
a. First-line antibiotic therapy includes use of third-generation cephalosporins, which include ceftriaxone (Rocephin) and ampicillin/sulbactam (Unasyn).
b. If unable to use these due to allergy and so forth, other alternatives include chloramphenicol, cefuroxime (Ceftin), and clindamycin (Cleocin).

FOLLOW-UP

A. Follow-up care occurs in the hospital.
B. An airway specialist should evaluate the client in the operating room.

CONSULTATION/REFERRAL

A. If you suspect epiglottitis, consult with the physician/specialist immediately.
B. The client should be transferred to the ED for treatment and care.
C. Maintain a patent airway while transferring the client to the ED.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. Epiglottitis may induce intractable vomiting. Geriatrics are at high risk of aspiration pneumonia and must be monitored closely.
2. Although rare, poor oral healthcare may cause esophageal inflammation and tongue-base abscess that could cause obstruction. Presentation mirrors epiglottitis: pain, neck swelling, drooling, dyspnea, and respiratory compromise. Promote appropriate mouth care to prevent epiglottitis and infections.

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ORAL CANCER, LEUKOPLAKIA

DEFINITION

A. Oral cancer is cancer of the buccal mucosa, tongue, gingiva, hard palate, soft palate, or lips. White patches, known as leukoplakia, or red, velvety patches, known as erythroplakia, on the buccal mucosa may indicate premalignant lesions.

INCIDENCE

A. Oral cancer is primarily seen in older adults. Approximately 90% of oral cancers are diagnosed as squamous cell carcinoma. The male to female predominance is 2 to 1; oral cancer is higher in African American than Caucasian adults. The death rate is fairly high for oral cancer; it is secondary to the cancer being diagnosed in the late stages of development.

B. There were over 49,000 Americans newly diagnosed with oral cancer in 2017 and 9,000 deaths will occur each year.

C. Oral cancer represents 3% of all newly diagnosed cancers and 2% of all cancer-related deaths.

D. The frequency of oral cancer of cheek and gum rises 50-fold among long-term users of smokeless tobacco.

E. Clients diagnosed with oral cancer are at greater risk of developing cancer in another part of the body, such as the lung, larynx, esophagus, and other sites. Therefore, follow-up exams are recommended for the remainder of the client's life.

PATHOGENESIS

A. Pathogenesis is unknown; 50% of oral cancers already have metastasized at time of diagnosis. The following factors are involved:

1. Use of tobacco in all its forms is highly correlated with risk of oral cancer.

2. Risk of oral cancer is also high with heavy alcohol consumption. Whether this is due to a direct effect of alcohol on the oral mucosa or to associated smoking or vitamin deficiency remains unclear.

3. Chronic iron deficiency leading to Plummer–Vinson syndrome is known to alter mucosal tissues, and this change may be related to increased risk of oral cancer. Research has shown that a diet low in fruits and vegetables contributes to oral cancer.

4. Epstein–Barr virus and papillomavirus have been found in cells of the tongue manifesting oral hairy leukoplakia, a hyperplastic change found in AIDS clients. Human papillomavirus (HPV) is found in approximately 20% to 30% of cases of oral cancer.

5. Occupational hazards also exist from sun exposure. It is estimated that 30% of those with oral cancer worked outdoors.

PREDISPOSING FACTORS

A. Male sex (although leukoplakia is more common in females).

B. Age older than 40 years for males and older than 50 years for females.

C. African American ancestry.

D. Smoking or use of other tobacco products, including such smokeless products as snuff and dip.

E. Alcohol consumption.

F. Sun exposure.

G. Poor diet, deficient in vitamins A, C, and E, and high in salted or smoked meats, fats, and oils.

H. Previous cancer.

COMMON COMPLAINTS

A. Oral sores that do not heal; this is what most commonly leads clients to seek medical care.

B. Poorly fitting dentures.

C. Bleeding mucosa or gingiva without apparent cause.

D. Difficulty swallowing, usually indicating more advanced disease.

E. Altered sensations: burning or numbness, usually indicating more advanced disease.

F. Leukoplakia (50% occur on tongue) or erythroplakia.

OTHER SIGNS AND SYMPTOMS

A. No symptoms, possibly.

B. Decreased appetite related to altered taste.

C. Increased salivation.

D. Sore throat.

E. Foul breath odor.

F. Oral or neck mass.

G. Lymphadenopathy.

SUBJECTIVE DATA

A. Review onset, course, and duration of symptoms. Question the client regarding altered taste, sensations, difficulty swallowing, and foul breath.

B. Evaluate for risk factors. See the "Predisposing Factors" section.

C. Ask the client about history of cancer and treatments.

D. Review the client's use of tobacco products, including age of onset, amount of daily use, and quit dates.

E. Evaluate client's amount of alcohol intake, including age of onset, amount of daily use, and quit dates.

F. Review the client's general health history for other chronic conditions.

G. Review the client's medication history, including prescription and over-the-counter drug use, especially aspirin.

H. Take client's dental history, including previous gum surgery, how long ago dentures were fitted, and if they always fit well.

I. Establish client's usual weight. Is there any weight loss related to altered taste, and if so how much and in what length of time?

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure, and weight.

B. Inspect:

1. Observe general appearance.

2. Note quality of voice patterns.

3. Note odor of breath.

4. Inspect lips, gums, tongue, buccal mucosa for swelling, discoloration, bleeding, asymmetry, texture, limited movement of tongue, abnormal ulcerations, leukoplakia, and erythroplasia. Take out dentures first.

5. Assess for tenderness or pain in mouth/tongue.

a. Leukoplakia ranges from slightly raised, white, translucent areas to dense, white, opaque plaques, ▶

with or without adjacent ulceration. Normal intraoral mucosa is pinkish or salmon-colored.

b. Mucosal erythroplasia is red, inflammatory, or erythroplastic mucosal changes. It appears smooth, granular, and minimally elevated, with or without leukoplakia, and persists more than 14 days.

c. Erythroplakia may mimic inflammatory lesions, but can be differentiated by failure of the affected area to blanch with light pressure. **Erythroplakia is a malignant change seen as a red, velvety, plaque-like lesion on mucous membrane.**

d. Other oral lesions appear black, blue, or brown.

e. Inspect the tongue, oropharynx (soft palate, lingual aspect of the retromolar trigone, anterior tonsillar pillar), and floor of the mouth.

f. Cancer of the lip is a lesion that fails to heal.

g. Signs and symptoms of cancer of the tongue are swelling, ulceration, areas of tenderness or bleeding, abnormal texture, and limited movement.

C. Palpate:

1. Palpate mouth for masses. Try to remove or scrape patches.

2. Palpate lymph nodes: cervical (anterior/posterior chain), submandibular, sublingual, and submental, pre-/postauricular; check nodes for size, firmness, and tenderness.

D. Auscultate:

1. Auscultate lungs and heart: The lungs are the most frequently involved extranodal metastatic site.

DIAGNOSTIC TESTS

A. Check serum blood for HIV, if indicated.

B. Stain the oral lesion with toluidine blue: Lesion stains dark blue after rinsing with acetic acid. Normal tissue does not absorb the stain.

C. Perform biopsy for persistent lesions (>2 weeks): It is essential to differentiate from blue-black lesion of malignant melanoma.

D. Perform chest radiography to rule out metastasis.

E. Consider CT, MRI, or bone scan to rule out metastasis.

DIFFERENTIAL DIAGNOSES

A. Oral leukoplakia.
B. Actinic keratosis.
C. Periapical abscess.
D. Gingivitis.
E. Periodontitis.
F. Lichen planus.
G. Oral candidiasis.
H. Discoid lupus.
I. Pemphigus vulgaris.

PLAN

A. General interventions:

1. If oral cancer is suspected, refer to a physician or otolaryngologist/dentist for evaluation.

2. Suspicious lesions should have biopsy.

3. Leukoplakia ranges from slightly raised, white, translucent areas to dense, white, opaque plaques, with or without adjacent ulceration. Normal intraoral mucosa is pinkish or salmon-colored.

4. Mucosal erythroplasia is red, inflammatory, or erythroplastic mucosal changes. It appears smooth, granular, and minimally elevated, with or without leukoplakia, and persists more than 14 days.

5. Erythroplakia may mimic inflammatory lesions, but can be differentiated by failure of the affected area to blanch with light pressure.

6. Cancer of the lip is a lesion that fails to heal.

B. Client teaching:

1. Advise the client to stop smoking and stop using oral tobacco products.

2. Advise the client to reduce/eliminate alcohol consumption.

3. Encourage routine dental care and exams.

4. Review dietary intake and educate the client regarding benefits of increasing dietary intake of vitamins A, C, and E. Encourage client to reduce dietary intake of foods that are high in salt, smoked meats, fats, and oils.

5. Recommend wearing sunscreen/lip balm with sun protection factor (SPF) of 15 or greater.

C. Pharmaceutical therapy:

1. Erythroplakia does not respond to antifungal therapy.

2. For mouth pain, topical anesthetic or Magic mouthwash (common compounded solution includes Maalox/diphenhydramine/lidocaine combined at a 1:1:1 ratio).

FOLLOW-UP

A. If immediate biopsy is not indicated, ask the client to return for reevaluation in 2 weeks, after eliminating irritants and noxious agents.

CONSULTATION/REFERRAL

A. Refer the client to an otolaryngologist and/or dentist for immediate biopsy for deeply ulcerative or fungating lesions. Follow-up treatment may include one or more of the following: wide excision, radical neck dissection, radiation, and chemotherapy.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. The American Cancer Society recommends that people between age 20 and 40 undergo an oral cancer screening every 3 years and that those older than 40 years be screened every year. Oral screening should be considered annually in adults who use tobacco and/or alcohol.

B. Geriatrics:

1. Oral cavity cancer within the geriatric population predominantly arises between 50 and 70 years old secondary to susceptibility to mucosal disorders. Natural aging process decreases salivary secretion and risks ulceration and mucosa breakdown, which eventually lead to immune system dysfunction. This increases risk of a potential malignant disorder.

2. Evidence-based research indicated that mortality was reduced when clients over 65 years old with leukoplakia were screened and diagnosed with oral cavity cancer at an early stage.

3. A chronically dry mouth (xerostomia) affects quality of life, reduces adequate ingestion of foods, causes burning sensations and taste disturbances, and increases risk of malnutrition.

4. Thoroughly evaluate any oral pain with the geriatric population to rule out oral cancer. Normal age-related changes include yellowing/darkening of teeth, thickening composition/abrasion, gingival recession leading to root caries, and reduced sensitivity secondary to the number ▶

of blood vessels entering the tooth, which decreases the enamel.

5. Precancerous lesions might be subtle and asymptomatic. Meticulously examine lateral borders of tongue, lips, and floor of mouth, where oral cancer most commonly occurs. A lesion may be white or dull red and will progress to ulceration.

RESOURCES

American Academy of Family Physicians: www.aafp.org
American Cancer Society: www.cancer.org
National Cancer Institute: www.cancer.gov
Oral Cancer Foundation: www.oralcancerfoundation.org

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PHARYNGITIS

DEFINITION

A. Pharyngitis is inflammation of the pharynx and surrounding lymph tissue.

INCIDENCE

A. Pharyngitis is the fourth most common condition seen in medical practice. Group A *Streptococcus* accounts for approximately 5% to 15% of adults who present for evaluation and treatment of pharyngitis. Viral pharyngitis accounts for approximately 40% to 60% of all cases of sore throat.

PATHOGENESIS

A. Pharyngitis may be due to viral, bacterial, and fungal agents, as well as other atypical agents:

1. Approximately 50% to 80% of cases of sore throat are viral. Viral agents include rhinoviruses/adenoviruses, coxsackievirus, enteric cytopathic human orphan (ECHO) viruses, and Epstein–Barr virus.

2. Approximately 3% to 36% of cases of sore throat are bacterial. Bacterial agents include group A beta-hemolytic streptococcus (GAS), *Neisseria gonorrhoeae*, and *Corynebacterium diphtheriae*.

3. The fungal source is *Candida albicans*.

4. Atypical agents include *Mycoplasma pneumoniae* and *Chlamydia trachomatis* (rare).

5. Noninfectious causes include allergic rhinitis, postnasal drip, mouth-breathing, mechanical/caustic agents, and trauma.

PREDISPOSING FACTORS

A. Cigarette smoking.
B. Allergies.
C. Upper respiratory infections.
D. Oral sex.
E. Drugs (antibiotics and immunosuppressants).
F. Debilitating illnesses (such as cancer) that can cause *C. albicans* to proliferate.
G. Recent exposure to a person testing positive for GAS.

COMMON COMPLAINTS

A. Sore and/or scratchy throat.
B. Fever.
C. Headache.
D. Malaise.
E. Tonsillar exudate.
F. Anterior cervical lymph node swelling.
G. Skin rash.
H. Bacterial pharyngitis symptoms include acute onset of sore throat, anterior cervical lymphadenopathy/tenderness, tonsillar exudate, skin rash, fever.

OTHER SIGNS AND SYMPTOMS

A. Oral vesicles.
B. Fatigue.
C. Dysphasia, hoarseness.
D. Abdominal pain.
E. Vomiting.
F. Viral pharyngitis symptoms include cough, fever, malaise, nasal congestion, oral ulcers, coryza, conjunctivitis.

POTENTIAL COMPLICATIONS

A. Without proper antimicrobial treatment, streptococcal pharyngitis can lead to serious complications such as the following:

1. Suppurative adenitis with tender, enlarged lymph nodes.
2. Scarlet fever.
3. Peritonsillar abscess.
4. Glomerulonephritis.
5. Rheumatic fever.

SUBJECTIVE DATA

A. Ask the client about onset, course, and duration of symptoms. Ask about dyspnea or dysphagia.
B. Inquire about mouth lesions, rhinorrhea, cough, drooling, and fever.
C. Ask about malaise, headache, fatigue, and fever; these are symptoms of mononucleosis.
D. Take a sexual history, if indicated. Ask if family members or sexual partners have the same signs and symptoms. Pharyngeal gonorrhea has no symptoms, so high-risk clients should be tested.
E. Ask whether symptoms have caused decreased intake of food and fluid.
F. Determine history of heart disease, strep pharyngitis, rheumatic fever, and other respiratory diseases such as asthma, emphysema, and chronic allergies.
G. If rash is present, find out when it first occurred and if it has spread.
H. Ask about signs and symptoms of urinary tract infection and pyelonephritis.
I. Ask about a history of herpes, immunosuppressive disorders, and steroid use.
J. Review immunization history.

PHYSICAL EXAMINATION

A. Check temperature and blood pressure, if indicated.
B. Inspect:

1. Observe general appearance.
2. Examine the mouth, pharynx, tonsils, and hard and soft palate for vesicles and ulcers, candidal patches, erythema, ▶

hypertrophy, exudate, and stomatitis. Check the gum and palate for petechiae and tongue for color and inflammation.

3. Examine the ears, nose, and throat. Assess patency of airway if tonsils are enlarged.

4. Inspect skin for rashes:

a. Pastia lines are petechiae present in a linear pattern along major skin folds in axillae and antecubital fossa that are seen with GAS.

b. Erythema marginatum, caused by GAS, is an evanescent, nonpruritic, pink rash mainly on the trunk and extremities. It may be brought out by heat application.

C. Auscultate:

1. Auscultate the heart and lungs.

D. Percuss:

1. Percuss abdomen, especially spleen area.

2. Percuss the chest.

E. Palpate:

1. Palpate lymph nodes, especially of the anterior and posterior cervical chains, axilla, and groin.

2. Palpate abdomen for organomegaly and suprapubic tenderness.

3. Palpate back for costovertebral angle (CVA) tenderness.

F. Perform neurologic exam:

1. Check for nuchal rigidity and meningeal irritation.

G. Use the Modified Centor Clinical Prediction Rule for group A streptococcal infection (Table 11.1).

TABLE 11.1 MODIFIED CENTOR CLINICAL PREDICTION RULE

Points	Criteria
+1	Tonsillar exudates present
+1	Tender anterior chain cervical adenopathy
+1	Absence of a cough
+1	Fever by history
+1	3–14 years of age
0	15–44 years of age
–1	≥45 years of age
Total score	

Once scored: If 4 points, + predictive value of 80%, treat empirically. If 2 to 3 points, positive predictive value of 50%, complete rapid strep antigen, treat as group A beta-hemolytic *Streptococcus* positive. If 0 or 1 point, positive predictive value of 20%, do not test, treat empirically only, with follow-up as needed.

DIAGNOSTIC TESTS

A. Rapid strep test; if negative, perform throat culture and sensitivity.

1. Throat culture is recommended if the client has been exposed to an area at high risk of GAS, has history of acute renal failure (ARF), immunosuppression-compromised, or has close contact with clients with ARF or immunosuppression. **Throat culture and sensitivity is the gold standard for diagnosis**.

B. Monospot test.

C. Complete blood count (CBC) with differential.

D. COVID-19 screening recommended if illness appears viral.

E. HIV and gonorrhea culture recommended for sexually transmitted infection concerns.

F. Blood cultures if sepsis is suspected.

G. Radiograph of neck if possible trauma.

DIFFERENTIAL DIAGNOSES

A. Pharyngitis.

B. COVID-19.

C. Stomatitis.

D. Rhinitis.

E. Sinusitis with postnasal drip.

F. Epiglottis.

G. Peritonsillar abscess.

H. Mononucleosis.

I. Herpes simplex.

J. Coxsackie A virus.

K. *C. diphtheriae.*

L. Trench mouth.

M. Vincent angina.

N. *C. albicans.*

O. HIV.

PLAN

A. General interventions:

1. Clients with a history of rheumatic fever and those who have a household member with a documented group A streptococcal infection need immediate treatment without prior testing.

2. Herpangina is characterized by small oral vesicles on the fauces and soft palate caused by the coxsackievirus.

3. Herpes causes vesicles and small ulcers (stomatitis) of the buccal mucosa, tongue, and pharynx.

4. Trench mouth (gingivitis) and necrotic tonsillar ulcers (Vincent angina) cause foul breath, pain, pharyngeal exudate, and a gray membranous inflammation that bleeds easily.

5. *C. albicans* (thrush) may be painful and causes cheesy, white exudate on the tongue and inside cheeks.

6. Oral candidiasis may be the first symptom of HIV.

7. Peritonsillar cellulitis causes inflamed, edematous tonsils, grayish-white exudate, high fever, rigors, and leukocytosis. Peritonsillar abscess (palpable mass) may also develop.

8. Mononucleosis causes tonsillar exudates in 50% of clients; 33% develop petechiae at junction of hard and soft palate.

9. *C. diphtheriae* causes a whitish-blue pharyngeal exudate "pseudomembrane" that covers the pharynx and bleeds if removal is attempted.

10. COVID-19 symptoms include fever, cough, difficulty breathing, congestion, sore throat for a few days, and loss of taste/smell. Self-isolation recommended per Centers for Disease Control and Prevention guidelines.

11. Do not put instruments in the airway if you suspect epiglottitis.

B. Client teaching: *See Client Teaching Guide for this chapter, "Pharyngitis."*

1. Recommend rest, fluids, and staying well-hydrated.

2. Avoid contact with others for 24 hours after starting antibiotics.

3. Treat symptomatically: ibuprofen/acetaminophen as needed for body aches/sore throat/fever. Warm saltwater gargles/throat lozenges for sore throat. Sterilize toothbrush or buy new toothbrush.

4. Take antibiotics/medication as prescribed. ▶

5. Call for fever not responding to medication/treatment, worsening symptoms, or not improving in the next 3 to 4 days.

6. Call 911 for difficulty with breathing, throat/neck swelling, drooling, or other severe symptoms.

7. If concerns of mononucleosis, advise no contact sports until diagnosis is made to prevent injury to the spleen.

C. Pharmaceutical therapy:

1. Drug of choice: Prescribe one of the following penicillins for bacterial pharyngitis:

a. Penicillin V potassium (Pen-Vee-K): 500 mg BID for 10 days.

b. Amoxicillin: 500 or 875 mg BID for 10 days.

c. Penicillin G benzathine: 1.2 million units intramuscular (IM) injection, one dose.

d. Cephalexin: 500 mg BID for 10 days.

2. If the client is allergic to penicillin:

a. Clindamycin: 600 mg TID for 10 days.

b. Azithromycin: 500 mg on day 1 followed by 250 mg daily on days 2 to 5.

c. Clarithromycin: 250 mg BID for 10 days.

3. Recurrent bacterial pharyngitis:

a. Clindamycin: 600 mg TID for 10 days.

b. Penicillin and rifampin: penicillin V 500 mg BID for 10 days.

c. Amoxicillin-clavulanic acid: 500 mg Q8H or Q12H for 10 days; or 875 mg Q12H for 10 days; not for use in creatinine clearance less than 30 mL/min.

d. Penicillin G benzathine: 1.2 million units IM injection, plus one dose of rifampin 600 mg/d orally for 4 days.

4. For severe symptoms:

a. Single-dose corticosteroids, dexamethasone, may be prescribed; however literature/evidence of use is limited.

5. For pharyngeal gonorrhea:

a. Ceftriaxone (Rocephin) 500 mg to 1 g by IM injection (provider should prescribe to cover for *C. trachomatis* as well).

6. For *M. pneumoniae* and *C. trachomatis*:

a. Erythromycin (E-Mycin) 250 mg orally three to four times daily for 10 days or azithromycin 1 g orally single dose or doxycycline 100 mg orally BID for 7 days.

7. For pharyngeal candidiasis in the immunocompromised client:

a. Oral nystatin suspension (100,000 U/mL): 15 mL by swish-and-swallow method QID.

b. Clotrimazole troche: 10 mg held in the mouth for 15 to 30 minutes three to five times daily for 7 to 10 days.

FOLLOW-UP

A. If symptoms do not improve in 3 to 4 days, advise client to call/return for evaluation.

B. Treat sexual partners of those diagnosed with pharyngeal gonorrhea.

CONSULTATION/REFERRAL

A. Consult a physician/specialist if client has severe dysphagia or dyspnea, signaling possible airway obstruction.

B. Refer the client to an otolaryngologist if peritonsillar abscess is noted.

INDIVIDUAL CONSIDERATIONS

A. Recommendations include treating clients with oral antibiotics for 10 days to eradicate infection. For clients who are not able to tolerate oral antibiotics, penicillin G benzathine may be given via IM route.

B. Geriatrics:

1. Acetaminophen for sore throat is treatment of choice for the geriatric population.

2. Antibiotic treatment for pharyngitis is warranted if client is ≥65 years old with an acute cough and has two or more of the following criteria: hospitalized in the past year, diabetic, congestive heart failure, and on glucocorticoid therapy. If the client is ≥80 years old with pharyngitis and cough, treat with antibiotic if one or more of the latter criteria applies.

3. Older adult clients with chronic illnesses (such as diabetes, congestive heart failure, and skin diseases) who have pharyngitis caused by GAS are at an increased risk of bacteremia and more likely to die from complications.

4. Viral pharyngitis with symptoms of runny nose, stuffy chest, congestion, cough, and red/itchy eyes should be treated symptomatically and avoid antibiotics.

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STOMATITIS, RECURRENT APHTHOUS STOMATITIS

DEFINITION

A. Stomatitis is tender, round, discrete, oval, shallow, 1- to 5-mm ulcers in the oral cavity. The ulcers are gray-white or yellow, on nonkeratinized skin, and surrounded by erythematous halos. They typically involve the labial and buccal mucosa and tongue, and adjacent tissue appears healthy.

B. Major recurrent aphthous stomatitis (RAS) has larger, deeper ulcers, lasts a longer period of time, usually recurs up to four times a year, and frequently leaves scars. It can cause significant dysphagia.

INCIDENCE

A. Stomatitis affects 20% to 50% of the population. It is very common in North America.

PATHOGENESIS

A. Cause is poorly understood. Genetic, immunologic, viral, or nutritional causes are possible.

PREDISPOSING FACTORS

A. Minor trauma.

B. History of RAS.

C. Possible nutritional deficiency in iron, vitamin B_{12}, folic acid, or zinc.

D. Hormonal changes.

COMMON COMPLAINT

A. Painful sore in mouth.

OTHER SIGNS AND SYMPTOMS

A. Burning sensation in mouth for 24 to 48 hours before lesions appear. ▶

SUBJECTIVE DATA

A. Elicit history of aphthous stomatitis.
B. Ask the client about prodrome of burning or stinging in the mouth.
C. Elicit information regarding previous illness and trauma.
D. Inquire regarding nutritional intake and dietary (balanced meals) and oral supplements (B_{12}, folic acid, and zinc).

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.
B. Inspect:
  1. Inspect mouth for ulcers.
  2. Inspect ears, nose, and throat.
  3. Inspect skin, especially palms and soles, for lesions; indicates hand-foot-and-mouth disease.
C. Auscultate:
  1. Auscultate the heart and lungs.

DIAGNOSTIC TESTS

A. There is no specific diagnostic testing for stomatitis.
B. Consider herpes simplex virus (HSV) culture if HSV is considered for diagnosis.
C. If syphilis is of concern, order serum rapid plasma reagin (RPR).

DIFFERENTIAL DIAGNOSES

A. Aphthous stomatitis.
B. Herpetic stomatitis.
C. Behçet disease.
D. Crohn disease.
E. HIV.
F. Kawasaki syndrome.
G. Hand-foot-and-mouth disease.

PLAN

A. General interventions:
  1. Avoid spicy, salty, or hot foods.
  2. Encourage cold foods, such as fluids, ice pops, and so forth to help with pain.
  3. Avoid hard, sharp food that is difficult to chew.
  4. Recommend using a soft-bristle toothbrush when brushing teeth. Sodium lauryl sulfate found in toothpaste may delay healing time.
  5. Consider monitoring clients with RAS for systemic / autoimmune disorders such as Behçet disease, Grave disease, Hashimoto thyroiditis, systemic lupus erythematosus (SLE), gout, rheumatoid arthritis, and ankylosing spondylitis.
B. Client teaching: *See Client Teaching Guide for this chapter, "Aphthous Stomatitis."*
C. Pharmaceutical therapy:
  1. Mouthwash made of diphenhydramine (Benadryl), with Kaopectate, or Maalox or sucralfate, and viscous lidocaine three to four times a day. Tell the client not to swallow medication.
  2. Sucralfate (Carafate) suspension one teaspoon QID may be used to swish in mouth and spit out for oral comfort.
  3. Topical corticosteroids, such as dexamethasone, triamcinolone, fluocinonide, or clobetasol, are highly recommended. For severe cases, systemic corticosteroids may be needed, for example, glucocorticoid gel such as fluocinonide gel (Lidex) 0.05% two to four times a day, one of which is always at bedtime.
  4. Orabase with or without triamcinolone acetonide (Kenalog) applied to ulcer two to four times a day until healed.
  5. Immunomodulatory agents, antimicrobials, and anesthetic agents may also be used.

FOLLOW-UP

A. Follow-up as needed for treatment of recurrences.

CONSULTATION/REFERRAL

A. Refer the client to, or consult with, a physician/specialist if ulcers are deeper or larger than 1 to 5 mm, if Kawasaki disease is suspected, or if no improvement is seen with adequate treatment.
B. Any lesion lasting longer than 3 weeks should be evaluated by a dentist or oral surgeon to rule out cancer.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
  1. Avoid use of fluocinonide and triamcinolone acetonide (Kenalog) in pregnant or nursing clients.
B. Adults:
  1. Consider underlying immunologic conditions in clients who have recurrent oral ulcers.
C. Geriatrics:
  1. Ask older adult clients about their dentures, if there is adequate fit (looseness or too tight), and if there is any pain.
  2. Dentures should be removed and cleaned at least once daily. Encourage clients to remove dentures at night.
  3. Oral inflammation and denture-related stomatitis have recently been linked through evidence-based research with the pathogenesis of cardiovascular disease (atherosclerosis and hypertension). Geriatric clients must be screened for denture-related stomatitis and immediate antifungal treatment is advised.
  4. Nutritional challenges in the older adult population can lead to stomatitis and aphthous ulcers. Assess chewing ability. Evaluate geriatric clients for iron, B_{12}, and folic acid deficiencies.

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THRUSH

DEFINITION

A. Thrush is a fungal infection of the oral cavity and/or the pharynx caused by *Candida*.

INCIDENCE

A. Approximately 9% to 31% of clients with AIDS and 20% of clients diagnosed with cancer will have thrush.

B. Healthy adults may also experience an episode of thrush; however, these adults usually have other risk factors, such as the frequent use of inhaled steroids and/or use of oral antibiotics/steroids.

PATHOGENESIS

A. Thrush is an overgrowth of yeast cells, *Candida albicans*, on the oral mucosa, which leads to desquamation of the epithelial cells, creating a pseudomembrane over the normal oral mucosa. Other species of yeast cells may include *C. glabrata*, *C. krusei*, and *C. tropicalis*.

PREDISPOSING FACTORS

A. Use of broad-spectrum antibiotics.
B. HIV.
C. Prolonged steroid use (systemic or inhaled corticosteroids).
D. Cancer treatments (radiation/chemotherapy).
E. Dentures.
F. Malnutrition.

COMMON COMPLAINTS

A. Soreness and pain in the mouth.
B. White plaque coating the buccal mucosa, palate, tongue, or oropharynx.

OTHER SIGNS AND SYMPTOMS

A. Erythemic, sore tongue, buccal mucosa, or oropharynx.

SUBJECTIVE DATA

A. Determine onset, duration, and course of illness.
B. Has the client used antibiotics or other medications in the previous weeks?
C. Does the client use inhaled or systemic steroid on a daily basis?
D. Has the client had difficulty swallowing?
E. Does the client wear dentures?

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.
B. Inspect:
1. Inspect oral cavity for white, curd-like plaques that cannot be removed.
2. Inspect ears, nose, and throat.

DIAGNOSTIC TESTS

A. Gram stain or potassium hydroxide (KOH) performed on scraping specimen of lesions. Diagnosis is confirmed with the presence of yeast and pseudohyphae. Gram stain or KOH testing is recommended for recurrent infections.
B. If treatment prescribed is not working, fungal culture should be sent for diagnosis.

DIFFERENTIAL DIAGNOSES

A. Thrush.
B. Esophageal candidiasis (hallmark symptom is odynophagia or pain with swallowing).
C. Stomatitis.
D. Aphthous ulcer.
E. Hairy leukoplakia.

PLAN

A. General interventions:
1. Instruct the client/family on proper use and cleaning/rinsing of inhalers/dentures to prevent reoccurrence of thrush.
2. Instruct client on the use of oral medications to treat thrush.
B. Client teaching: *See Client Teaching Guide for this chapter, "Thrush."*
C. Pharmaceutical therapy:
1. Oral candidiasis: nystatin (Mycostatin) oral suspension (400,000–600,000 units) 4 to 6 mL QID for 7 to 10 days. Place medication in front of the mouth on each side. Rub directly on plaques with a cotton swab. Nystatin pastilles: 200,000-unit lozenge QID for 7 to 14 days, or swish and swallow 500,000-unit QID for 14 days or two 500,000-unit tablets TID for 14 days.
2. Clotrimazole troche (Mycelex): 10 mg five times daily for 14 days; monitor for side effects.
3. Fluconazole: for adults, 400 mg once (load dose), then 200 mg daily for 14 days.
4. Mary's magic mouthwash (diphenhydramine, nystatin, Maalox—equal amounts of each) may be used for maintenance once thrush has resolved. Swish and spit this mouthwash one to three times a day as needed. If painful, viscous lidocaine can also be added.

FOLLOW-UP

A. Advise the client to telephone the office if there is no improvement, if thrush lasts more than 10 days, or if there is unexplained fever.

CONSULTATION/REFERRAL

A. Consult a physician/specialist if thrush does not resolve with adequate antifungal treatment.
B. Immunosuppressed clients are at higher risk of developing systemic dissemination and candidal esophagitis. Consult a physician if this is suspected.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. Ask older adult clients about their dentures, if there is adequate fit (looseness or too tight), and if there is any pain.
2. Dentures should be removed and cleaned at least once daily. Encourage clients to remove dentures at night.
3. Oral inflammation and denture-related stomatitis have recently been linked through evidence-based research with the pathogenesis of cardiovascular disease (atherosclerosis and hypertension). Geriatric clients must be screened for denture-related stomatitis and immediate antifungal treatment is advised.
4. Nutritional challenges in the older adult population can lead to stomatitis and aphthous ulcers. Assess chewing ability. Evaluate geriatric clients for iron, vitamin B_{12}, and folic acid deficiencies.
5. Certain conditions may cause overgrowth of *Candida* thrush and/or increase occurrences of aphthous stomatitis in the geriatric population, which could include use of steroid inhaler, tobacco use, systemic steroid/antibiotics, ▶

radiation/chemotherapy, food allergies, asthma, diabetes or other endocrine disorders, anxiety, and lack of sleep.

6. If pain is involved, evaluate client's food/drink intake, weight, and nutritional status.

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CLIENT TEACHING GUIDE

APHTHOUS STOMATITIS

PROBLEM Aphthous stomatitis refers to tender ulcers in the mouth that recur.

CAUSE The cause is unknown, but possible causes include diet (lack of iron, zinc, or B vitamins), menstrual or hormonal changes, and viruses.

TREATMENT PLAN

A. Use an over-the-counter gel, such as Anbesol or Orajel, four times daily.
B. You may be prescribed a mouthwash made of diphenhydramine (Benadryl), Maalox, and lidocaine or fluocinonide gel to "swish" in your mouth two to four times daily.

Activity: No restrictions are required.

Diet:
A. Avoiding spicy, salty, or hot foods may help.
B. Using a straw when drinking may reduce pain.
C. Cold foods may be easier to tolerate.
D. Avoid hard or sharp food.
E. Use a soft toothbrush.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. Worse symptoms than seen at the office visit today.
B. Ulcers that do not heal in approximately 1 to 2 weeks.
C. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

PHARYNGITIS

PROBLEM Pharyngitis (sore throat) is a condition that occurs when your throat becomes inflamed.

CAUSE The inflammation can be caused by a virus, a bacterium, or a fungus. Other noninfectious causes include postnasal drip, allergies, mouth-breathing, and trauma.

PREVENTION

A. Avoid sick people and crowds. Stay at home if you are sick.
B. Cover your mouth when coughing.
C. Do not share a drinking glass, kiss, or have close contact with anyone who has an upper respiratory infection.
D. Gargle with warm saltwater frequently. Use over-the-counter pain medications (such as acetaminophen or ibuprofen) for pain relief.

TREATMENT PLAN

A. Hot tea, soup, and throat lozenges soothe your throat.
B. Use disposable tissues when sneezing and when blowing your nose. If no tissue is available, do the "elbow sneeze" into the bend of your arm (away from your open hands). Dispose of the tissues and then wash your hands.
C. Avoid smoking and secondhand smoke.

Activity: If you have strep throat, do not return to school or work until you have completed a full 24 hours of antibiotic. Rest or nap as often as possible while you are sick. If diagnosed with strep throat, please change your toothbrush to prevent reinfection after treatment.

Diet: Eat a healthy diet. If swallowing is difficult, eat soft foods such as ice cream, Jell-O, pudding, and soup. Avoid salt and spicy foods. Increase your fluid intake to 10 to 12 glasses a day.

Medications: You will be prescribed antibiotics if you have a bacterial infection. If your sore throat is caused by a virus, antibiotics would not help. Do not share your prescription medications with other family members who are also sick. They need a full prescription, too. Many other medications are available over the counter, such as throat lozenges and cough suppressants.

You Have Been Prescribed: _____

You Need to Take: _____

If you are prescribed antibiotics, complete all of the doses.

You Need to Notify the Office If:
A. You have difficulty breathing because of the sore throat or enlarged tonsils.
B. Your symptoms are worse after 24 hours of antibiotics.
C. You are unable to keep down your antibiotic because of vomiting.
D. You develop a rash or itching after starting the antibiotic.
E. You are a diabetic and your blood glucose is high, or you have ketones in your urine.
F. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

THRUSH

PROBLEM Oral thrush is characterized by white patches that coat the inside of the mouth and tongue. It mainly affects adults with risk factors such as a compromised immune system, use of frequent antibiotics, and/or chronic use of steroid inhalers.

CAUSE Thrush is caused by a yeast called *Candida*, which grows rapidly on the lining of the mouth and tongue. It may also occur after a course of antibiotic medication.

PREVENTION

A. Avoid overuse of antibiotics.
B. Clean inhalers and dentures daily. Use a denture cleaner daily. You may soak the dentures overnight in the denture cleaner. Rinse dentures well before putting them into your mouth. Rinse your mouth after inhaled steroid use.
C. Practice good oral hygiene daily.
D. Change your toothbrush or sanitize the toothbrush when diagnosed with thrush to prevent reinfecting the mouth with yeast.
E. Rinse your mouth after using steroid inhaler.

TREATMENT PLAN

A. Try to remove any large plaques with a moistened cotton-tipped applicator or gauze pad.
B. Cleanse the mouth before giving medication. The use of salt or warm saltwater (with iodized salt or baking soda) helps relieve irritation, as well as creates a poor environment for fungal growth.
C. Place the medication in the front of the mouth on each side.
D. Rub it directly on the plaques with a cotton swab.

Diet: As tolerated. Limit foods high in sugar and yeast products such as beer, wine, and bread. Increase diet to include yogurt and products that contain *acidophilus*.

Medications: Nystatin is an oral medication used to treat thrush. Nystatin suspension four times a day after meals or 30 minutes prior to eating. Patches should improve within 2 to 3 days of using the medication.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. You have difficulty with eating or drinking because of pain.
B. Symptoms do not improve or thrush lasts longer than 10 days.
C. Unexplained fever occurs.
D. Secondary infection occurs in the mouth (pain, tenderness, sores).
E. Other: _____

Phone: _____

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CHAPTER 12

RESPIRATORY GUIDELINES

Audra Malone Cave

ASTHMA

DEFINITION

A. Asthma is a chronic airway inflammation characterized by recurrent episodes of wheezing, breathlessness, chest tightness, and cough, particularly at night and in the early morning. Episodes are associated with widespread, variable, often reversible airflow obstruction and bronchial hyperresponsiveness when airways are exposed to various stimuli or triggers. Asthma is responsible for lost school days, lost productivity, and presenteeism.

B. Classifications of asthma:

1. Mild intermittent:
- a. Symptoms of cough, wheezing, chest tightness, or difficulty breathing less than or equal to two per week.
- b. Flare-ups are brief, but intensity may vary.
- c. Nighttime symptoms less than or equal to two per month.
- d. No symptoms between flare-ups.
- e. Lung function test: forced expiratory volume in 1 second (FEV_1) is 80% or more above normal values.
- f. Peak flow has less than 20% variability from morning to next morning, or from morning to night, day-to-day.

2. Mild persistent:
- a. Symptoms of cough, wheezing, chest tightness, or difficulty breathing three to six times a week.
- b. Flare-ups may affect activity level.
- c. Nighttime symptoms three to four times a month.
- d. Lung function test FEV_1 is above 60% but below 80% of normal values.
- e. Peak flow has more than 30% variability.

3. Moderate persistent:
- a. Symptoms of cough, wheezing, chest tightness, or difficulty breathing daily.
- b. Flare-ups may affect activity level.
- c. Nighttime symptoms five or more times a month.
- d. Lung function test FEV_1 is above 60% but below 80% of normal values.
- e. Peak flow has more than 30% variability.

4. Severe persistent:
- a. Symptoms of cough, wheezing, chest tightness, or difficulty breathing are continual.
- b. Frequent nighttime symptoms.
- c. Lung function test FEV_1 is 60% or less of normal values.
- d. Peak flow has more than 30% variability.

INCIDENCE

A. Asthma affects 25 million people in the United States.

B. Asthma rates in the general population are increasing, including in developed countries.

C. Up to 80% of those with asthma also suffer from persistent rhinitis.

D. Asthma is often associated with other comorbid conditions, including gastroesophageal reflux disease (GERD) and obesity.

E. The diagnosis is more common in adult females than males.

F. Puerto Ricans, African Americans, Filipinos, and Irish Americans have higher rates than other ethnicities.

G. Asthma control and asthma outcomes are worse for lower socioeconomic groups.

PATHOGENESIS

A. Asthma arises from a complex cycle of processes initiated by airway inflammation resulting from physical, chemical, and pharmacologic agents (e.g., environmental irritants, allergens, furry animals, cockroaches, dust mites, pollens and mold, cold air, viral respiratory infections, and exercise). It progresses to airway hyperresponsiveness, bronchoconstriction, airway wall edema, chronic mucus plug formation, and chronic airway remodeling.

PREDISPOSING FACTORS

A. Family history.

B. Coexisting sinusitis, nasal polyps, and sensitivity to aspirin or other nonsteroidal anti-inflammatory drugs (NSAIDs).

C. Exposure to wood dust, metals, and animal products in the workplace.

D. Premenstrual asthma (PMA).

E. Smoking.

F. Early menarche.

G. Obesity.

H. Postmenopausal hormone replacement therapy.

I. Viral respiratory infections.

J. Gastroesophageal reflux.

K. Triggers:

1. Exposure to allergens.

2. Upper airway viral infection.

3. Medications: Beta-blockers, angiotensin-converting enzyme (ACE) inhibitors, cyclooxygenase (COX) inhibitors, aspirin, and NSAIDS may induce symptoms.

4. Exercise.

5. Situational factors: cold air, laughter, strong odors, air pollution, smoke exposure, pregnancy.

6. Foods.

The contributions of Melissa A. Hall to this chapter in prior editions are acknowledged here.

7. Hormones.
8. GERD.

COMMON COMPLAINTS

A. Recurrent cough (worse at night and early morning).
B. Recurrent wheezing.
C. Recurrent shortness of breath (SOB) or dyspnea.
D. Recurrent chest tightness (may worsen with moderate activity).

OTHER SIGNS AND SYMPTOMS

A. Nocturnal awakening from symptoms.
B. Variation of symptoms with seasons or environment.
C. Chest discomfort, tightness with moderate activity.

SUBJECTIVE DATA

A. Ask about onset, duration, pattern, and course of symptoms.
B. Inquire about sudden severe episodes of coughing, wheezing, and SOB and whether precipitating factors can be identified.
C. Ask whether the client has chest colds that take more than 10 days to resolve.
D. Ask if the client is a smoker, how much, for how long, and if interested in quitting.
E. Ask whether symptoms seem to occur during certain seasons or during exposure to the following environmental irritants:
1. Tobacco smoke.
2. Perfume.
3. Household pets.
4. Fireplaces.
5. Wood-burning stoves.
6. Mold.
7. Dust mites.
8. Cockroaches.
9. Occupational triggers.

F. Find out how often coughing, wheezing, or SOB awakens the client.
G. Ask if symptoms are caused or exacerbated by moderate exercise or physical activity.
H. Determine family history of asthma, allergies, and eczema.
I. Determine if the client is pregnant or has medical problems. In pregnancy, treatment is based on the same step therapy as in nonpregnant client.
J. Administer the asthma control test. The test is available online at www.asthma.com. A score greater than 20 points indicates the client's asthma is well-controlled. A scores of 16 to 19 points indicates the client's asthma is not well-controlled.
K. Evaluate if the client has ever been tested for allergies.
L. Ask how many times the client has gone to the ED or had to be hospitalized for an asthmatic attack.
M. Ask how many times they have been on oral steroids during the past year.
N. Review all medications including over-the-counter (OTC) and herbal supplements and their effectiveness for asthma control.
O. Ask if prescription or OTC inhalers are used and to what extent.

PHYSICAL EXAMINATION

A. Check temperature, blood pressure (BP), pulse, respirations, and pulse oximetry. Measure the client's height, weight, body mass index (BMI), and waist circumference because obesity is associated with asthma.
B. Inspect:
1. Observe comfort with respiratory pattern, skin tones, and use of accessory muscles of breathing.
2. Observe for hyperexpansion of thorax and signs that accessory muscles are being used (retractions, nasal flaring) or stridor.
3. Note appearance of hunched shoulders and/or chest deformity.
4. Inspect the ears, nose, and throat. Evaluate the presence of enlarged tonsils and adenoids, and nasal polyps.
5. Inspect the skin for eczema, dermatitis, or other irritation that might signal allergy.
6. Observe for allergic shiners and pebbled conjunctiva.
7. Observe for digital clubbing.

C. Auscultate:
1. Auscultate lung sounds. Note wheezing during normal expiration and prolonged expiration, which is seen with asthma.
2. Listen to all lung fields for an asymmetric wheeze.
3. Auscultate heart rate.

D. Percuss:
1. Percuss the lung fields.

DIAGNOSTIC TESTS

A. Spirometry is the gold standard. Peak flow meter measurements are not a substitute for spirometry. Evaluate the forced vital capacity (FVC) and FEV1 before and after the client inhales a short-acting bronchodilator.
B. Chest radiograph (CXR) is not recommended unless there is a compelling reason or suspicion for an alternative diagnosis and complete blood count (CBC) to assess for infection or eosinophilia.
C. Check peak expiratory flow rate (PEFR) after inhalation of short-acting beta-2 agonist (SABA). Diagnosis is confirmed if:
1. There is a 15% increase in PEFR after 15 to 20 minutes.
2. PEFR varies more than 20% between arising and 12 hours later in clients taking bronchodilators (or 10% without bronchodilators).
3. There is a greater than 15% decrease in PEFR after 6 minutes of running or exercise.

D. Consider a bronchial provocation test with histamine or methacholine for nondiagnostic spirometry.

DIFFERENTIAL DIAGNOSES

A. Asthma.
B. Chronic obstructive pulmonary disease (COPD).
C. GERD.
D. Congestive heart failure (CHF).
E. Cough secondary to medications such as an ACE inhibitor or beta-blocker.
F. Pneumonia.
G. Pulmonary embolism.
H. Laryngeal dysfunction.
I. Benign and malignant tumors.
J. Vocal cord dysfunction.
K. Obstructive sleep apnea (OSA).
L. Tuberculosis (TB).
M. Postviral syndrome.
N. Obesity.
O. Allergic rhinitis.
P. Aspiration.

PLAN

A. General interventions:

1. Review proper medication dosages. Short- and long-term agents come in several formulations: nebulizer, metered-dose inhaler (MDI), and a dry powder inhaler (DPI).

2. Demonstrate correct use of inhalers, spacers, and nebulizers. If the client does not use correct technique when using these devices, medication does not get delivered to the bronchioles and therefore the client may believe the medication does not work. Most often the medication works well when delivered to the bronchioles correctly. *See Client Teaching Guide for this chapter, "How to Use a Metered-Dose Inhaler."* The easiest delivery mechanism should be considered for the elderly.

3. Stress the importance of using a peak flow monitor at home to monitor progress of the disease. *See Client Teaching Guide for this chapter, "Asthma Action Plan and Peak Flow Monitoring."*

4. SABA is used for rescue from acute symptoms. Ask the client to state or identify which inhaler they use for "rescue."

5. Use of a SABA more than twice a week for symptom relief indicates the client has inadequate asthma control and needs an inhaled corticosteroid (ICS) as controller therapy.

6. Stress the need for an asthma action plan. *See Client Teaching Guide for this chapter, "Asthma Action Plan and Peak Flow Monitoring."*

B. Client teaching: *See Client Teaching Guide for this chapter, "Asthma."*

1. Asthma is more difficult to control in obesity. Weight reduction should be included in the treatment plan for obese clients with asthma; even 5% to 10% weight loss can improve asthma control.

C. Pharmaceutical therapy:

1. Drugs are prescribed in a stepwise fashion for the type of asthma. The amount of medication used depends on the severity of asthma.

2. Prior to any medication/dosage changes, evaluate if the client has been doing incorrect inhaler technique for medication delivery and monitor the client's compliance with use of medications.

3. Reevaluate the client's modifiable risk factors (e.g., smoking, weight) and evaluate if symptoms are due to comorbid conditions, for example, allergic rhinitis.

4. The 2021 Global Initiative for Asthma (GINA) guidelines outline the medication classes for the approach to Steps 1 to 5.

5. See Table 12.1 for a list of medications for asthma. The following treatments are recommended:

a. Step 1:
   i. Low-dose ICS-formoterol as needed.
   ii. SABA as needed for rescue medication; may be used Q4H as needed to treat exacerbation.
   iii. GINA guidelines add low-dose ICS-formoterol taken when the rescue inhaler is used as another option to prevent exacerbations.

b. Step 2:
   i. Preferred controller: low-dose ICS-formoterol as needed for symptoms and prior to exercise.
   ii. Alternative controller options:
      1) Daily low-dose ICS with SABA as needed for symptoms. GINA prefers low-dose ICS because adherence to daily ICS therapy may be low, predisposing clients to severe exacerbations if SABA is the only treatment the client is using.
      2) **Low-dose ICS taken whenever SABA is taken.**
      3) **Combination low-dose ICS and long-acting beta-2 agonist (LABA).**
      4) Leukotriene receptor antagonist (LTRA).
      5) OR low-dose ICS daily or low-dose ICS-formoterol as needed for clients with seasonal symptoms. Start the medication when symptoms begin and continue for 4 weeks.
   iii. Reliever: Continue to use SABAs as "rescue" as needed.

c. Step 3: Prior to stepping up to step 3, verify client medication administration technique, medication adherence, and potential triggers.
   i. Low-dose ICS-formoterol daily and as needed for symptoms: Maximum daily dose of budesonide formoterol is 72 mcg metered dose and 54 mcg delivered dose. Maximum daily dose of beclometasone formoterol is 48 mcg metered dose and 36 mcg delivered dose.
      1) ICS-formoterol used as needed for rescue has not been studied with ICS-LABA control regimens and should not be recommended.
   ii. Other controller options:
      1) ICS-LABA with SABA as needed for symptoms.
      2) Sublingual allergen immunotherapy (SLIT).
      3) Medium-dose ICS.
      4) Low-dose ICS plus either a leukotriene modifier or a low-dose sustained-release theophylline.
   iii. Reliever: Continue to use SABAs as "rescue" as needed OR low-dose ICS/formoterol.

d. Step 4: Assess medication administration technique, adherence, and exposure to triggers.
   i. Preferred controller: medium-dose ICS-formoterol with low-dose ICS-formoterol as needed for symptoms.
      1) Maximum daily dose of budesonide formoterol is 72 mcg metered dose and 54 mcg delivered dose. Maximum daily dose of beclometasone formoterol is 48 mcg metered dose and 36 mcg delivered dose.
      2) Medium-dose ICS with LABA and SABA for symptoms.
      3) Add a long-acting muscarinic inhibitor (LAMA) for persistent symptoms.
      4) SLIT for persistent symptoms with allergy component.
   ii. Other controller options:
      1) Medium-dose ICS plus either an LTRA or add theophylline.
      2) Tiotropium by mist inhaler is an add-on treatment for clients with a history of exacerbation (not indicated for children <12 years).
   iii. Reliever: Continue to use SABAs as "rescue" as needed OR low-dose ICS/formoterol.

e. Step 5: Refer for expert investigation and add-on treatments.
   i. Add-on treatments include the following:
      1) Tiotropium by mist inhaler for clients with a history of exacerbations (≥12 years of age). ▶

TABLE 12.1 MEDICATIONS FOR ASTHMA AND COPD (BY CLASS/ALPHABETICAL ORDER)

Brand	Generic	Drug Classification	Availability
SABA, for Rescue/Fast-Acting Rescue			
AccuNeb	Albuterol	Beta-2 agonist	Nebulizer
Albuterol	Albuterol sulfate	Beta-2 agonist	Inhaler solution Nebulizer Syrup
Alupent Combivent	Metaproterenol Ipratropium + albuterol	Beta-2 agonist Combination anticholinergic + beta-2 agonist	Oral Oral aerosol Nebulizer MDI
Brethine	Terbutaline sulfate	Beta-2 agonist	Tablet
Fenoterol Maxair Autohaler	Ipratropium + fenoterol Pirbuterol acetate	Beta-2 agonist Beta-2 agonist	Inhaler MDI Oral syrup
ProAir HFA inhaler	Albuterol	Beta-2 agonist	MDI
Proventil HFA inhaler	Albuterol	Beta-2 agonist	MDI
Tornalate Ventolin HFA	Bitolterol Albuterol sustained-release	Beta-2 agonist Beta-2 agonist	Aerosol Inhaler solution MDI
Vospire ER	Albuterol	Beta-2 agonist	ER tablet
Xopenex	Levalbuterol tartrate	Beta-2 agonist	HFA inhaler Concentrate solution for nebulizer
LABA, for Maintenance/Long-Acting Control (Partial List)			
Advair Diskus	Fluticasone + salmeterol	Combination-steroid + LABA	Dry powder Diskus MDI-HFA inhaler
AirDuo RespiClick Arcapta Breo Ellipta Brovana	Fluticasone, salmeterol Indacaterol Fluticasone, vilanterol Arformoterol	Corticosteroid + LABA Long-acting beta-1 agonist Corticosteroid + LABA Beta-2 agonist	Inhaler Nebulizer DPI Nebulizer
Dulera Foradil Aerolizer	Mometasone, formoterol Formoterol fumarate	Corticosteroid + LABA, LABA	Inhaler Nebulizer Dry powder Diskus
Perforomist	Formoterol solution	Beta-2 agonist	Nebulizer
Serevent Diskus Symbicort	Salmeterol Budesonide, formoterol	LABA LABA	Dry powder Diskus Pressurized MDI
Aerobid and Aerobid M	Flunisolide	ICS	MDI MDI with menthol
Alvesco	Ciclesonide	ICS	MDI
Asmanex	Mometasone	ICS	Twisthaler dry powder
Beclovent	Beclomethasone dipropionate	ICS	Dipro powder inhaler
Dulera Flovent HFA	Mometasone furoate + formoterol, fluticasone	Combination corticosteroid + LABA ICS	MDI MDI (also available as a DPI as Flovent Diskus)
Pulmicort	Budesonide	ICS	Flexhaler and respules
QVAR	Beclomethasone	ICS	MDI
Symbicort Breo Ellipta	Budesonide + formoterol Fluticasone + vilanterol	Combination corticosteroid + LABA Combined corticosteroid + LAMA	MDI DPI

(*continued*)

TABLE 12.1 MEDICATIONS FOR ASTHMA AND COPD (BY CLASS/ALPHABETICAL ORDER) (*CONTINUED*)

Brand	Generic	Drug Classification	Availability
Leukotriene Modifiers: Nonsteroidal Anti-Inflammatory			
Accolate	Zafirlukast	Leukotriene modifier	Tablet
Singulair	Montelukast	Leukotriene modifier	Chew tablet and granules
Zyflo	Zileuton	Leukotriene modifier	Filmtab
Anticholinergics/LAMA			
Anoro Ellipta Atrovent HFA	Umeclidinium + vilanterol Ipratropium	Anticholinergic/LABA Antimuscarinic/antispasmodic	DPI MDI and nebulizer
DuoNeb	Ipratropium + Albuterol	Combination antimuscarinic/antispasmodic + beta-2 agonist	Nebulizer
Seebri Neohaler Spiriva with HandiHaler Tudorza Pressair Incruse Ellipta	Glycopyrrolate Tiotropium Aclidinium Umeclidinium	Long-acting antimuscarinic Antimuscarinic/antispasmodic Anticholinergic Anticholinergic	DPI Dry powder Diskus DPI DPI
Interleukin-5 Antagonists			
NUCALA	**Mepolizumab**	**Interleukin-5 antagonists**	**Injection**
CINQAIR	**Reslizumab**	**Interleukin-5 antagonist**	**Injection**
Mast Cell Stabilizer			
Intal	Cromolyn sodium	Mast cell stabilizer	MDI
Tilade Gastrocrom	Nedocromil Cromolyn	Mast cell stabilizer Mast cell stabilizer	MDI MDI
Methylxanthine			
Aminophylline Theo-24	Theophylline + ethylenediamine Theophylline sustained-release	Methylxanthines respiratory smooth muscle relaxant Methylxanthines respiratory smooth muscle relaxant	Tablet Tablet
Anti-IgE (IgE Blocker) Monoclonal Antibody			
Xolair	Omalizumab	IgE blocker-immunomodulator	Subcutaneous injection
Systemic Corticosteroids: Used as a Short-Term "Burst" for Control			
Deltasone	Prednisone	Systemic corticosteroid	Tablet
Medrol	Methylprednisolone	Systemic corticosteroid	Tablet
Prelone	Prednisolone	Systemic corticosteroid	Tablet
PDE4 Inhibitors			
Daliresp	Roflumilast	Selective PDE4	Tablet

COPD, chronic obstructive pulmonary disease; DPI, dry powder inhaler; ER, extended-release; HFA, hydrofluoroalkanes; ICS, inhaled corticosteroid; IgE, immunoglobulin E; LABA, long-acting beta-2 agonist; LAMA, long-acting muscarinic antagonists; MDI, metered-dose inhaler; PDE4, phosphodiesterase 4; SABA, short-acting beta-2 agonist.

2) Anti-immunoglobulin E (IgE; subcutaneous [SC] omalizumab) for severe allergic asthma (≥6 years of age).

3) Anti-interleukin 5 for severe eosinophilic asthma (SC mepolizumab or SC benralizumab for ≥12 years of age or intravenous [IV] reslizumab for ≥18 years of age).

4) Sputum-guided treatment, if available, improves outcomes.

ii. Other controller options:

1) Some clients may benefit from low-dose oral corticosteroid (OCS) but long-term systemic side effects commonly occur.

iii. Reliever: Continue to use SABAs as "rescue" as needed OR low-dose ICS/formoterol.

▶

f. Mild to moderate asthma:
   i. Leukotriene modifiers are used to reduce the need for short-acting, inhaled beta-2 agonists. They are also an alternative to low-dose ICS for clients with mild persistent asthma.
g. Exercise-induced bronchospasm:
   i. Short-acting inhaled beta-2 agonists, two inhalations shortly before exercise, are effective for 2 to 3 hours.
   ii. LABA, two inhalations, is effective 10 to 12 hours. LABA should be used cautiously unless combined with an ICS.
h. Hypertension and asthma:
   i. Drug of choice is a calcium channel blocker. Asthmatics also tolerate diuretics well.
i. Theophylline can cause cardiac arrhythmias; therefore, use it with caution and always follow up with theophylline levels.
j. Always ask about asthma before prescribing NSAIDs, including aspirin—aspirin-exacerbated respiratory disease (AERD).
k. Vaccinations:
   i. Influenza vaccine should be administered annually to asthmatics.
   ii. Pneumococcal vaccines are recommended for clients age 19 and older with chronic lung disease, including asthma.

FOLLOW-UP

A. After acute episodes, follow up within 1 to 2 hours or next day to monitor improvement until client is stable.

B. For clients with mild intermittent or mild persistent asthma under control for at least 3 months, assess and follow up at least every 6 months to assess risk factors, provide education, and reinforce positive behaviors.

C. Gradually reduce medication dosage. If control is not achieved, consider increasing dosage after reviewing medication technique, compliance, and environmental control.

EMERGENT ISSUES/INSTRUCTIONS

A. If the client's SOB is not relieved by two inhalations of their SABA, or they are reading in the Red Zone of their peak flow, they should understand this signals a medical emergency and should call emergency medical service (EMS).

B. Clients should clearly state or show you their SABA used for "rescue."

CONSULTATION/REFERRAL

A. Consider hospitalization for clients with acute episodes who do not completely respond to treatment within 1 to 2 hours.

B. If all therapies fail—including a short burst of prednisone—refer the client to an asthma specialist.

C. Consult with a physician when the client is pregnant or has other medical problems, or when standard treatment is ineffective.

D. Refer if the client presents with atypical symptoms.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Risks of uncontrolled asthma far outweigh risks to mother or fetus from drugs used to control the disease.

2. Step therapy should be followed in pregnancy according to guidelines for adults.

3. Classes of drugs that cause risk include decongestants, antibiotics (tetracycline, sulfonamides, and ciprofloxacin), live virus vaccines, immunotherapy (if doses are increased), and iodides. Always weigh benefits against risks because adequate fetal oxygen supply is essential. The obstetrician should be consulted.

4. If corticosteroids are necessary, recommend aerosolized forms due to their lower systemic effects. Prednisone and methylprednisolone are preferred and should be prescribed at minimum effective doses.

5. Do not prescribe inhaled triamcinolone because it is teratogenic.

6. Drugs recommended during pregnancy:
   a. A beta-2 agonist, such as terbutaline, is preferred: two inhalations Q4H as needed up to eight inhalations per day. Regular daily use suggests a need for additional medications.
   b. Cromolyn: two inhalations QID as initial therapy for clients needing regular medication.
   c. Regular inhaled beclomethasone if cromolyn is not effective.
   d. Regular oral theophylline if beclomethasone is not effective.
   e. Oral prednisone if all other therapies fail: 1 week of 40 mg/d, followed by 1 to 2 weeks of tapering.

7. Leukotriene inhibitors should be prescribed in pregnancy only if clearly needed:
   a. Accolate is excreted in the breast milk and should not be prescribed to mothers who are breastfeeding.
   b. Fetal anomalies have been reported with Zyflo.

B. Geriatrics:

1. Asthma may be underdiagnosed in the elderly due to poor perception, an assumption that dyspnea is normal in old age, lack of fitness, or reduced activity.

2. Asthma may be overdiagnosed in the elderly through confusion with SOB due to left ventricular failure or ischemic heart disease.

3. Respiratory viruses may be a common trigger for the first episode of asthma in the geriatric client.

4. While obesity is a risk factor for asthma in geriatrics, malnutrition (e.g., undernutrition) may play an important a role as a risk factor for asthma.

5. Elderly clients with dementia are generally unable to perform the FVC and FEV1 diagnostic tests. In the elderly with dementia, consider COPD in older adults who have any of the following factors. The greater the number of factors, the more likely COPD is present:
   a. Dyspnea that is progressive or worse with exercise.
   b. Chronic cough, either productive or nonproductive.
   c. History of chronic exposure to tobacco smoke, smoke from heating fuels, dust, or chemicals.
   d. Family history of COPD.

6. Treatment is the same as with younger clients, with inhaled steroids the mainstay and oral steroids reserved for severe episodes. The elderly have more adverse effects from inhaled ICS.

7. Chronic steroid use requires careful monitoring for complications including cataracts, increased intraocular pressure, hyperglycemia, and accelerated loss of bone mass.

8. Inhaled anticholinergics and beta-2 agonists are second-line treatment.

9. Consider nebulized treatments for older adults in the following conditions who may not be able to appropriate use inhalers:
   a. Decreased manual dexterity due to arthritis or reduced grip strength. ▶

b. Cognitive impairment: Difficulty coordinating the required activation/inhalation steps to achieve proper penetration of medication into the lungs.

10. Theophylline is rarely effective in the elderly. Asthma medications may have increased adverse effects in the elderly or may aggravate coexisting medical conditions, requiring medication adjustments. Also consider drug interactions and drug and disease interactions.

11. For older adults who are cognitively intact or mildly impaired, or have manual dexterity/decreased grip strength, spacing chambers may be used to assist in the proper delivery of inhaled medications.

12. Depression plays an important role in asthma management; geriatrics especially should be monitored for depression and its effect on chronic management.

RESOURCES

Adult and Children Asthma Control tests: www.asthma.com
American Lung Association: www.lung.org
Asthma & Allergy Foundation of America: www.aafa.org
Global Initiative for Asthma (GINA) Instructions for Inhaler and Spacer Use: www.ginasthma.org
National Heart, Lung, and Blood Institute (NHLBI): www.nhlbi.nih.gov
Pulmonary Disease Aerosol Delivery Devices: A Guide for Physicians, Nurses, Pharmacists, and Other Health Care Professionals is available at www.aarc.org/wp -content/uploads/2018/03/aersol-guides-for-hcp.pdf

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BRONCHITIS, ACUTE

DEFINITION

A. Acute bronchitis is inflammation of the tracheobronchial tree. Bronchitis is nearly always self-limited in the otherwise healthy individual. Generally, the clinical course of acute bronchitis lasts 10 to 14 days. The cause is usually infectious, but allergens and irritants may also produce a similar clinical profile. Asthma can be mistaken as acute bronchitis if the client has no history of asthma.

INCIDENCE

A. Bronchitis is more common in fall and winter in relation to the common cold or other respiratory illness. It occurs in males more frequently than in females. Fewer than 5% of clients with bronchitis develop pneumonia. It is one of the most common conditions seen in outpatients. Bronchitis is listed as the sixth most common reason for office visits to a medical provider.

PATHOGENESIS

A. Most attacks are caused by viral agents, such as adenovirus, influenza, parainfluenza viruses, and respiratory syncytial virus (RSV).

B. Bacterial causes (account for only 6% of acute bronchitis cases) include *Bordetella pertussis, Mycobacterium tuberculosis, Corynebacterium diphtheriae*, and *Mycoplasma pneumoniae. B. pertussis* should be considered in adults with persistent coughs.

PREDISPOSING FACTORS

A. Viral infection.
B. Upper respiratory infection (URI).
C. Exposure to cigarette smoke.
D. Exposure to other irritants.
E. Allergens.
F. Chronic aspiration/gastroesophageal reflux disease (GERD).
G. Immunocompromised and frailty.

COMMON COMPLAINTS

A. The most common symptom initially is a dry, hacking, or raspy-sounding cough. The cough then loosens and becomes productive.

OTHER SIGNS AND SYMPTOMS

A. Sore throat.
B. Rhinorrhea or nasal congestion.
C. Rhonchi during respiration.
D. Low-grade fever.
E. Malaise.
F. Retrosternal pain during deep breathing and coughing.
G. Decreased/lack of appetite.
H. Headache.
I. Dyspnea.

SUBJECTIVE DATA

A. Ask about onset, duration, and course of symptoms. Note duration of cough symptoms.
B. Is cough productive?
C. Is there substernal discomfort?
D. Is there malaise or fatigue?
E. Has client had a fever?
F. Does client smoke? (Smoking aggravates bronchitis.)
G. A review of occupational history may be important in determining whether irritants play a role in symptoms.
H. Assess for symptoms of gastroesophageal reflux.
I. If diabetic, ask about home blood glucose readings.
J. Ask about recent cold symptoms.

PHYSICAL EXAMINATION

A. Check temperature, pulse, and blood pressure (BP). Consider pulse oximetry. Evaluate general appearance.
B. Inspect:
  1. Observe overall appearance, including level of consciousness (LOC).
  2. Inspect eyes, ears, nose, and throat (pharynx may be injected).
  3. Transilluminate sinuses.
C. Palpate:
  1. Palpate the lymph nodes, maxillary, and frontal sinuses.
D. Auscultate:
  1. Auscultate in all lung fields for crackles, wheezing, and rhonchi.

DIAGNOSTIC TESTS

A. Consider chest x-ray to exclude pneumonia if fever, tachycardia, or tachypnea is present.

DIFFERENTIAL DIAGNOSES

A. Bronchitis.
B. URI.
C. Asthma.
D. Sinusitis.
E. Cystic fibrosis (CF).
F. Aspiration.
G. Respiratory tract anomalies.
H. Foreign-body aspiration.
I. Pneumonia.

▶

J. Chronic obstructive pulmonary disease (COPD) and emphysema.
K. Pertussis.
L. GERD.
M. Malignancy.
N. Heart failure.
O. Postnasal drip.
P. Medications (angiotensin-converting enzyme inhibitor).

PLAN

A. General interventions:
1. Primarily supportive and should ensure the client is adequately oxygenating.
2. Instruct the client to increase fluid intake.
3. Suggest humidity and mist therapy.
4. Avoid irritants, such as smoke.
B. Client teaching: *See Client Teaching Guide for this chapter, "Bronchitis, Acute."*
C. Pharmaceutical therapy:
1. Acetaminophen (Tylenol) for fever and malaise.
a. Adults: 625 to 1,000 mg orally Q4H not to exceed 4 g/d.
2. Expectorants such as guaifenesin with dextromethorphan (Robitussin DM, Humibid DM, Mytussin) to treat minor cough from bronchial/throat irritation. Cough suppressants including opiates are rarely appropriate and include risk of sedation in elderly.
3. Among otherwise healthy individuals, antibiotics have not demonstrated to be beneficial for acute bronchitis and are against the Centers for Disease Control and Prevention (CDC) current recommendations. Symptoms can persist for up to 4 weeks.
4. Albuterol (Ventolin) for clients with wheezes or rhonchi or for clients with a history of bronchoconstriction.
a. Two puffs every 4 to 6 hours or 2 to 4 mg PO three to four times a day.

FOLLOW-UP

A. Follow up if symptoms persist for more than 3 weeks. Educate on symptoms that warrant sooner follow-up, such as fever, dyspnea, and bloody sputum.
B. Recommend yearly influenza vaccinations.
C. Ensure older adults have both pneumonia vaccinations recommended by the CDC.

EMERGENT ISSUES/INSTRUCTIONS

A. Clients should understand to call the office or go to the ED if shortness of breath (SOB) develops.

CONSULTATION/REFERRAL

A. In uncomplicated cases, mucus production decreases and cough disappears in 7 to 10 days. If symptoms persist, refer the client to a physician.
B. Refer the client if you note respiratory distress or worsening condition.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. Use of handheld bronchodilator should be demonstrated. Older adults who are cognitively intact or mildly impaired may be unable to use standard inhalers. Spacing chambers may be used to assist in proper delivery of inhaled medications. Nebulized treatments should also be considered for the following issues:
a. Decreased manual dexterity due to arthritis or reduced grip strength.
b. Cognitive impairment: difficulty coordinating the required activation/inhalation steps to achieve proper penetration of medication into the lungs.
2. Geriatric red flags:
a. Monitor elderly clients for complications such as pneumonia. The elderly have a greater morbidity and mortality rate and may present with confusion or loss of appetite only.
b. Follow-up may be needed every 24 hours to ensure improvement and to assess changes associated with sepsis.

RESOURCE

Pulmonary Disease Aerosol Delivery Devices: A Guide for Physicians, Nurses, Pharmacists, and Other Health Care Professionals is available at www.aarc.org/wp -content/uploads/2018/03/aersol-guides-for-hcp.pdf

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BRONCHITIS, CHRONIC

DEFINITION

A. Chronic bronchitis is excessive mucus secretion with chronic or recurrent productive cough occurring in 3 successive months a year for 2 consecutive years.
B. Others limit the definition to a productive cough that lasts more than 2 weeks despite therapy.
C. Clients with chronic bronchitis have more mucus than normal due to either increased production or decreased clearance. Coughing is the mechanism for clearing excess secretion.

INCIDENCE

A. The incidence of chronic bronchitis is uncertain. There is a lack of definitive diagnostic criteria and there is considerable overlap with asthma. Visits for bronchitis are second only to visits for otitis media and are slightly more common than for asthma.
B. The prevalence of chronic bronchitis increases with age.
C. The rates of chronic bronchitis are highest in African American and Caucasian ethnicities.

PATHOGENESIS

A. Mucociliary clearance is delayed due toexcess mucus production and loss of ciliated cells, leading to a productive cough.
B. The bacteria most often implicated are *Streptococcus pneumoniae, Haemophilus influenzae, Mycoplasma pneumoniae*, and *Moraxella catarrhalis.*
C. Specific occupational exposures are associated with symptoms of chronic bronchitis, including coal, cement, welding fumes, organic dusts, engine exhausts, fire smoke, and secondhand smoke.

PREDISPOSING FACTORS

A. Cigarette smoking.
B. Cold weather.
C. Acute viral infection.
D. Chronic obstructive pulmonary disease (COPD)/emphysema.

E. Occupational exposure to other airborne irritants.
F. Chronic, recurrent aspiration or gastroesophageal reflux.
G. Allergies.
H. Immunosuppression.
I. Frailty.

COMMON COMPLAINTS

A. Worsening cough: hacking, harsh, or raspy sounding.
B. Changes in color (yellow, white, or greenish), amount, and viscosity of sputum.
C. "Rattling" sound in chest.
D. Dyspnea/breathlessness.
E. Wheezing.

OTHER SIGNS AND SYMPTOMS

A. Difficulty breathing and retrosternal pain during a deep breath or cough.
B. Rapid respirations.
C. Fatigue.
D. Headache.
E. Loss of appetite.
F. Fever.
G. Myalgias.
H. Arthralgias.
I. Sleep disturbance due to cough.

SUBJECTIVE DATA

A. Determine the onset, course, and duration of illness.
B. Is the client having trouble breathing?
C. Has there been a fever?
D. How is the client's appetite? Is the client drinking enough fluids?
E. Does the client smoke, or is the client exposed to secondhand smoke?
F. Review occupational history to evaluate exposure to irritants.
G. Does the client have a history of asthma?

Chronic bronchitis has a long history of a productive cough and late-onset wheezing. Clients with asthma with a chronic obstruction have a long history of wheezing with a late onset of productive cough.

H. Is there chest pain in any certain area?
I. When was the client's last illness?
J. Determine blood glucose levels if diabetic.
K. Determine the frequency of short-acting beta-2 agonists (SABA) inhaler use and effectiveness.
L. Determine any confusion or lack of appetite (especially in advanced age).
M. Assess the severity of symptoms using the modified Medical Research Council (mMRC) Dyspnea Scale or the COPD Assessment Test (CAT) or the Refined ABCD Assessment Tool.
N. Review medications. There is mixed evidence on the safety of beta-blockers and gabapentinoids as each may increase the risk of breathlessness.

PHYSICAL EXAMINATION

A. Check temperature, pulse, blood pressure (BP), respirations, and pulse oximetry.
B. Inspect:
1. Observe overall appearance, including signs of respiratory distress.
2. Inspect eyes, ears, nose, and throat.
a. Pharynx may be injected.
b. Conjunctivitis suggests adenovirus.
3. Transilluminate sinuses.
4. Assess skin turgor and mucous membranes for dehydration.
5. Assess nutritional status as cachexia may be noted in advanced disease.
C. Auscultate:
1. Auscultate the lungs in all fields. Lung sounds may sound normal to scattered, bilateral crackles, rhonchi, or large airway wheezing.
2. Auscultate heart.
D. Percuss:
1. Percuss the chest.
E. Palpate:
1. Anterior/posterior cervical lymph nodes.
2. Maxillary and frontal sinuses.

DIAGNOSTIC TESTS

A. Clients with uncomplicated respiratory illness need little, if any, laboratory evaluation.
B. Pulse oximetry.
C. Sputum culture to identify bacteria.
D. Chest radiograph (CXR) may help exclude other diseases or complications.
E. Pulmonary function studies may be indicated.
F. EKG and pulmonary function tests (PFTs) may be required for COPD clients.
G. Consider C-reactive protein(CRP) to help guide the decision to start antibiotics. Low CRP may be an indicator that antibiotics are not necessary.

DIFFERENTIAL DIAGNOSES

A. Chronic bronchitis.
B. Acute bronchitis.
C. Asthma.
D. Sinusitis.
E. Cystic fibrosis (CF).
F. Bronchiectasis.
G. Central airway obstruction.
H. Pneumonia.
I. Lung cancer.
J. Aspiration syndrome.
K. Gastroesophageal reflux.
L. Tuberculosis (TB).
M. Foreign body.
N. Heart failure.

PLAN

A. General interventions:
1. Rest during early phase of illness.
2. Encourage stopping smoking and staying away from secondhand smoke.
3. Suggest exercise for clients with COPD.
4. The client's goal is to improve symptoms and to reduce cough and production of sputum.
5. Inform the clients that increased sputum production may occur after smoking cessation and that they may have airway reactivity (wheezing), especially seen in asthmatics.
6. Discuss the clients's choices of advance directives preferably before exacerbation occurs.
B. Client teaching: *See Client Teaching Guide for this chapter, "Bronchitis, Chronic."*
C. Dietary management:
1. Increase fluids unless contraindicated.
2. Eat nutritious food.

D. Pharmaceutical therapy:

1. Bronchodilators should be considered for bronchospasm.

a. Albuterol sulfate (Proventil, Ventolin):

i. Adults: metered-dose inhaler (MDI) actuations (90 mcg/actuation) inhaled every 4 to 6 hours.

2. Analgesics and antipyretics are used to control fever, myalgias, and arthralgias.

3. Consider oral steroids to reduce inflammation.

a. Adults: 5 to 60 mg/d PO.

b. Tapering steroids is not necessary with short courses.

4. Inhaled corticosteroid (ICS) may be effective.

a. Beclomethasone (QVAR): available as an MDI that delivers 40 or 80 mcg/actuation:

i. 40 to 80 mcg inhaled PO BID; not to exceed 320 mcg BID.

b. Fluticasone (Flovent HFA, Flovent Diskus): available as MDI (44, 110, or 220 mcg/actuation) and Diskus powder for inhalation (50, 100, or 250 mcg/actuation):

i. Adults:

1) MDI: 88 mcg inhaled PO BID; not to exceed 440 mcg BID.

2) Diskus: 100 mcg inhaled PO BID; not to exceed 500 mcg BID.

5. Antibiotics for bacterial infection (two of the following should be present to consider antibiotics: dyspnea, increased cough, or purulent sputum).

a. Amoxicillin-clavulanic acid (Augmentin) 250 to 500 mg PO Q8H.

b. Doxycycline 100 mg PO Q12H.

c. Macrolide antibiotics such as erythromycin or azithromycin.

d. Suspected *Pseudomonas* coverage for 65 or older with cardiac disease: levofloxacin 500 mg daily.

FOLLOW-UP

A. Follow up if there is no improvement in 3 to 4 days after starting therapy.

B. Recommend yearly influenza vaccinations and vaccinations against pneumonia.

EMERGENT ISSUES/INSTRUCTIONS

A. Client should understand to seek emergency care should their shortness of breath (SOB) worsen, in accordance with the Advance Directive.

CONSULTATION/REFERRAL

A. Refer clients with respiratory distress to a physician. If respiratory failure occurs (rare), hospitalization may be needed.

B. Refer clients with COPD to a physician or pulmonary specialist.

C. Pulmonary rehab should be considered once recovered.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Follow-up may be needed every 24 hours to ensure improvement and to assess for worsening clinical presentation and assessment for developing sepsis.

2. Use of handheld bronchodilator should be demonstrated. Older adults who are cognitively intact or mildly impaired may be unable to use standard inhalers. Spacing chambers may be used to assist in proper delivery of inhaled medications. Nebulized treatments should also be considered for the following issues:

a. Decreased manual dexterity due to arthritis or reduced grip strength.

b. Cognitive impairment: difficulty coordinating the required activation/inhalation steps to achieve proper penetration of medication into the lungs.

3. The client's choice for aggressive care, including ventilation, should be noted in the chart and shared with family members.

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CHRONIC OBSTRUCTIVE PULMONARY DISEASE

DEFINITION

A. Chronic obstructive pulmonary disease (COPD) is a progressive, chronic, expiratory airway obstruction due to chronic bronchitis or emphysema. The relief of bronchoconstriction due to inflammation has some reversibility. Chronic bronchitis is a chronic productive cough lasting 3 months during 2 consecutive years, after all causes of chronic cough have been excluded. Emphysema is an abnormal, permanent enlargement (hyperinflation) of the air sacs, as well as the destruction of the elastic recoil. Many clients have both types of air restriction symptoms of chronic bronchitis and emphysematous destruction leading to COPD. Clients with asthma whose airflow obstruction is completely reversible are not considered to have COPD. When asthmatic clients do not have complete reversible airflow obstruction, they are considered to have COPD.

B. Irreversible airflow obstruction is a key factor in the client's disability. The goal of COPD management is to improve daily quality of life (QOL) and the recurrence of exacerbations. Smoking cessation continues to be the most important therapeutic intervention.

C. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) staging criteria are as follows:

1. Stage I—mild obstruction: forced expiratory volume in 1 second (FEV_1) greater than 80% of predicted value, some sputum, and chronic cough.

2. Stage II—moderate obstruction: FEV_1 between 50% and 80% of predicted value, shortness of breath (SOB) on exertion, and chronic symptoms.

3. Stage III—severe obstruction: FEV_1 between 30% and 50% of predicted value, dyspnea, reduced exercise tolerance, and exacerbations affecting QOL.

4. Stage IV—very severe obstruction chronic respiratory failure: FEV_1 less than 30% of predicted value; or moderate obstruction: FEV_1 less than 50% of the predicted value; and chronic respiratory failure.

5. Comorbidities commonly seen with COPD include hypertension, cardiac disorders including atrial fibrillation and heart failure, diabetes/metabolic syndrome, gastrointestinal disorders, lung cancer, depression, and osteoporosis.

INCIDENCE

A. Approximately 16 million people in the United States have been diagnosed with COPD. It is the third leading cause of death in the United States. Since 2011, COPD has been more commonly seen in females than in males. The exact worldwide prevalence is unknown as up to 50% of people with low lung function are undiagnosed.

PATHOGENESIS

A. Chronic bronchitis leads to the narrowing of the airway caliber and increase in airway resistance. Mucus gland enlargement is the histologic hallmark of chronic bronchitis.

B. In emphysema, loss of the air sac's elastic recoil causes air limitation. Emphysema caused by smoking is the most severe in the upper lobes. Most clients with COPD have smoked one pack of cigarettes a day for 20 or more years before the symptomatic dyspnea, cough, and sputum appear.

PREDISPOSING FACTORS

A. Cigarette smoking.

B. Occupational, environmental, or atmospheric pollutants:

1. Dust.

2. Chemical fumes.

3. Secondhand smoke.

4. Air pollution.

C. Genetic factor: alpha-1 antitrypsin (AAT) deficiency.

D. Recurrent or chronic lower respiratory infections or disease (childhood and as adult).

E. Age (most common after the fifth decade of life).

F. Females and Native Americans/Alaskan Natives have higher risk.

G. History of asthma.

H. Unemployment, including retirement.

I. Single status (divorced, widowed, or separated).

J. Less than high school education.

COMMON COMPLAINTS

A. Chronic cough and colorless sputum, usually worse in the morning.

B. Dyspnea with exertion, progressing to dyspnea at rest.

C. Wheezing.

D. Difficulty speaking or performing tasks.

E. Weight loss (decrease in fat-free mass).

OTHER SIGNS AND SYMPTOMS

A. Pursed-lip breathing.

B. Use of accessory muscles.

C. Tripod position.

D. Barrel chest.

E. Cyanosis (fingertips, tip of nose, around lips).

F. Tachypnea.

G. Tachycardia.

H. Difficulty speaking or performing tasks.

I. Jugular venous distension.

J. Abnormal, diminished, or absent lung sounds.

K. Mental status changes.

L. Anxiety and depression.

M. Pulmonary hypertension.

N. Cor pulmonale.

O. Left-sided heart failure.

SUBJECTIVE DATA

A. Ask the client about past respiratory problems and infections. Do they currently have fever, chills, or other signs of infection?

B. Ask about onset of cough and characteristics of sputum (amount, color, and presence of blood).

C. Determine cigar use and cigarette pack-year history (pack/day times the number of years smoked) and current interest in quitting.

D. Inquire about exposure to occupational or environmental irritants.

E. How far can the client walk before becoming breathless? Is there more breathlessness when the client walks on a slight incline? Have symptoms caused lost work days or productivity?

F. Does the client become breathless or tired when performing activities of daily living (ADL)?

G. Ask about insomnia, anxiety, restlessness, edema, and weight change.

H. How many pillows does the client sleep on? Do they have to sleep in a recliner or sitting up?

I. Assess the client's ability to perform ADL and instrumental activities of daily living (IADL), including grooming and personal hygiene, performing chores around the house, shopping, cooking, and driving.

J. Ask about alcohol use.

K. Review all medications, including over-the-counter (OTC) and herbal products.

L. Review further assessment questions based on existing comorbidities.

M. Depression screening.

N. Inquire about use of ED or need for hospitalization. Previous ventilation?

PHYSICAL EXAMINATION

A. Record temperature, blood pressure (BP), pulse, respirations, and pulse oximetry. The respiratory rate increases proportionally with disease severity. Take height and weight to calculate the body mass index (BMI). **The client may have a normal examination early in the disease.**

B. Inspect:

1. Observe general appearance: skin color, affect, posture, gait, and amount of respiratory effort when walking and speaking; note increased anterior–posterior chest diameter.

2. Examine sputum: Frothy pink signals pulmonary edema. Hemoptysis is seen in tuberculosis (TB).

3. Examine lips, fingertips, and nose for cyanosis. (Finger clubbing is not characteristic of COPD.)

4. Observe the neck for distended veins and peripheral edema (advanced disease).

5. Check for pursed-lip breathing and use of accessory muscles.

C. Auscultate:

1. Auscultate the heart.

2. Auscultate lungs for wheezes, crackles, decreased breath sounds, and prolonged forced expiratory rate.

3. Assess for vocal fremitus (vibration) and egophony (increased resonance and high-pitched bleating quality). Air trapping causes air pockets that do not transmit sound well. Absent vesicular lung sounds are a distinctive characteristic of COPD.

4. Auscultate carotids for bruits.

D. Percuss:

1. Percuss the chest for presence of hyperresonance and for signs of consolidation.

E. Palpate:

1. Palpate the neck and the superior and inferior clavicular spaces for lymphadenopathy.

2. Palpate the chest.

3. Palpate/percuss abdomen for organomegaly.

4. Grade dependent edema.

F. Mental status:

1. Assess for decreased level of consciousness (LOC).

G. Perform 6-minute walking distance (6MWD) testing to evaluate oxygen desaturation.

H. Further physical examinations are dependent on comorbidities. ▶

DIAGNOSTIC TESTS

A. Spirometry is the gold standard for diagnosing COPD. Pulmonary function tests (PFTs) are used to diagnose, determine severity, and follow the disease progression of COPD. Perform spirometry before and after using bronchodilator.

1. FEV1 is used as an index to airflow obstruction and evaluates the prognosis in emphysema.
2. Forced vital capacity (FVC).
3. FEV1/FVC ratios less than 0.70.

B. Chest radiograph (CXR; not required to diagnose COPD but rules out other diagnoses).

C. Complete blood count (CBC): Evaluate polycythemia due to chronic hypoxia.

D. Sputum specimen for culture.

E. If the client is younger than 40 years of age or has a family history of early onset of emphysema, measure AAT levels.

F. Arterial blood gas (ABG) for baseline.

G. EKG: Note sinus tachycardia and atrial arrhythmias.

H. Two-dimensional echocardiogram is used to evaluate secondary pulmonary hypertension.

I. Chest CT is an alternative imaging study for emphysema; however, it is not required as a diagnostic tool. Low-dose CT should be recommended as appropriate for annual lung cancer screening.

J. Perform a purified protein derivative (PPD) test if TB is suspected.

K. Brain natriuretic peptide (BNP).

L. Theophylline level (if applicable).

DIFFERENTIAL DIAGNOSES

A. COPD.
B. Asthma.
C. Heart failure.
D. Bronchiectasis.
E. Pulmonary edema.
F. TB.
G. AAT deficiency.
H. Pneumonia.
I. Pulmonary embolism.
J. Cystic fibrosis.
K. Cancers.
L. Central airway stenosis.
M. Diffuse panbronchiolitis (predominately seen in those of Asian descent).
N. Obliterative bronchiolitis.

PLAN

A. General interventions:

1. Educate and encourage active participation in the plan of care, including medication adherence.
2. A smoking cessation plan is an essential part of a comprehensive treatment plan. Develop a smoking cessation plan; assess readiness to quit. Set a quit date; encourage a group smoking cessation program. Pharmacotherapy and nicotine replacement reliably increase long-term smoking abstinence rates. **Discuss smoking at every subsequent visit. The effectiveness and safety of e-cigarettes as a smoking cessation aid are uncertain at this time.** *See Client Teaching Guide for Chapter 25, "Nicotine Dependence."*
3. The GOLD guidelines note a five-step program for smoking intervention:
   a. ASK: Systematically identify all tobacco users at every visit; *EVERY* client at *EVERY* clinic visit; is queried about tobacco use status.
   b. ADVISE: Strongly urge all tobacco users to quit in a clear, strong, and personalized manner.
   c. ASSESS: Determine willingness and rationale of the client's desire to make a quit attempt. Ask every tobacco user if they are willing to make a quit attempt at this time (e.g., within the next 30 days).
   d. ASSIST: Aid in quitting, make a quit plan, provide practical counseling, provide educational materials, and help the client obtain social support. Pharmacotherapy if appropriate.
   e. ARRANGE: Schedule a follow-up contact either in person or by telephone.
4. Consider pulmonary rehabilitation for all stages of COPD. Pulmonary rehab programs vary in format and length. Most programs use a small group format and include the following:
   a. Education about symptoms, medications, and oxygen.
   b. Supervised exercise classes and instruction.
   c. Breathing techniques.
   d. Nutritional counseling.
   e. Emotional health support.
5. The selection of inhalers is dependent on the client's age and ability to use the inhaler. Clients should be evaluated as to their coordination and inspiration abilities necessary to use inhalers; otherwise, aerosol medication via a nebulizer is the best delivery method.
6. Have the client bring in their medication/spacers to demonstrate correct use.
7. Consider group visits for teaching sessions.

B. Client teaching: *See Client Teaching Guide for this chapter, "Chronic Obstructive Pulmonary Disease."*

1. Advise to stay away from secondhand smoke and limit exposure to other pulmonary irritants, including aerosols/vapors and extreme temperature changes.
2. Advise exercise with provider approval.
3. Educate and counsel the client regarding advance directives.

C. Dietary management:

1. About 25% of COPD clients are malnourished due to coexisting medical conditions, depression, and inability to shop for or prepare food.
2. Suggest a low-carbohydrate diet. High-carbohydrate intake may increase respiratory work by increasing CO_2 production.

D. Pharmaceutical therapy: Treatment guidelines are based on spirometry. Prior to any medication/dosage changes, monitor the client's compliance (see Table 12.1).

1. Key points for use of bronchodilators:
   a. Long-acting beta-2 agonists (LABAs) and long-acting muscarinic agonists (LAMAs) are preferred over short-acting agents except for clients with only occasional dyspnea.
   b. Clients may start on a single long-acting bronchodilator or dual long-acting bronchodilator therapy. Clients with persistent dyspnea on one bronchodilator should be escalated to two.
   c. Inhaled bronchodilators are recommended over oral bronchodilators.

▶

d. Theophylline is not recommended unless other long-term treatment bronchodilators are unavailable or unaffordable. Theophylline has a narrow therapeutic range and frequent toxicity.

The target theophylline concentration is 5 to 10 µg/mL. Prescribing theophylline requires weight-based dosing. Dosage adjustment therapy is based on serum theophylline concentrations. Monitor theophylline level whenever there is a new illness, worsening chronic illness, or a change in medications that may alter theophylline clearance (e.g., fever >102°F sustained for ≥24 hours or altered liver function including hepatitis). Check drug interactions using an online checker: www.rxlist.com/drug-interaction-checker.htm.

i. Dosage should be titrated up. Titration steps include the following: the starting dose; after 3 days, *if tolerated*, increase dose; and after 3 more days, *if tolerated and if needed*, increase dose.

ii. Common side effects of theophylline include loss of appetite, abdominal pain, diarrhea, weight loss, restlessness, tremor, insomnia, headache, lightheadedness, dizziness, and sweating.

iii. Instruct the client to call 911 and get emergency medical help for severe headache, hyperglycemia, severe or persistent vomiting, cardiac arrhythmias, seizures, or confusion.

2. Key points for anti-inflammatory agents:

a. Long-term monotherapy with inhaled corticosteroid (ICS) is not recommended.

b. Long-term treatment with ICS may be considered in association with LABA in clients with a history of exacerbations despite appropriate treatment with long-acting bronchodilators.

c. Long-term therapy with oral corticosteroids is not recommended.

d. For clients with exacerbations despite LABA/ICS or LABA/LAMA/ICS, chronic bronchitis and severe to very severe airflow obstruction, the addition of a phosphodiesterase 4 (PDE4) can be considered.

Peak flow meters should not be used to diagnose or monitor COPD.

3. GOLD 1 (mild: FEV1 ≥80% predicted): The client may be unaware they have COPD. Give influenza vaccine and pneumococcal vaccines and and use anticholinergics or short-acting beta-2 agonists (SABA) bronchodilators as needed.

4. GOLD 2 (moderate: 50% ≤ FEV1 < 80% predicted): Give influenza vaccine, pneumonia vaccines, SABA bronchodilators as needed, plus long-acting anticholinergics or beta-2 bronchodilator(s) (LABA) plus cardiopulmonary rehabilitation.

5. GOLD 3 (severe: 30% ≤ FEV1 <50% predicted): Give influenza vaccine, pneumonia vaccines, SABA bronchodilators as needed, plus long-acting anticholinergic or beta-2 bronchodilator(s), plus cardiopulmonary rehabilitation, plus inhaled glucocorticoid steroids if client has repeated exacerbations.

6. GOLD 4 (very severe: FEV1 <30%): Give influenza vaccine, pneumonia vaccines, SABA bronchodilator as needed, plus long-acting anticholinergic or beta-2 bronchodilator(s), plus cardiopulmonary rehabilitation, plus inhaled glucocorticoid steroids if repeated exacerbations, plus long-term oxygen therapy (if the client meets the criteria for O_2). To qualify for home oxygen therapy, Medicare guidelines require the PaO_2 to be less than 55 mmHg or the resting oxygen saturation to be below 88% on room air.

7. For maximum effectiveness, utilization of a spacer/holding chamber for inhalers should be encouraged, especially in the elderly or those with difficulty activating metered-dose inhalers (MDIs).

8. Administer pneumonia vaccine in clients 65 years and older or beginning at age 19 if immunocompromised or chronic respiratory condition is present. Consider revaccination every 5 to 10 years in high-risk clients.

a. Sequential administration of the pneumococcal conjugate vaccine (PCV13) and pneumococcal polysaccharide vaccine (PPSV23) is recommended (download the Centers for Disease Control and Prevention's (CDC) guide at: www.cdc.gov/vaccines/vpd/pneumo/downloads/pneumo -vaccine-timing.pdf). In the vaccine-naive client, give PCV13 first, then the PPSV23 12 months later.

b. In older adults who received PPSV23 at the age of 65 or older, give PCV13 in 1 year or more.

c. In older adults who received PPSV23 BEFORE the age of 65, give PCV13, then 5 years after the initial PPSV23 give a second PPSV23.

d. Give a onetime second dose of PPSV23 in persons with chronic renal failure or nephrotic syndrome, functional or anatomic asplenia, or immunocompromising conditions.

Administer yearly flu vaccine. Trivalent or quadrivalent influenza vaccine is essential for all COPD clients. Those 65 years of age or older should receive high-dose trivalent vaccine. Give the client the vaccine in late September, at least 6 weeks before the flu season.

9. Prescribe pharmacologic agents/nicotine replacement therapy (NRT) for smoking cessation according to client preference:

a. Nicotine chewing gum produces better quit rates than counseling alone.

b. Transdermal nicotine patches have a long-term success rate of 22% to 42%.

c. The use of vapor nicotine products should be discussed as their long-term adverse effects on lung function are unknown.

d. Use of an antidepressant, such as bupropion sustained-release (Zyban), has been shown to be effective in smoking cessation by reducing the enjoyment of smoking, reducing withdrawal symptoms, and limiting weight gain. Bupropion may be used in combination with NRT. Begin dosing 1 week before quit date.

i. Days 1 to 3 of treatment: 150 mg every morning.

ii. Day 3 until the end of treatment: 150 mg BID, at least 8 hours apart.

iii. The duration of treatment should be for 7 to 12 weeks. If the client has not quit smoking after 7 to 12 weeks, it is unlikely that they will quit during that attempt so treatment should be discontinued and the treatment plan reassessed.

iv. Medication doses and/or frequency interval are required for renal and mild hepatitis impairment.

v. Moderate to severe hepatic impairment: 150 mg every other day.

e. Varenicline (Chantix) is a partial agonist selective for alpha-4, beta-2 nicotinic acetylcholine receptors. (Chantix is currently off the market as of June 2021. It was pulled from the market by the manufacturer due to having higher levels of nitrosamines, which may be ▶

carcinogenic. Generic forms are available.) Varenicline mimics the effect of nicotine; therefore, it reduces the urge to smoke and relieves withdrawal symptoms. It blocks or blunts the effect of nicotine if the person relapses and has a cigarette. Review the client's history of renal impairment, psychiatric disorders, seizure history/risk, alcohol use, and cardiovascular disease.

10. Antibiotics are not recommended in COPD clients except with acute exacerbation; symptoms of increased dyspnea, increased sputum volume, and increased sputum purulence; or changes in cough, fever, or other evidence of an infection such as an infiltrate on CXR. Antibiotics are prescribed for COPD clients on mechanical ventilation.

11. Mucolytic agents have small benefits and are not usually recommended.

12. Antitussives are not recommended.

13. Cardioselective beta-blockers are not contraindicated in COPD. A noncardioselective beta-blocker may contribute to bronchospasm.

14. Long-term oxygen has been shown to increase survival in clients with severe resting hypoxemia. Target oxygen saturation is 88% to 92%. Refer to a medical supply company if long-term oxygen or assistive devices are needed. To qualify for home oxygen therapy, Medicare guidelines require the PaO2 to be less than 55 mmHg or the resting oxygen saturation to be below 88% on room air.

FOLLOW-UP

A. For acute exacerbations, follow up on the same day or the following day.

B. Follow up stable, chronic COPD every 1 to 2 months, depending on the client's needs.

C. Serial PFTs may help guide therapy and offer prognostic information.

D. Reevaluate clients on oxygen therapy in 1 to 3 months after starting oxygen, including their current smoking status with oxygen in the home.

E. Evaluate for osteoporosis; bone mineral density is lower in COPD clients.

F. Monitor body weight.

EMERGENT ISSUES/INSTRUCTIONS

A. Monitor theophylline levels due to the drug's potential for toxicity.

1. Instruct the client to call 911 and get emergency medical help for severe headache, hyperglycemia, severe or persistent vomiting, cardiac arrhythmias, seizures, and confusion.

CONSULTATION/REFERRAL

A. Consult with a physician if the client has acute respiratory decompensation, severe cor pulmonale (jugular venous distension, hepatomegaly, dependent peripheral edema, ascites, or pleural effusion).

B. Refer the client to a pulmonary specialist for rehabilitation, if available:

1. Outpatient education for the client and the family.

2. Exercise training.

3. Breathing retraining, that is, pursed-lip breathing and huff coughing.

4. Correct administration of medications.

C. Refer to a registered dietitian (RD) to provide medical nutrition therapy (MNT). RDs focus on the prevention and treatment of weight loss associated with COPD and other comorbidities.

D. Send to a pulmonologist for evaluation for continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP).

E. Send to a pulmonologist to evaluate for surgical intervention, such as bullectomy, lung volume reduction surgery, or lung transplantation.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. COPD is rare except in AAT deficiency.

2. Monitor drug treatment for potential teratogenic effects.

B. Adults:

1. Sexual dysfunction is common in clients with COPD; encourage other ways to display affection.

2. Adults with COPD have twice the incidence and prevalence of disability compared with those without COPD. The association between COPD and disability is as strong as that for other chronic diseases, including stroke, diabetes, and heart disease. Monitor access to social support systems.

C. Geriatrics:

1. Presentation may be atypical.

2. Appearance or progression of one or more of these geriatric syndromes in a client with COPD is a red flag for poor outcomes. Monitor for the following:

a. Incontinence: difficulty in getting to the bathroom due to exertional dyspnea or equipment such as oxygen.

b. Falls: dizziness with exertion, often related to ADLs such as dressing and bathing; hazards, such as tripping over oxygen tubing.

c. Weight loss: SOB contributes to appetite loss and reduced ability to meet daily nutritional needs.

d. COPD is a disease requiring skills in self-management. Efforts to identify cognitive impairment and early deficits in self-care may be as important as other areas of COPD care.

3. Testing:

a. Elderly clients with dementia are generally unable to perform the FVC and FEV_1 diagnostic tests.

4. Medication administration:

a. Handheld MDIs, dry powder, or other types of inhalers cannot be used effectively in adults with cognitive impairment, reduced manual dexterity, or decreased hand grip strength and poor hand–eye coordination.

b. Clients should demonstrate use of their inhaler. If there are concerns, nebulizer therapy should be prescribed to improve the benefit of pharmacotherapy.

c. Clients should have annual flu vaccination and pneumococcal vaccinations according to the CDC guidelines. The 13- and 23-valent vaccines should be given sequentially. Download a guide to the sequential administration according to individual health considerations at: www.cdc.gov/vaccines/vpd/pneumo/downloads/pneumo-vaccine -timing.pdf.

5. Adults with COPD have twice the incidence and prevalence of disability compared with those without COPD. The association between COPD and disability is as strong as that for other chronic diseases, including stroke, diabetes, and heart disease. Monitor access to social support systems.

6. Discuss the trajectory of illness, living wills, advance directives, and resuscitation status early, before a crisis ▶

occurs. Make sure clients understand the potential for prolonged mechanical ventilation and potential need for tracheostomy should they become ventilator–dependent. Encourage clients to discuss their preferences with their family.

7. Beers list caution: theophylline:

a. Theophylline is on the Beers list of drugs to use with caution in the geriatric population related to cardiovascular, renal, hepatic, insomnia, and peptic ulcers.

i. Theophylline has a drug–drug interaction with cimetidine, increasing the risk of theophylline toxicity, and should be avoided (refer to Appendix C [Beers Criteria], Table C.4.)

RESOURCES

Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines: www.goldcopd.org

Pulmonary Disease Aerosol Delivery Devices: A Guide for Physicians, Nurses, Pharmacists, and Other Health Care Professionals is available at www.aarc.org/wp -content/uploads/2018/03/aersol-guides-for-hcp.pdf

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COMMON COLD/UPPER RESPIRATORY INFECTION

DEFINITION

A. The common cold is a self-limiting acute respiratory tract infection (ARTI) resulting from viral infection of the upper respiratory tract. It is also called acute nasopharyngitis. ARTI is characterized by mild coryzal symptoms, rhinorrhea, nasal obstruction, headache, sore throat, myalgia, and sneezing.

INCIDENCE

A. Upper respiratory tract infections are among the most frequent reasons for office visits. However, the true incidence is not known because clients treat themselves with over-the-counter (OTC) and home remedies and there is seasonal and geographic variability. Most adults have two to three colds a year.

B. ARTIs cause 40% of lost workdays.

PATHOGENESIS

A. Over 25% to 80% of ARTIs are caused by a rhinovirus (>100 antigenic serotypes). Other viral agents include coronavirus (10%–20%), respiratory syncytial virus (RSV), adenoviruses (5%), influenza viruses (10%–15%), and parainfluenza viruses. Incubation period is 1 to 5 days, with viral shedding lasting up to 2 weeks.

B. Rhinoviral infections are chiefly limited to the upper respiratory tract but may cause otitis media and sinusitis.

PREDISPOSING FACTORS

A. Exposure to airborne droplets.

B. Direct contact with virus by touching hands or skin of infected people or by touching surfaces they touched and then touching eyes or nose.

C. Very young or old ages.

D. Smoking, which increases risk by 50%.

E. Crowded conditions such as long-term care facilities and college dormitories.

COMMON COMPLAINTS

A. Low-grade fever.

B. Generalized malaise.

C. Nasal congestion and discharge (initially clear, then yellow and thick).

D. Sneezing.

E. Sore throat or hoarseness.

F. Watery and/or inflamed eyes.

OTHER SIGNS AND SYMPTOMS

A. Headache.

B. Cough.

SUBJECTIVE DATA

A. Elicit the onset, course, and duration of symptoms.

B. Inquire about color and other characteristics of nasal discharge and sputum. **Purulent nasal discharge after 14 days signals bacterial sinusitis.**

C. Inquire about other discomforts and exposure to people with similar symptoms.

D. Review allergens, seasonal problems, and exposure to irritants and smoke.

E. Review history for other respiratory problems, such as asthma, chronic bronchitis, and emphysema.

F. Determine the frequency of similar symptoms, including the last ARTI.

G. Note symptoms that may indicate more significant competing diagnosis (respiratory distress, altered mental status, vomiting, dehydration, rash, stiff neck).

H. Ask about immune compromise.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure (BP). Check pulse oximetry as needed.

B. Inspect:

1. Observe general appearance.

2. Inspect eyes. Note "allergic shiners," tearing, and eyelid swelling.

3. Observe ears, throat, and mouth. Otitis media is indicated by redness and bulging of tympanic membrane, or by membrane perforation with drainage.

4. Inspect nose for nasal redness, swelling, polyps, enlarged turbinates, septal deviation, and foreign bodies.

5. Transilluminate the sinuses.

a. Group A streptococci: tonsillar enlargement, exudates, palatine petechiae.

b. Allergies: "cobblestoned" pharyngeal mucosa.

c. Mononucleosis: About half of clients with mononucleosis develop tonsillar exudates and about one-third develop petechiae at the junction of the hard and soft palate, which is highly suggestive of the disease or coinfection with group A beta-hemolytic strep.

C. Auscultate:

1. Auscultate all lung fields. Note decreased breath sounds, rales, or wheezing.

2. Auscultate heart.

D. Percuss:

1. Percuss sinus cavities and mastoid process of temporal bone to rule out otitis media.

2. Percuss chest for consolidation.

3. Percuss the abdomen for organomegaly.

E. Palpate:

1. Palpate face for sinus tenderness.

2. Examine head and neck for enlarged, tender lymph nodes.

DIAGNOSTIC TESTS

A. Diagnosis may be made from history and physical. Because common cold manifestations are so prevalent, an aggressive workup is rarely necessary.

B. Consider rapid strep test if the client has symptoms, positive exam findings, or recent exposure.

C. Consider throat culture if the client has negative rapid strep and is symptomatic or has positive exam findings.

D. Consider testing for influenza A and B.

E. Chest x-ray is not indicated in the absence of physical findings consistent with pneumonia, with comorbid conditions, or with symptoms greater than 3 weeks.

DIFFERENTIAL DIAGNOSES

A. Upper respiratory infection (URI).
B. Allergic rhinitis.
C. Foreign body.
D. Sinusitis.
E. Influenza.
F. Group A strep pharyngitis.
G. Otitis media.
H. Pneumonia.
I. Acute bronchitis.
J. Immunocompromised if history of excessive ARTIs.
K. Asthma.

PLAN

A. General interventions:

1. Supportive care with rest and fluids.

2. Controlled trials reveal minimal therapeutic benefits of vitamin C for treatment and prevention of colds. Zinc has no proven benefit. Echinacea has not shown any differences in rates of infection or severity of symptoms when compared with placebo. Validation and standardization of herbal products have not been completed.

B. Client teaching: *See Client Teaching Guide for this chapter, "Common Cold."*

C. Pharmaceutical therapy:

1. The American College of Chest Physicians released clinical practice guidelines in 2006 for the management of cough.

2. Antibiotics are ineffective in treating viral infection.

3. Corticosteroids may actually increase viral replication and have no impact on cold symptoms.

4. Topical decongestants for rhinorrhea and nasal congestion:

a. Intranasal ipratropium bromide 0.06% two sprays, each nostril, three to four times daily.

b. Intranasal cromolyn sodium one spray, each nostril, three to four times daily.

c. Pseudoephedrine (Afrin) nasal spray 0.05% two to three sprays per nostril BID or phenylephrine (Neo-Synephrine) nasal spray 0.25% to 1% two to three sprays per nostril Q4H as needed. Limit use to 2 to 3 days maximum to avoid rebound rhinitis.

5. Oral decongestants such as pseudoephedrine (Sudafed) have no proven benefits and the side effects can be problematic in the elderly.

6. Analgesics, such as acetaminophen (Tylenol) and ibuprofen (Advil), may be used for headache relief.

7. Cough suppressants are not recommended as side effects outweigh benefits. Dextromethorphan can be used for cough suppression to improve sleep as needed.

8. Colds have no allergic mechanism, so antihistamines are ineffective. The atropine-like drying effect from antihistamines may exacerbate congestion and obstruct the upper airway by impairing mucus flow.

FOLLOW-UP

A. None recommended unless symptoms persist longer than 7 days from onset or the client develops symptoms of lower respiratory infection.

CONSULTATION/REFERRAL

A. Consult a physician if the client has been reevaluated and given a new treatment plan but still has symptoms.

B. Make a same-day referral to an otolaryngologist if tonsillar abscess is suspected.

INDIVIDUAL CONSIDERATIONS

A. Adolescents:

1. Dextromethorphan may be used as a recreational drug with potential for abuse.

B. Geriatrics:

1. Potential benefits from cold preparations do not outweigh the risks, including sedation and urinary retention.

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COUGH

DEFINITION

A. Coughing is a mechanism that clears the airway of secretions and inhaled particles. The act of coughing has the potential to traumatize the upper airway (e.g., vocal cords). A chronic cough is one that lasts longer than 8 weeks.

B. Because coughing can be an affective behavior, psychological issues must be considered as a cause or effect of coughing.

INCIDENCE

A. Data on the incidence of coughing are not available. However, most healthy people do not cough and the main reason for coughing is airway clearance. A chronic cough is the most common presenting symptom in adults who seek medical treatment in an ambulatory setting. Chronic cough is present in 10% to 20% of adults.

PATHOGENESIS

A. Stimulation of mucosal neural receptors in the nasopharynx, ears, larynx, trachea, and bronchi can produce a cough, as can acute inflammation and/or irritation of the respiratory tract. Cough is a reflex response that is mediated by the medulla but is subject to voluntary control. There is clear evidence that vagal afferent nerves regulate involuntary coughing.

B. Pertussis (whooping cough) is caused by the bacterium *Bordetella pertussis*.

C. The **pathogenic triad of chronic cough** responsible for 92% to 100% of chronic coughs is as follows:

1. Upper airway cough syndrome (UACS), previously referred to as postnasal drip syndrome.

2. Asthma.

3. Gastroesophageal reflux disease (GERD).

PREDISPOSING FACTORS

A. Pharyngeal irritants including postnasal drainage.
B. Foreign-body aspiration.
C. Tuberculosis (TB; persons in prisons and nursing homes and immigrants from endemic areas of TB).
D. Psychogenic factors.
E. Mediastinal or pulmonary masses.
F. Congestive heart failure (CHF).
G. Cystic fibrosis (CF).
H. Congenital malformations.
I. Viral bronchitis.
J. Asthma (sole symptom in 28%).
K. *Mycoplasma* infection.
L. UACS, previously referred to as postnasal drip.
M. Chronic sinusitis.
N. Allergic rhinitis.
O. Environmental irritants.
P. GERD.
Q. Chronic bronchitis.
R. Pulmonary edema.
S. Medications, including angiotensin-converting enzyme (ACE) inhibitors.
T. Impacted cerumen and external otitis.
U. Nonasthmatic eosinophilic bronchitis (NAEB; 13%–33%).
V. Swallowing dysfunction in the elderly.

COMMON COMPLAINTS

A. A common complaint is a cough that interferes with activities of daily living (ADL) and sleeping, leading to a decrease in the client's quality of life (QOL).
B. Pertussis cough is uncontrollable and violent. Following coughing, a "whooping" sound follows with a deep breath.

OTHER SIGNS AND SYMPTOMS

A. Fatigue.
B. Rhinitis.
C. Epistaxis.
D. Tickle in throat.
E. Pharyngitis.
F. Night sweats.
G. Dyspnea.
H. Fever.
I. Sputum production.
J. Hoarseness.

SUBJECTIVE DATA

A. Elicit information about onset, duration, and course of the cough. Was onset recent or gradual? **Does the cough occur at night? Nocturnal cough may be caused by chronic interstitial pulmonary edema and may signal left-sided heart failure. Cough caused by asthma is also worse at night. Morning cough with sputum suggests bronchitis.**
B. Inquire about the cough's characteristics. For example, is it productive, dry, bronchospastic, brassy, wheezy, strong, or weak? If it is productive, is it bloody or mucoid? What is the color, consistency, odor, and amount of sputum or mucus? Dry, irritative cough suggests viral respiratory infection. Severe or changing cough should be evaluated for bronchogenic carcinoma. Rusty-colored sputum suggests bacterial pneumonia. Green or very purulent sputum is due to degeneration of white cells. HIV cough also produces purulent sputum.
C. Inquire whether the cough is associated with eating and choking episodes. Wheezing or stridor with coughing may indicate a foreign body or aspiration.
D. Ask whether the cough is associated with postnasal drip, which produces a chronic cough, clear sputum, edematous nasal mucosa, and a "cobblestoned" pharyngeal mucosa.
E. Find out if the cough is associated with heartburn or a sour taste in the mouth, indicating GERD.
F. Ask about precipitating factors, such as exercise, cold air, or laughing. Also ask about alleviating factors. Cough from asthma can be triggered or exacerbated by exposure to environmental irritants, allergens, cold, or exercise.
G. Ask about current and previous work. Is the client exposed to occupational and environmental irritants, such as dust, fumes, or gases? If so, what are the type, level, and duration of exposures?
H. Ask about family history of respiratory illness, such as CF or asthma.
I. Is the client a smoker? If so, how much do they smoke and how long have they smoked? Are they interested in cessation? Are they exposed to secondhand smoke? How much of the day? **Smoking is the main cause of chronic cough**.
J. Find out the date of the client's last tuberculin skin test. Note recent exposure to TB.
K. Inquire about any exposure to the flu.
L. Does the client have a history of heart problems?
M. Does the client have a history of respiratory problems or other medical problems? Chronic bronchitis is a major cause of chronic cough and sputum production. Cough may also be an early sign of lung cancer; in late stages, cough occurs along with weight loss, anorexia, and dyspnea.
N. Review medications such as ACE inhibitors. Cough related to ACE inhibitors usually subsides within 2 weeks, but the median time is up to 26 days.
O. Is there weight loss or worsening dyspnea?
P. Exposure to TB. (Confined living conditions? Incarceration?)
Q. Ask about pain in the back or bony skeleton.
R. Ask of any previous screening for lung cancer (as appropriate) with low-dose CT.

PHYSICAL EXAMINATION

A. Record temperature, blood pressure (BP), respirations, heart rate, and weight. Compare previous changes in weight.
B. Inspect:
  1. Observe general appearance for cyanosis, difficulty breathing, use of axillary muscles, and finger clubbing.
  2. Examine ears, nose, and throat.
C. Auscultate:
  1. Auscultate heart and lungs.
D. Percuss:
  1. Percuss sinus cavities and mastoid process.
  2. Percuss chest and lungs for consolidation.
E. Palpate:
  1. Palpate face for sinus tenderness.
  2. Examine head and neck for lymph nodes, masses, and jugular vein distension (JVD).

DIAGNOSTIC TESTS

A. Testing can be held to a minimum by careful review of history and physical exam.
B. White blood cell (WBC) if infection suspected.
C. HIV test if suspected.
D. Sputum for eosinophils, Gram stain, and/or culture. ▶

E. Mantoux test if indicated.
F. Chest radiograph (CXR) for hemoptysis, dyspnea, fever, chest pain, or weight loss.
G. Pulmonary function testing/spirometry.
H. Methacholine challenge to rule out asthma.
I. Esophageal pH monitoring to rule out GERD.
J. CT with consideration of contrast scan if necessary (the radiologist may need to determine).

DIFFERENTIAL DIAGNOSES

A. Environmental irritants:
  1. Cigarette, cigar, or pipe smoking.
  2. Pollutants (wood smoke, smog, burning leaves, etc.).
  3. Dust.
  4. Lack of humidity.
  5. Chemical exposure in home or workplace, including illicit substances.
B. Lower respiratory tract problems:
  1. Lung cancer.
  2. Asthma.
  3. Chronic obstructive lung disease (includes bronchitis).
  4. Interstitial lung disease.
  5. CHF.
  6. Pneumonitis.
  7. Bronchiectasis.
  8. Foreign body or aspiration.
  9. Tracheal–esophageal fistula.
  10. Pulmonary embolism.
  11. Pulmonary edema.
  12. Pneumonia.
C. Upper respiratory tract problems:
  1. Chronic rhinitis.
  2. Chronic sinusitis.
  3. Disease of external auditory canal.
  4. Pharyngitis.
D. Medication-induced cough from ACE inhibitors.
E. Extrinsic compression lesions:
  1. Adenopathy.
  2. Malignancy.
  3. Aortic aneurysm.
F. Psychogenic factors.
G. Gastrointestinal problems such as reflux esophagitis.
H. Genetic problems such as CF.

PLAN

A. General interventions:
  1. If sputum is purulent, obtain a sample for examination.
  2. Clients with chronic obstructive pulmonary disease (COPD) and CF should be taught huffing as an adjunct to other methods of sputum clearance.
B. Client education: *See Client Teaching Guide for this chapter, "Cough."*
C. Pharmaceutical therapy:
  1. The risk of cough suppressants outweighs the benefits of use.
  2. Antibiotics should not be prescribed for coughs unless a bacterial infection is suspected.
  3. Therapy depends on various acute inflammatory and chronic irritating processes and on cause of cough. Refer to applicable sections of this chapter, such as "Asthma" and "Tuberculosis," and see the "Gastroesophageal Reflux Disease and Dyspepsia" section of Chapter 14.
  4. Inhaled or oral antihistamines could be considered if cough is related to allergic rhinitis. Potential side effects versus benefits should be considered, especially in the elderly.

FOLLOW-UP

A. The client with a normal CXR and no risk factors for lung cancer (e.g., smoking or occupational exposure) can be followed expectantly without further testing.
B. In clients whose cough resolves after cessation of ACE inhibitors and for whom there is a compelling reason to treat with these agents, substitution with an angiotensin II receptor blocker (ARB) is recommended.
C. See applicable sections for specific diagnoses.
D. Pertussis vaccination booster should be administered to all adults according to the Centers for Disease Control and Prevention (CDC) adult vaccine guidelines. Pertussis cases should be reported to the local health department.

CONSULTATION/REFERRAL

A. Consult a physician if symptoms persist after treatment. Reevaluate client in 2 weeks if they are no better.
B. When a cough lasts more than 2 weeks without another apparent cause and is accompanied by paroxysms of coughing, posttussive vomiting, and/or an inspiratory whooping sound, the diagnosis of a *B. pertussis* infection should be made unless another diagnosis is proven.
C. Clients whose condition remains undiagnosed after a workup and therapy may need referral to a cough specialist.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
  1. Cough may be an early symptom of pulmonary edema. Watch intrapartum clients for signs of edema.
B. Adolescents:
  1. Dextromethorphan has been associated with abuse potential.
C. Geriatrics:
  1. The risk of side effects from over-the-counter (OTC) cough suppressants should be discussed with the client.
  2. Elderly clients with dementia are generally unable to perform the forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV1) diagnostic tests. In the elderly with dementia, consider COPD in older adults who have any of the following factors. The greater the number of factors, the more likely COPD is present:
    a. Dyspnea that is progressive or worse with exercise.
    b. Chronic cough, either productive or nonproductive.
    c. History of chronic exposure to tobacco smoke, smoke from heating fuels, dust, or chemicals.
    d. Family history of COPD.

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DYSPNEA

DEFINITION

A. Dyspnea occurs when a client experiences any level of breathing discomfort. The symptom is often associated with pulmonary disorders and cardiac ischemia and is a significant predictor of mortality. The risk of experiencing dyspnea increases with age. Up to 37% of people older than the age of 70 experience dyspnea. Treatment is directed at identifying and managing the underlying pathologic concern.

INCIDENCE

A. Dyspnea affects up to half of clients seen in hospitals and up to one-fourth of clients seeking outpatient care.
B. Millions of clients living at home experience chronic dyspnea related to pulmonary and cardiac morbidities.
C. Between three and four million ED visits annually are due to some level of dyspnea.
D. Dyspnea is associated with cardiovascular disease (the leading cause of death in the United States) and pulmonary disease (asthma and chronic obstructive pulmonary disease [COPD], affecting more than 25 million).

PATHOGENESIS

A. Respiration is controlled by peripheral chemoreceptors in the carotid bodies and aortic arch the central chemoreceptors in the medulla.
B. Chemoreceptors are stimulated by a drop in partial pressure of oxygen in arterial blood, a drop in pH, and a change in pCO_2 levels.
C. Additional receptors play a role in maintaining oxygenation, including mechanoreceptors in the upper airway, pulmonary receptors, and chest wall receptors.
D. Breathing discomfort occurs when a client recognizes hypoxia and hypercapnia. Dyspnea is experienced due to physical, psychological, social, and environmental factors.
E. For adequate gas exchange to occur, oxygen must reach the alveoli from the environment. If any part of the airway is compromised, including the alveolar beds, the process is dysfunctional to some degree.
F. In addition to intact respiratory receptors and a clear airway for gas exchange, the act of breathing also requires muscular strength and adequate hemoglobin levels to transport oxygen.

PREDISPOSING FACTORS

A. Asthma.
B. COPD.
C. Interstitial lung disease.
D. Cardiovascular disease.
E. Deconditioning/obesity.
F. Severe anemia.
G. Foreign body.

COMMON COMPLAINTS

A. Difficulty breathing or the sensation of "air hunger."
B. Change in respiratory pattern.
C. Potential wheezing.
D. Weakness.
E. Fatigue.
F. Functional disability.

OTHER SIGNS AND SYMPTOMS

A. Chest tightness.
B. Rapid or shallow breathing.
C. Breathing feels "heavy."
D. Chronic dyspnea is associated with depression.
E. Severe, acute dyspnea is associated with a sensation of impending death.
F. Headache.
G. Anorexia.

POTENTIAL COMPLICATIONS

A. Hypoxemia.
B. pH imbalance.
C. Cardiac dysrhythmias.
D. Confusion.

SUBJECTIVE DATA

A. Determine the onset, duration, and course of dyspnea.
B. Determine if any short-acting beta-2 agonist (SABA; rescue) inhaler was used, the name, quantity used, effectiveness, and when it was last used.
C. Establish medical history, including pulmonary, cardiac, neuromuscular, or hematologic impairments.
D. Determine smoking history, including inhalation of illicit substances (methamphetamines).
E. Rate the degree of dyspnea on a dyspnea scale.
F. Ask the client to list all medications currently being taken, including oral or aerosolized herbal products.
G. Ask about exposures to caustic substances, including fumes, smoke, carbon monoxide in the home or workplace, and occupational exposures to inhaled toxins.
H. Is there associated cough or wheezing?
I. Ask of any pain in chest wall, mental confusion, or extreme fatigue.
J. Determine if there is a history of anxiety or panic episodes.
K. Ask about use of emergency services or need for hospitalization due to dyspnea.
L. Consider if the client has established advance directives and made any change from previous directives.

PHYSICAL EXAMINATION

A. Check temperature, blood pressure (BP) pulse, pulse oximetry, level of consciousness (LOC), and ability to speak in complete sentences without obvious air hunger.
B. Inspect:
  1. Inspect general appearance, noting degree of dyspnea and the respiratory pattern.
  2. Check trachea for deviations.
  3. Inspect for use of accessory muscles of breathing.
  4. Inspect skin for pallor, cyanosis, bruising, open wounds, petechiae, and turgor.
  5. Inspect facial features for fear, lethargy, or angioedema.
  6. Inspect legs for edema.
  7. Capillary refill.
C. Palpate:
  1. Palpate chest wall for tenderness/injury.
  2. Palpate radial and peripheral pulses for amplitude and rhythm.
  3. Palpate chest wall for crepitus.
D. Auscultate:
  1. Cardiac for rhythm, S3/S4.
  2. Lungs in all fields for adventitious sounds.
  3. Abdomen for distension.

DIAGNOSTIC TESTS

A. EKG shows inverted T waves, ST-segment elevation, and Q waves. One normal EKG initially does not always rule out myocardial infarction (MI); perform serial EKGs if MI suspected.
B. Arterial blood gas (ABG).
C. Chest x-ray.
D. Complete blood count (CBC) with differential.
E. Comprehensive metabolic panel (glucose, electrolytes, renal and hepatic functions).
F. Brain natriuretic peptide (BNP).
G. Thyroid-stimulating hormone (TSH).
H. Pulmonary function tests (PFTs).

DIFFERENTIAL DIAGNOSES

A. Acute MI.
B. Pulmonary embolus.

C. Aortic dissection.
D. Pneumothorax.
E. Poisoning (secondary to inhaled elicit substances or unintentional: carbon monoxide).
F. Asthma, uncontrolled.
G. COPD exacerbation.
H. Pulmonary fibrosis.
I. Pulmonary edema.
J. Neuromuscular dysfunction.
K. Respiratory suppression secondary to medication overdose.
L. Anemia.
M. Anxiety/panic episode.
N. Developing pregnancy with gravid uterus.
O. End-stage heart failure.
P. Pulmonary hypertension.
Q. Lung cancer.
R. Progression of morbid obesity and deconditioning.
S. Aspiration or foreign body.
T. Allergic response/angioedema.

PLAN

A. General interventions:
1. The treatment of dyspnea is determined by the underlying etiology.
2. If coronary heart disease (CHD) is present:
a. Make sure client is aware of signs and symptoms of MI.
b. Make sure client knows to seek medical attention or dial 911 if signs and symptoms occur.
c. Encourage CPR training for the family and/or close friends.
B. Client teaching:
1. Educate the client about modifying controllable risk factors for pulmonary and cardiovascular disease. Smoking cessation should be addressed at each client encounter.
2. Discuss the benefits of cardiovascular exercise.
3. Client should carry fast-acting nitroglycerin at all times and know how to use it.
C. Dietary management:
1. If applicable, counsel client on benefits of weight loss to comfort of breathing.
2. Encourage well-balanced meals and adequate fluid intake.
D. Pharmaceutical therapy:
1. Oxygen should be delivered based on client status. For conscious clients in outpatient settings, oxygen is most effectively delivered through a nonrebreather mask. The hypoxic drive is not a consideration for limiting oxygenation in acute emergencies.
2. When MI is suspected, give aspirin 160 to 325 mg (four 81-mg baby aspirin) chewed or swallowed as soon as possible. Enteric-coated aspirin delays absorption and therefore is not recommended.
3. For bronchodilation, a SABA should be delivered by inhaler or nebulization.
4. Further intervention must be under the direction of a specialist after a full evaluation.

FOLLOW-UP

A. Follow-up is determined by the client's condition and primary diagnosis. Timely follow-up is essential and is based on individual client considerations. In the elderly who live alone, next-day follow-up should be considered if the client will not be hospitalized.

EMERGENT ISSUES/INSTRUCTIONS

A. If you suspect MI as the cause of dyspnea, refer the client through emergency medical service (EMS) for immediate ED evaluation.
B. For suspected pulmonary emboli or dissecting aneurysms, client should be transported by EMS.

CONSULTATION/REFERRAL

A. Consultation for chronic dyspnea with a pulmonologist if lung disorder.
B. Consultation with appropriate specialist if cardiac, neuromuscular, or hematologic disorder.
C. Referral to pulmonary and/or cardiac rehabilitation services.
D. Referral to occupational therapy for functional rehabilitation

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. For optimal oxygenation, clients with a gravid uterus should be placed on their left side while they are receiving supplemental oxygen.
2. Obstetric referral is recommended for significant dyspnea during pregnancy.
3. Control of asthma or any chronic lung disorder is crucial as the mother must maintain adequate oxygenation to sustain the fetus.
B. Geriatrics:
1. Frailty is a risk marker of morbidity and mortality for all causes of dyspnea. The differential list for dyspnea is lengthy and covers multiple systems. A thorough review of systems and physical exam is required to differentiate the final diagnosis.
2. Adult vaccination guidelines from the Centers for Disease Control and Prevention should be followed for prevention; these are available at www.cdc.gov/vaccines/schedules/downloads/adult/adult-combined-schedule.pdf.
3. The primary care provider should be aware of the client's choices in worsening dyspnea. Advance directives should be available on the outpatient medical record.
4. Consideration for hospice or palliative care services should be recommended to support the client and the family early in the course of progressive dyspnea and chronic illness.

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EMPHYSEMA

DEFINITION

A. Emphysema is an abnormal dilation and destruction of alveolar ducts and airspaces distal to the terminal bronchioles. Lung function slowly deteriorates over many years before the illness develops. Emphysema is one of the chronic obstructive pulmonary diseases (COPDs)—a term that refers to conditions characterized by continued increased resistance to expiratory airflow. Chronic bronchitis, emphysema, and asthma comprise COPD. Chronic bronchitis and emphysema with airflow obstruction commonly occur together. ▶

B. There are three morphologic types of emphysema:
1. Centriacinar emphysema: associated with long-term smoking; primarily involves the upper half of the lungs.
2. Panacinar emphysema: found predominantly in the lower half of the lungs; this is observed in clients with alpha-1 antitrypsin (AAT) deficiency.
3. Paraseptal emphysema: involves the distal airway.

INCIDENCE

A. Emphysema typically occurs in people older than age 50, with peak occurrence after the age of 65.
B. The prevalence of emphysema is 20.2 cases per 1,000.
C. The prevalence is greater in Caucasians than other ethnicities.
D. Emphysema is slightly greater in females (21.4/1,000) than in males (19.0/1,000).

PATHOGENESIS

A. Decreased gas exchange occurs due to focal destruction limited to the airspaces distal to the respiratory bronchioles, causing airway obstruction, hyperinflation, loss of lung recoil, and destruction of the alveolar–capillary interface.

PREDISPOSING FACTORS

A. Long-term cigarette smoking.
B. Occupational and environmental exposure to toxic agents:
1. Dust.
2. Chemical fumes.
3. Secondhand smoke.
4. Air pollution.
5. Gases.
C. Alpha-1 protease inhibitor deficiency.
D. Intravenous (IV) drug use secondary to pulmonary vascular damage from the insoluble fillers (e.g., cornstarch, cotton fibers, cellulose, talc).
E. Connective tissue disorders (e.g., Marfan syndrome and Ehlers–Danlos).
F. HIV.

COMMON COMPLAINTS

A. Gradually progressing exertional dyspnea.
B. Chronic cough.
C. Wheezing.
D. Fatigue.
E. Weight loss.

OTHER SIGNS AND SYMPTOMS

A. Cough with mild to moderate sputum production and clear to mucoid sputum.
B. Early morning cough.
C. Shortness of breath (SOB).
D. Tachypnea.
E. Use of accessory muscles for breathing, pursed-lip breathing, *prolonged* expiration.
F. Barrel chest (increased anterior to posterior chest diameter).
G. Flushed skin.
H. Clubbed fingers.
I. Decreased libido.
J. Thin, wasted appearance.
K. Wheezing, particularly during exertion and exacerbations of emphysema.
L. Functional impairment related to dyspnea.
M. Depression.

SUBJECTIVE DATA

A. Elicit information about onset, duration, and course of symptoms.
B. Determine if the client is a smoker. Determine cigar use and cigarette pack-year history (pack/day times the number of years smoked). If so, how much and for how long? Evaluate exposure to secondhand smoke. How much of the day? Are they interested in quitting?
C. Ask about current and previous work. Is the client exposed to occupational and environmental irritants, such as dust, fumes, or gases? If so, what are the type, level, and duration of exposures?
D. Inquire about the cough's characteristics. Is it productive, dry, bronchospastic, brassy, wheezy, strong, or weak?
E. Question the client about episodes of tachypnea, frequency of respiratory infections, and incidence of angina during exertion.
F. Does the client have a hereditary disease (e.g., cystic fibrosis [CF] or AAT deficiency), asthma, nasal abnormalities (e.g., deviated septum), or other respiratory problems?
G. Determine the last tuberculin skin test and recent risk for tuberculosis (TB) exposure.
H. Find out the client's usual weight and assess how much weight loss has occurred and over what time period.
I. Evaluate current vaccination status for pneumonia and influenza.
J. Review all medications, including over-the-counter (OTC) and herbal products.
K. Assess the client's ability to perform activities of daily living (ADL) and instrumental activities of daily living (IADL), including grooming and personal hygiene, performing chores around the home, shopping, cooking, and driving.
L. Ask about alcohol use.
M. Screen for depression.
N. Ask about frequency of use of short-acting beta-2 agonists (SABA) and effectiveness.
O. Inquire about use of ED or need for hospitalization. Previous ventilation?
P. Assess severity of symptoms using the modified Medical Research Council Dyspnea Scale and the COPD Assessment Test.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure (BP), and weight for changes. Consider pulse oximetry.
B. Inspect:
1. Observe general appearance; note flushed skin color, use of accessory muscles, pallor around lips, pursed-lip breathing, barrel chest (lung hyperinflation), and thinness.
2. Assess for peripheral edema.
3. Perform dermal examination: Note finger clubbing and cyanosis.
4. Observe mental status and psychiatric concerns, including depression.
C. Auscultate:
1. Auscultate heart.
2. Auscultate lungs for wheezes, crackles, and decreased breath sounds (generally diffuse decreased breath sound).
3. Assess for vocal fremitus (vibration) and egophony (increased resonance and high-pitch bleating quality). Air trapping causes air pockets that do not transmit sound well.
D. Percuss:
1. Percuss chest for presence of hyperresonance and signs of consolidation.
2. Percuss abdomen for organomegaly.

E. Palpate:
1. Palpate the neck and the superior and inferior clavicular spaces for lymphadenopathy.
2. Palpate abdomen for organomegaly.
3. Grade dependent edema.
F. Further physical examinations are dependent on comorbidities.
G. Mental status:
1. Assess for decreased level of consciousness (LOC).

DIAGNOSTIC TESTS

A. Pulse oximetry: blood gases if indicated. Baseline should be considered as early in disease process as client presents.
B. AAT to rule out hereditary deficiency.
C. Tuberculin skin test.
D. Pulmonary function tests (PFTs) reveal increased total lung capacity with poor respiratory expulsion and increased respiratory volume.
E. EKG reveals sinus or supraventricular tachycardia.
F. Chest radiograph (CXR) reveals hyperinflation, flat diaphragm, and enlarged heart.
G. Sputum evaluation and/or culture.

DIFFERENTIAL DIAGNOSES

A. Emphysema.
B. Chronic bronchitis.
C. Chronic asthma.
D. Bronchiectasis.
E. CF.
F. Chronic asthmatic bronchitis.
G. TB.
H. AAT deficiency.
I. Congestive heart failure (CHF).

PLAN

A. General interventions:
1. Medical management: Supplemental oxygen therapy is indicated if the client has a resting PaO2 less than 55 mmHg or a PaO2 less than 60 mmHg, along with right heart failure or secondary polycythemia. The goal is to achieve a PaO2 of greater than 55 mmHg (usually 1–3 L/min).
B. Client teaching: *See Client Teaching Guide for this chapter, "Emphysema."*
1. Develop a smoking cessation plan: Assess readiness to quit. Set a quit date and encourage a group smoking cessation program. A smoking cessation plan is an essential part of a comprehensive treatment plan.
2. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines note a five-step program for smoking intervention:
a. ASK: Systematically identify all tobacco users at every visit; *EVERY* client at *EVERY* clinic visit is queried about tobacco-use status.
b. ADVISE: Strongly urge all tobacco users to quit in a clear, strong, and personalized manner.
c. ASSESS: Determine willingness and the rationale of the client's desire to make a quit attempt. Ask every tobacco user if they are willing to make a quit attempt at this time (e.g., within the next 30 days).
d. ASSIST: Aid in quitting, make a quit plan, provide practical counseling, provide educational materials, and help the client obtain social support. Pharmacotherapy if appropriate.
e. ARRANGE: Schedule a follow-up contact either in person or by telephone.
C. Pharmaceutical therapy:
1. Prescription medication and nicotine replacement reliably increase long-term smoking abstinence rates. **The effectiveness and safety of e-cigarettes as a smoking cessation aid are uncertain at this time.**
2. *See Client Teaching Guide for Chapter 25, "Nicotine Dependence."*
3. Nicotine chewing gum produces better quit rates than counseling alone.
4. Transdermal nicotine patches have a long-term success rate of 22% to 42%.
5. The use of vapor nicotine products should be discussed as their long-term adverse effects on lung function are unknown.
6. The use of an antidepressant such as bupropion sustained-release (Zyban) has been shown to be effective in smoking cessation by reducing the enjoyment of smoking, reducing withdrawal symptoms, and limiting weight gain. Bupropion may be used in combination with nicotine replacement therapy (NRT). Begin dosing 1 week before quit date.
a. Days 1 to 3 of treatment: 150 mg every morning.
b. Day 3 until the end of treatment: 150 mg BID, at least 8 hours apart.
c. Duration of treatment should be for 7 to 12 weeks. If the client has not quit smoking after 7 to 12 weeks, it is unlikely that they will quit during that attempt so treatment should be discontinued and the treatment plan reassessed.
d. Medication doses and/or frequency intervals are required for renal and mild hepatitis impairment.
e. Moderate to severe hepatic impairment: 150 mg every other day.
7. Varenicline (Chantix) is a partial agonist selective for alpha-4, beta-2 nicotinic acetylcholine receptors. Varenicline mimics the effect of nicotine; therefore, it reduces the urge to smoke and relieves withdrawal symptoms. It blocks or blunts the effect of nicotine if the person relapses and has a cigarette. Review the client's history of renal impairment, psychiatric disorders, seizure history/risk, alcohol use, and cardiovascular disease. (Chantix is currently off the market due to high levels of nitrosamine, which is said to be carcinogenic.)
8. Oral and inhaled medications are used for clients with stable emphysema to reduce dyspnea and improve exercise tolerance. GOLD guidelines recommend medications based on the results of symptom severity assessments. **Prior to any medication/dosage changes, monitor the client's compliance (see Table 12.1). A spacer/chamber device should be used to improve drug delivery and reduce adverse effects.**
a. Bronchodilators decrease muscle tone in both the small and large airways thereby, increasing ventilation. This category includes beta-adrenergic agents, methylxanthines, and anticholinergics.
i. Albuterol (Proventil, Ventolin), a beta-2 agonist, relaxes the smooth muscle with little effect on cardiac muscle contractility. Albuterol is available as a liquid for nebulizers, and as a metered-dose inhaler (MDI) and dry powder inhaler (DPI).
ii. Metaproterenol (Alupent), a beta-2 agonist, relaxes the smooth muscle with little effect on cardiac muscle contractility. Metaproterenol is available as liquid for nebulizer, MDI, and DPI.

iii. Levalbuterol (Xopenex) is a selective beta-2 agonist that is also used for treatment or prevention of bronchospasm.

iv. Ipratropium (Atrovent) is chemically related to atropine with antisecretory properties. It is used on a fixed schedule with a beta-agonist.

v. Salmeterol (Serevent) relaxes the smooth muscles and can relieve bronchospasm. It may also facilitate expectoration. The bronchodilating effect lasts longer than 12 hours. It is used on a fixed schedule in addition to regular use of anticholinergic agents.

vi. Formoterol (Foradil) is a beta-agonist with rapid onset and long duration. The bronchodilating effects last longer than 12 hours. Formoterol is used on a fixed schedule in addition to regular use of anticholinergic agents.

vii. Tiotropium (Spiriva) is a quaternary ammonium compound that elicits anticholinergic/antimuscarinics effects with inhibitory effects on M3 receptors on airway smooth muscles, leading to bronchodilation. It is available as a capsule dosage form containing dry powder for oral inhalation via HandiHaler.

viii. Theophylline (aminophylline, Theo-24, Theo-Dur, Slo-Bid) potentiates exogenous catecholamines and diaphragmatic muscular relaxation, which stimulates bronchodilation. Theophylline has a narrow therapeutic range and frequent toxicity.

The target theophylline concentration is 5 to 10 µg/mL. Prescribing theophylline requires weight-based dosing. Dosage adjustment therapy is based on serum theophylline concentrations. Monitor theophylline level whenever there is a new worsening chronic illness, or a change in medications that may alter theophylline clearance (e.g., fever >102°F sustained for ≥24 hours or altered liver function including hepatitis). Check drug interaction using an online checker: www.rxlist.com/drug-interaction-checker.htm.

1) Dosage should be titrated up. Titration steps include the following: the starting dose; after 3 days, *if tolerated*, increase dose; and after 3 more days, *if tolerated and if needed*, increase dose.

2) Common side effects of theophylline include loss of appetite, abdominal pain, diarrhea, weight loss, restlessness, tremor, insomnia, headache, lightheadedness, dizziness, and sweating.

3) Instruct the client to call 911 and get emergency medical help for severe headache, hyperglycemia, severe or persistent vomiting, cardiac arrhythmias, seizures, or confusion.

a) Aclidinium (Tudorza Pressair) is an M1 to M3 muscarinic agonist (long-acting muscarinic agonists [LAMAs]). It inhibits M3 receptors, leading to muscle relaxation and subsequent bronchodilation.

b. Corticosteroids attempt to moderate the inflammatory component. They should only be added to a regimen that includes a long-acting bronchodilator:

i. Fluticasone inhaled: vasoconstrictive and anti-inflammatory activity.

ii. Budesonide inhaled.

c. Phosphodiesterase 4 (PDE4) inhibitors: Selective PDE4 inhibitors reduce exacerbations, improve dyspnea, and increase lung function in clients with severe COPD.

d. Oral steroids should be used to treat outpatients with acute exacerbations. Corticosteroids reduce mucosal edema, inhibit prostaglandins that cause bronchoconstriction, and increase responsiveness to bronchodilators. Taper dose as soon as bronchospasm is controlled. A minority of clients who respond to oral steroids can be maintained on long-term inhaled steroids.

e. In clients with emphysema, chronic infection or colonization of the lower airways is common. The goal of antibiotic therapy is not to eliminate the organisms, but to treat acute exacerbations. If infection is present, give one of the following:

i. Macrolides (azithromycin) are preferred for clients with COPD exacerbation.

ii. Augmentin 500 mg orally BID.

iii. Doxycycline 100 mg orally BID.

iv. Trimethoprim-sulfamethoxazole 80 to 160 mg orally Q12H.

v. If *pseudomonas* is considered, levofloxacin 500 mg daily.

f. Mucolytic agents in clinical practice are not recommended currently due to a lack of evidence on their benefit.

g. Trivalent influenza vaccine is essential for all COPD clients.

Administer yearly flu vaccine. High-dose trivalent or quadrivalent influenza vaccine is essential for all COPD clients. Those 65 years of age or older should receive high-dose trivalent vaccine. Give the client the vaccine in late September, at least 6 weeks before the onset of flu season.

h. Administer pneumonia vaccine in clients 65 years and older, or beginning at age 19 if immunocompromised or chronic respiratory condition is present. Consider revaccination every 5 to 10 years in high-risk clients. Download a guide to sequential administration according to individual health considerations at www.cdc.gov/vaccines/vpd/pneumo/downloads/pneumo-vaccine-timing.pdf.

i. Sequential administration of the pneumococcal conjugate vaccine (PCV13) and pneumococcal polysaccharide vaccine (PPSV23) is recommended (download the Centers for Disease Control and Prevention's [CDC] guide at www.cdc.gov/vaccines/vpd/pneumo/downloads/pneumo-vaccine-timing.pdf). In the vaccine-naive client, give PCV13 first, then the PPSV23 12 months later.

ii. In older adults who received PPSV23 at age 65 or older, give PCV13 in 1 year or more.

iii. In older adults who received PPSV23 BEFORE the age of 65, give PCV13, then 5 years after the initial PPSV23 give a second PPSV23.

iv. Give a onetime second dose of PPSV23 in persons with chronic renal failure or nephrotic syndrome, functional or anatomic asplenia, or immunocompromising conditions.

i. AAT is needed for significant antitrypsin deficiency (<80 mg/dL). Clients get weekly or monthly infusions. Consult with a physician before therapy. A history of smoking rules out candidacy. ▶

FOLLOW-UP

A. If the client is acute, contact by phone in 24 to 48 hours and consider immediate referral.

B. Monitor the client's body weight.

C. Serial PFTs may help guide therapy and offer prognostic information.

D. Monitor theophylline levels due to the drug's potential for toxicity. Adverse effects, including nausea and nervousness, are the most common. Other adverse effects include abdominal pain with cramps, anorexia, tremors, insomnia, cardiac arrhythmia, and seizures. Theophylline doses: 100 to 200 mg every 6 to 8 hours.

EMERGENT ISSUES/INSTRUCTIONS

A. Monitor theophylline levels due to the drug's potential for toxicity.

1. Instruct the client to call 911 and get emergency medical help for severe headache, hyperglycemia, severe or persistent vomiting, cardiac arrhythmias, seizures, and confusion.

CONSULTATION/REFERRAL

A. If the client's condition remains acute after 48 hours of treatment, consider immediate referral to a physician.

B. Refer the client to a social worker for help in getting Meals on Wheels, handicapped parking, and finding other community resources.

C. A consultation with a pulmonary specialist is recommended.

D. Pulmonary rehabilitation should be considered early in the disease process. Pulmonary rehab programs vary in format and length. Most programs use a small group format and include the following:

1. Education about symptoms, medications, and oxygen.
2. Supervised exercise classes and instruction.
3. Breathing techniques.
4. Nutritional counseling.
5. Emotional health support.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. Sexual dysfunction is common in clients with COPD.

B. Geriatrics:

1. Older adults are at higher risk for restrictive lung diseases. Therapy follows the underlying cause. These diseases fall into four broad categories:

a. Intrinsic: idiopathic pulmonary fibrosis, radiation-induced, drug-induced, postinflammatory lung fibrosis, connective tissue disease.
b. Extrinsic: kyphosis/kyphoscoliosis and obesity.
c. Neuromuscular: amyotrophic lateral sclerosis (ALS), thyroid disorders, adrenal disorders, postpolio syndrome, vitamin D deficiency.
d. Central nervous system disorders: Parkinson disease, multisystemic atrophy, multiple sclerosis, progressive supranuclear palsy.

2. Discuss course of disease, living wills, advance directives, and resuscitation status early, before a crisis occurs. Encourage the client to discuss their preferences with their family.

3. Older adults may be unable to use standard inhalers for the following reasons:

a. Decreased manual dexterity due to arthritis, reduced grip strength, tremors, muscle weakness, and poor hand–eye coordination.
b. Cognitive impairment: difficulty coordinating the required activation/inhalation steps to achieve proper penetration of medication into the lungs.
c. Clients should demonstrate use of their inhaler. If concerns, nebulizer therapy should be prescribed to improve the benefit of pharmacotherapy.

4. Beers list caution:

a. **Theophylline**: theophylline is on the Beers list of drugs to use with caution in the geriatric population related to cardiovascular, renal, hepatic, insomnia, and peptic ulcers.
b. Theophylline has a drug–drug interaction with multiple medications, increasing the risk of theophylline toxicity. For example, theophylline has a drug–drug interaction with cimetidine (refer to Appendix C [Beers Criteria], Table C.4.)

RESOURCES

A Patient's Guide to Aerosol Drug Delivery is available at www.copd-alert.com/AerosolDrug.pdf

Drug interaction checker: www.rxlist.com/drug-interaction-checker.htm

Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines: www.goldcopd.org

National Emphysema Foundation: www.emphysema foundation.org

Pulmonary Disease Aerosol Delivery Devices: A Guide for Physicians, Nurses, Pharmacists, and Other Health Care Professionals is available at www.aarc.org/wp-content/uploads/2018/03/aersol-guides-for-hcp.pdf

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INFLUENZA (FLU)

DEFINITION

A. Influenza is a common, acute, viral infection that is a self-limiting, febrile illness of the respiratory tract. Illness is spread person-to-person, primarily by respiratory secretions that can be spread from infected persons through sneezing, coughing, talking, and self-inoculation of secretions through direct contact routes. Influenza is one of the top 10 causes of death in the United States when it occurs with pneumonia.

INCIDENCE

A. Epidemics occur yearly, primarily in the winter months, in both the northern and southern hemispheres. Travelers should be reminded that the flu season is different by hemisphere and can occur on cruise ships. Attack rates may be as high as 10% to 20% of the population. Mortality is highest in the geriatric population older than 65 years of age, except during pandemics when 50% of influenza deaths occur in individuals younger than 65 years of age. Extraordinarily high attacks have occurred in the institutionalized and crowded populations.

PATHOGENESIS

A. Influenza A and B are viruses that have the ability to undergo periodic antigenic changes of their envelope glycoproteins, the hemagglutinin and neuraminidase. Among influenza A viruses that infect humans, there are three major subtypes of hemagglutinins (H1, H2, and H3) and two subtypes of neuraminidases (N1 and N2). Influenza A outbreaks typically start abruptly, peak over 2 to 3 weeks, and last approximately 2 to 3 months. H1N1 (swine flu) is an influenza A virus. ▶

B. The avian flu was an N5N1 and H7N7 viral infection associated with recent exposure to dead or ill poultry. Following exposure, the incubation period for human H5N1 infection was 7 days or less. Clusters of human-to-human transmission of avian flu had a typical incubation period of 3 to 5 days.

C. Influenza B outbreaks are generally less extensive and less severe. Outbreaks associated with the B virus have been reported in schools, military camps, nursing homes, and cruise ships.

D. *Haemophilus influenzae* is a gram-negative coccobacillus. *H. influenzae* is an invasive bacterial disease that can cause meningitis, otitis media, sinusitis, epiglottitis, septic arthritis, occult febrile bacteremia, cellulitis, pneumonia, and empyema; occasionally, this virulent organism causes neonatal meningitis.

E. The incubation period for *H. influenzae* is from between 18 and 72 hours to 5 days after exposure. The exact period of communicability for *H. influenzae* is unknown, but may be for as long as the organism is present in the upper respiratory tract.

PREDISPOSING FACTORS

A. The primary mode of spread is via exposure to viral strain respiratory secretions from respiratory droplets (coughing or sneezing) and direct contact with contaminated surfaces.

1. Adults:
   a. Aged older than 65 years (dependent on the viral strain).
   b. Pregnancy.
   c. High-exposure jobs: teachers, healthcare workers, police, and firefighters.
   d. Recent illnesses or state that has lowered resistance (stress, excessive fatigue, poor nutrition).
   e. Immunosuppression from drugs, illness, or chronic illness (transplant recipients, lung disease, heart disease).
   f. Crowded living conditions, including military camps and institutions such as nursing homes.
   g. Travel in endemic areas.
   h. Avian flu: exposure to dead or ill poultry.
   i. Obesity.

COMMON COMPLAINTS

A. Clinical manifestations of influenza depend on age and previous experience with the influenza virus.

B. Rapid-onset respiratory illness is the most common complaint.

C. Abrupt onset of fever and/or chills.

D. Joint pain.

E. Headache.

F. Conjunctivitis (avian flu).

OTHER SIGNS AND SYMPTOMS

A. Upper respiratory congestion (watery eyes, clear nasal drainage, headache, sore throat, and hoarseness).

B. Malaise or fatigue.

C. Anorexia.

D. Swollen lymph nodes.

E. Nonproductive cough (persisting for weeks).

F. Muscle aches.

G. Gastrointestinal symptoms.

H. Febrile seizures.

I. Otitis media.

SUBJECTIVE DATA

A. Review the onset, course, and duration of symptoms, especially myalgia and malaise.

B. Query the client if they have had a flu shot and when the last flu vaccination was received.

C. Review symptoms of other family members or coworkers who are also ill.

D. Review for recent travel location and use of cruise ships or planes.

E. Evaluate living conditions for exposure risks.

F. Is the client a smoker?

G. Review all medications, including over-the-counter (OTC) and herbal products. Has the client taken any medications for the symptoms?

H. Does the client have a history of asthma or chronic obstructive pulmonary disease (COPD)?

I. Is the client immunocompromised (e.g has HIV, is a transplant recipient, or is on chemotherapy)?

J. What other medical comorbidities, such as diabetes, does the client have?

K. In clients 65 and older, is illness associated with new-onset confusion, which may indicate sepsis?

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure (BP).

B. Inspect:

1. Observe overall appearance for pallor and for any respiratory distress.
2. Assess hydration status of mucous membranes.
3. Conduct an eye, ear, nose, and throat exam.
4. Assess skin for rashes, petechiae, and turgor.

C. Auscultate:

1. Auscultate the lung fields, observing for wheezing and crackles.
2. Auscultate the heart.

D. Palpate:

1. Palpate the neck and lymph nodes: preauricular, posterior auricular, submental and sublingual, anterior cervical chain, and supraclavicular nodes.

E. Perform neurologic exam:

1. Assess the level of consciousness (LOC).
2. Assess for nuchal rigidity.
3. Assess for meningeal signs.
   a. Signs of meningeal irritation include nuchal rigidity.
   b. Positive Brudzinski and Kernig signs (Figures 12.1 and 12.2):
      i. Brudzinski sign: Place the client supine and flex the head upward. Resulting flexion of both hips, knees, and ankles with neck flexion indicates meningeal irritation.
      ii. Kernig sign: Place the client supine. Keeping one leg straight, flex the other hip and knee to a bent knee to form a 90° angle. Slowly extend the lower leg. This places a stretch on the meninges, resulting in pain and spasm for the hamstring muscle. Resistance to further extension can be felt.

DIAGNOSTIC TESTS

A. Usually none required. However, if the client appears ill, consider the following:

1. White blood cell (WBC) and complete blood count (CBC).
2. Viral RNA cultures: Obtain during the first 72 hours of illness because the quality of virus shed subsequently ▶

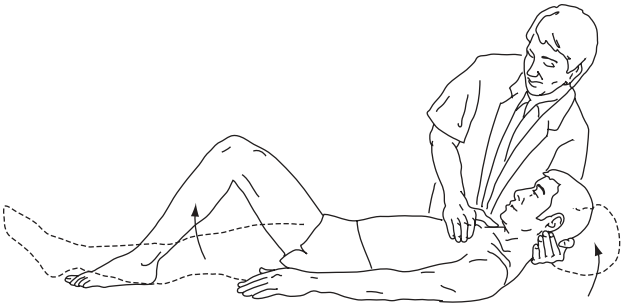


FIGURE 12.1 Brudzinski sign.



FIGURE 12.2 Kernig sign.

decreases rapidly. There is some evidence that a throat sampling yields an improved specimen. Nasopharyngeal secretions obtained by swab or aspirate should be placed in an appropriate transport medium for culture.

3. Rapid antigen test (usually less sensitive in the detection of influenza A than the polymerase chain reaction [PCR]; a negative rapid diagnostic test should be confirmed with a viral culture or other means).

4. Monospot test: **Monospot test is negative with the flu and positive with mononucleosis.**

5. Chest radiograph (CXR; only if pneumonia is suspected).

6. Sputum culture (complications only).

7. Lumbar puncture (complications only).

8. Rapid plasma reagin (RPR) test (for high-risk HIV factors): negative RPR to rule out syphilis.

9. PCR assay (can differentiate between influenza subtypes and offers high sensitivity and specificity but is not readily available for clinical use).

DIFFERENTIAL DIAGNOSES

A. Influenza:
- **1.** Influenza A.
- **2.** Influenza B.
- **3.** Avian flu (H5N1).
- **4.** H1N1 (swine flu).

B. Pneumonia.
C. Bronchitis.
D. Mononucleosis.
E. Early HIV.
F. Severe acute respiratory syndrome (SARS).
G. Meningitis.

PLAN

A. General interventions:

1. Management is usually treatment of symptoms.

2. Encourage flu vaccine for clients in susceptible populations prior to flu season.

3. Clients should expect to have a persistent cough and malaise after the initial acute phase.

B. Client teaching**:** *See Client Teaching Guide for this chapter, "Influenza (Flu)."*

C. Pharmaceutical therapy:

Influenza can alter the metabolism of certain medications, especially theophylline, possibly resulting in the development of toxicity from high serum concentrations.

1. Acetaminophen (Tylenol) as needed for fever.

2. Nonsteroidal anti-inflammatory drugs (NSAIDs) as needed for body aches.

3. Amantadine and rimantadine (class of medications known as adamantanes) are not recommended for antiviral treatment or chemoprophylaxis of currently circulating influenza A virus strains.

4. Antivirals started within the first 48 hours confer the greatest benefit. Antiviral therapy recommendations vary by type of influenza, age group, renal function, and risk factor. To prescribe the most current antiviral therapy, refer to the Centers for Disease Control and Prevention (CDC) website for the most up-to-date recommendations: www.cdc.gov/flu/professionals/antivirals/summary-clinicians.htm.

5. The recommended duration of treatment is 5 days. Longer treatment regimens may be necessary in persons with immunosuppression or for hospitalized clients.

6. Zanamivir (Relenza):
- a. This is not recommended for persons with underlying airway disease, including asthma or COPD.
- b. Zanamivir is for uncomplicated acute illness due to influenza A or B in adults who have been symptomatic for no more than 2 days.
- c. See Table 12.2 for the recommended dosage and schedule of influenza antivirals for treatment and chemoprophylaxis.

7. Oseltamivir (Tamiflu):
- a. For adults and adolescents 13 years of age or older who have been symptomatic for no more than 2 days.
- b. Preferred treatment for pregnant clients. Pregnant clients are recommended to receive the same antiviral dosing as those who are not pregnant.
- c. See Table 12.2 for the recommended dosage and schedule of influenza antivirals for treatment and chemoprophylaxis.

8. Vaccination:
- a. Flu vaccine should not be administered to persons allergic to eggs.
- b. The vaccine should be administered in the autumn prior to the flu season, at least 6 weeks before the onset of the season.
- c. Influenza vaccines may be administered concurrently with other live or inactivated vaccines.
- d. **Immunization is the major means of influenza prevention.** Each year the vaccine is produced with influenza strains. The vaccine may be trivalent or quadrivalent formulations. ▶

TABLE 12.2 RECOMMENDED DOSAGE AND SCHEDULE OF INFLUENZA ANTIVIRAL MEDICATIONS[A] FOR TREATMENT[B] AND CHEMOPROPHYLAXIS[C]

Antiviral Agent		Age Group (Years)	
		13–64	≥65
Zanamivir	Treatment, influenza A and B	10 mg (two inhalations) BID	10 mg (two 5–mg inhalations) BID
	Chemoprophylaxis, influenza A and B	10 mg (two inhalations) once dailyBID	10 mg (two 5-mg inhalations) BID
Oseltamivir	Treatment, influenza A and B	75 mg BID	75 mg BID
	Chemoprophylaxis, influenza A and B	75 mg once daily	75 mg once daily
Peramivir	Treatment, influenza A and B	600 mg IV × one dose	600 mg IV once
	Chemoprophylaxis, influenza A and B	N/A	N/A

[a]Zanamivir is manufactured by GlaxoSmithKline (Relenza inhaled powder). Zanamivir is approved for treatment of persons older than 7 years of age and approved for chemoprophylaxis of persons older than 5 years of age. Zanamivir is administered through oral inhalation by using a plastic device included in the medication package. Clients will benefit from instruction and demonstration of the correct use of the device. Zanamivir is not recommended for persons with underlying airway disease. Oseltamivir is manufactured by Roche Pharmaceuticals (Tamiflu—tablet). Oseltamivir is approved for treatment or chemoprophylaxis of persons 1 year of age or older Oseltamivir is available for oral administration in 30-45- and 75-mg capsules and liquid suspension.

[b]The recommended duration for antiviral treatment is 5 days. Longer treatment courses can be considered for clients who remain severely ill after 5 days of treatment.

[c]The recommended duration is 10 days when administered after a household exposure and 7 days after the most recent known exposure in other situations. For control of outbreaks in long-term care facilities and hospitals, the Centers for Disease Control and Prevention recommends antiviral chemoprophylaxis for a minimum of 2 weeks and up to 1 week after the most recent known case was identified.

IV, intravenously; N/A, not approved.

This information is based on data published by the Food and Drug Administration (FDA), available at www.fda.gov/Drugs/DrugSafety/InformationbyDrugClass/ucm100228.htm.

e. Two types of administration of the vaccine are available:
   i. Inactivated influenza vaccine (IIV), previously called trivalent inactivated vaccine (TIV), is administered intramuscularly.
   ii. Live attenuated influenza vaccine (LAIV) is administered intranasally. The LAIV is available for adults up to 49 years of age.

f. A high-dose influenza vaccine is also available for adults older than 65 years.

g. The recommended site of vaccination in adults is the deltoid muscle.

h. Oseltamivir (Tamiflu) should not be administered with a LAIV within 2 weeks prior to or 48 hours after treatment.

FOLLOW-UP

A. Schedule a follow-up visit within 2 to 5 days if symptoms do not improve:

1. People at highest risk for influenza complications:
   a. All persons 50 years of age and older.
   b. Anyone with chronic pulmonary (including asthma) or cardiovascular (except isolated hypertension), renal, hepatic, neurologic, hematologic, or metabolic disorders, including diabetes mellitus.
   c. Persons who have immunosuppression, including immunosuppression caused by medications or HIV infection.
   d. Persons who are or will be pregnant during the influenza season.
   e. Residents of nursing homes and other long-term care facilities.
   f. American Indians and Alaska Natives.
   g. Persons who are morbidly obese (body mass index [BMI] ≥40).

B. Monitor the client for pulmonary and neurologic complications.

C. Because antiviral resistance patterns can change over time, clinicians should monitor local antiviral resistance surveillance data. Local and state health departments are the points of contact for information about current influenza.

EMERGENT ISSUES/INSTRUCTIONS

A. Clients demonstrating the following should receive same-day ED evaluation:

1. Respiratory compromise.

2. Neurologic changes including obvious central nervous system (CNS) involvement.

3. Profound dehydration or alterations in vital signs.

CONSULTATION/REFERRAL

A. Refer the client to a physician or a neurologist for any complications.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. The American College of Obstetricians and Gynecologists (ACOG) in their 2018 Committee Opinion notes that the flu vaccine is an essential element of preconception, prenatal, and postpartum care.

2. Pregnancy predisposes the client to an increased risk for influenzal pneumonia.

3. Immunization of pregnant persons is considered safe at any stage of pregnancy with the IIV.

4. LAIV, available as an intranasal spray, is not recommended for those who are pregnant but is safe for postpartum clients. ▶

5. The inactivated vaccine is safe for breastfeeding mothers and their infants.

6. Zanamivir is the antiviral of choice for pregnancy dut to its limited systemic absorption.

7. Oseltamivir is used in pregnancy for those with asthma secondary to a higher risk for complications.

B. Adults:

1. Persons with high-exposure occupations such as teachers, healthcare workers, police, and firefighters should consider yearly immunization. Vaccine should be offered/administered to persons with chronic metabolic diseases, renal dysfunction, HIV infection, and immunosuppression.

C. Geriatrics:

1. Ninety percent of flu-related deaths and half of flu-related hospitalization occur in people age 65 and older. Pneumococcal pneumonia and influenza are significant causes of mortality and morbidity in the elderly.

2. Factors that contribute to more severe infections include general frailty, decreased functional reserve, weakened immune systems, decreased lung compliance, and decreased respiratory muscle strength, all due to advancing age.

3. Influenza vaccine is recommended yearly for clients older than the age of 60. The vaccine should be especially encourage in nursing home residents and clients with cardiopulmonary disease.

4. Older adults are at very high risk for complications and death from influenza. Tamiflu (Oseltamivir) is the preferred antiviral therapy.

5. For those with a positive flu test, 75 mg Q12H × 5 days if renal function is known to be normal. For stage 3 chronic renal impairment (creatinine clearance [CrCl] 30–60), give 30 mg Q12H × 5 days. For severe renal impairment (CrCl < 30), 30 mg once daily × 5 days. In end-stage renal disease (ESRD) clients on hemodialysis, give 30 mg immediately, then 30 mg after each hemodialysis cycle for 5 days.

6. For those with exposure to influenza, give prophylaxis: with normal renal function, give 75 mg; daily; for those with CrCl 30 to 60, give 30 mg daily. In hemodialysis, 30 mg immediately, then 30 mg after every other dialysis. For exposure from close contacts within a household, treat for 10 days. In institutional settings, give for 14 days or up to 7 days after the last known case.

7. Geriatric red flags: Geriatric clients with influenza may have worsening of any of the following geriatric syndromes:

a. Weight loss related to anorexia with viral illness.
b. Delirium/confusion due to illness.
c. Falls related to orthostasis with viral illness.

RESOURCE

Centers for Disease and Control and Prevention: www.cdc.gov/flu/professionals

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OBSTRUCTIVE SLEEP APNEA

DEFINITION

A. Obstructive sleep apnea (OSA) is the periodic reduction (hypopnea) or cessation (apnea) of breathing due to a narrowing or occlusion of the upper airway during sleep. OSA has been linked to motor vehicle accidents, cardiac diseases, stroke, diabetes, depression, sexual dysfunction, inattention, poor work performance, and visceral obesity. OSA is also associated with nocturnal cardiac arrhythmias and chronic and acute cardiac events, and is a risk factor for strokes. OSA worsens in the supine sleeping position. Either of the two following conditions is diagnostic of OSA:

1. The presence of 15 or more apneas, hypopneas, or respiratory effort-related arousals per hour of sleep in an asymptomatic client. More than 75% of the apneas and hypopneas must be obstructive.

2. Five or more obstructive apneas, obstructive hypopneas, or respiratory effort-related arousals per hour of sleep in a client with symptoms or signs of disturbed sleep. More than 75% of the apneas or hypopneas must be obstructive.

INCIDENCE

A. The incidence of OSA in the morbidly obese population is between 38% and 88%.

B. Obesity adds a fourfold added risk for disordered breathing.

C. OSA is the most common sleep-related breathing disorder and is increasing in prevalence due to increasing rates of obesity.

D. OSA is more common in males than in females.

PATHOGENESIS

A. Increased tissue thickness of the structures of the tongue and soft tissues in the pharyngeal cavity that decreases the passageway for air to the trachea is thought to be the mechanism of OSA. At night, the muscles of the oropharynx relax , resulting in the relative obstruction of the airway. Obesity and hypertrophy of the tonsils and/or adenoids play a major role in OSA. Clients with severe OSA left untreated have a two- to threefold risk for all-cause mortalities.

PREDISPOSING FACTORS

A. Obesity.
B. Increased neck circumference.
C. Age: increases with age and plateaus in the sixth decade.
D. Sex: male.
E. Postmenopausal females.
F. Hypothyroidism.
G. Tonsillar hypertrophy.
H. Alcohol.
I. Craniofacial abnormalities.
J. Medications:

1. Benzodiazepines.
2. Antipsychotics.
3. Opioid analgesics.
4. Beta-blockers.
5. Barbiturates.
6. Antihistamines.
7. Sedative antidepressants.

K. Allergic rhinitis.

L. Genetic conditions (e.g., Down syndrome, Pierre Robin anomalies, and Marfan syndrome).

▶

M. Ethnic (e.g., African American, Asian, Hispanic, American Indians, Pacific Islanders).
N. Acromegaly.
O. Smoking.
P. End-stage renal disease.
Q. Pregnancy.
R. Congestive heart failure (CHF).
S. Chronic obstructive pulmonary disease (COPD).

COMMON COMPLAINTS

A. Daytime sleepiness.
B. Loud snoring, gasping, or snorting during sleep.
C. Fatigue.
D. Insomnia.

OTHER SIGNS AND SYMPTOMS

A. Adults:
1. Asymptomatic: Clients may not recognize they have OSA because they are able to go to sleep anytime.
2. Restless sleep.
3. Dry mouth or sore throat.
4. Lack of physical or mental energy.
5. Falling asleep when watching television, reading, or driving/riding in a car.
6. Morning headaches.
7. Decreased libido and impotence.
8. Cognitive deficits.
9. Depression.
10. Mood disorders.

SUBJECTIVE DATA

A. Does the client feel sleepy during the day? Is daytime sleepiness a problem?
B. Does the client struggle to stay awake during the day?
C. Does the client take naps? How often and for how long?
D. Does the client feel physically and mentally exhausted?
E. Does the client's bed partner complain about snoring, gasping, choking sensation, or snorting?
F. Ask the Epworth Sleepiness Scale questions related to how often the client dozes off or falls asleep (in contrast to just feeling tired). Each situation is scored from 0 = would never doze, to 1 = a slight chance of dozing, 2 = moderate chance of dozing, and 3 = a high chance of dozing. There are eight situations to which the client responds:
1. Sitting and reading.
2. Watching television.
3. Sitting inactive in a public place (e.g., a theater or a meeting).
4. As a passenger in a car for an hour without a break.
5. Lying down to rest during the day when circumstances permit.
6. Sitting and talking to someone.
7. Sitting quietly after lunch without alcohol.
8. In a car, while stopped for a few minutes in traffic.
G. Ask client to list all medications currently being taken, particularly substances not prescribed, including over-the-counter (OTC) and herbal products.
H. Review alcohol use.
I. Males who present with sleep disorders should also be questioned about the presence of erectile dysfunction.

PHYSICAL EXAMINATION

A. Check blood pressure, pulse, respirations, height, weight, body mass index (BMI), neck and waist circumference.
B. Inspect:
1. Inspect of face for deformities.
2. Perfom oropharynx examination for:
a. Peritonsillar narrowing or hypertrophy.
b. Tongue (evaluate for macroglossia).
c. Elongated or enlarged uvula.
d. Palate (high arch or narrow palate).
3. Measure neck circumference (>16 in. in females or 17 in. in males).
4. Perfom nasal examination:
a. Look for septal deviation and nasal polyps.
5. Inspect for signs of pulmonary hypertension or cor pulmonale:
a. Jugular venous distension.
b. Peripheral edema.
C. Palpate:
1. Thyroid gland.
D. Auscultate:
1. Heart and lungs.
E. Mental status:
1. Assess for confusion, flat affect, level of attention/alertness, and somnolence.

DIAGNOSTIC TESTS

A. Polysomnography (PSG) is the standard method of diagnosis. The apnea hypopnea index (AHI) or the respiratory disturbance index (RDI) is used to quantify hypopneas and classify the degree of sleep disturbance:
1. Full-night PSG (gold standard).
2. Split-night PSG.
3. Home testing with portable monitors.

Clients with cardiac, respiratory, or neurologic disease may be at the greatest risk for central sleep apnea, and the American Academy of Sleep Medicine (AASM) does not recommend the use of portable monitors for diagnosis in these clients.

B. Routine lab work is not helpful in the confirmation or exclusion of OSA.

DIFFERENTIAL DIAGNOSES

A. OSA.
B. Primary snoring.
C. Narcolepsy.
D. Restless legs syndrome.
E. Swallowing disorder.
F. Nocturnal seizures.
G. Gastroesophageal reflux disease (GERD).
H. Obesity hypoventilation syndrome.
I. Sleep deprivation, including shift-work disorder.
J. Neurodegenerative disease (e.g., Parkinson, dementia, Alzheimer).
K. Substance abuse.
L. Alcoholism.
M. Asthma.
N. Central sleep apnea.

PLAN

A. General interventions:
1. Continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BiPAP) are the mainstay of treatment of moderate to severe OSA. Treatment with CPAP and BiPAP is required at all times during the night and during naps.
B. Client teaching: *See Client Teaching Guide for this chapter, "Sleep Apnea."*

1. Educate the client about modifying controllable risk factors such as keeping diabetes and hypertension under control, diet, weight loss, exercise, and stopping smoking.

2. Behavioral strategies include sleeping in a nonsupine position using a positioning device (e.g., alarm, pillow, backpack, and tennis ball are used for positional therapy).

C. Dietary management:

1. Encourage dietary changes and exercise to promote weight loss.

2. Even a modest weight loss of 10% to 20% has been associated with an improvement.

D. Nonsurgical treatment:

1. Oral appliances (OAs) require a thorough dental examination.

a. Custom-made OAs may improve airway patency during sleep by enlarging the upper airway and/or by decreasing the upper airway collapse.

b. Mandibular repositioning appliances (MRAs) cover the upper and lower teeth and hold the mandible in an advanced position.

c. Tongue-retaining devices (TRDs) hold the tongue in a forward position without mandibular repositioning.

2. Avoid alcohol even during daytime due to central nervous system (CNS) depression and worsening apnea.

3. Avoid prescribing medications with sedating properties unless the benefit outweighs worsening sleep apnea (anticonvulsants for seizure control may not be avoided as benefits outweigh risk).

4. Encourage smoking cessation. *See Client Teaching Guide for* Chapter 25*, "Nicotine Dependence."*

E. Surgical treatment:

1. Tracheostomy can eliminate OSA but not central hypoventilation syndromes. This procedure should be considered only when other options have failed or when it is considered necessary by clinical urgency.

2. Maxillary-mandibular advancement (MMA) is indicated when the client cannot tolerate/refuses CPAP and an OA is not appropriate/effective.

3. Multilevel or stepwise surgery (MLS) is a combined procedure or serves as a stepwise multiple surgery.

4. Bariatric surgery's weight loss has been effective in improving sleep efficiency and REM sleep. The severity of presurgical OSA determines the degree to which OSA improves postbariatric surgery.

5. Radiofrequency ablation (RFA) is for treatment of mild to moderate OSA when the client cannot tolerate/refuses CPAP and an OA is not appropriate/effective.

6. Laser-assisted uvulopalatoplasty is not recommended for OSA.

FOLLOW-UP

A. There is no standard for recommending repeat PSG testing or a CPAP titration study after significant weight loss.

CONSULTATION/REFERRAL

A. Refer client to a dentist for an OA.

B. Refer client to a pulmonologist for management of therapy and/or surgical treatment.

C. Refer client to a cardiologist as needed.

D. If client is uninsured, refer to a social worker for assistance in treatment purchases as needed.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. OSA in older people is associated with worsening cardio-cerebrovascular, cognitive, and functional outcomes.

2. Fatigue, poor focus, and irritability can be sometimes attributed to dementia and treatment for any sleep breathing problem may improve some or all of those symptoms.

3. The use of positive pressure airway devices must be thoughtfully considered for clients with cognitive dysfunction and neuromuscular weakness. Clients who use CPAP should be instructed to travel with their device.

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BACTERIAL COMMUNITY–ACQUIRED PNEUMONIA

DEFINITION

A. Pneumonia is inflammation and consolidation of lung tissue due to a bacterial pathogen. The causative agent and the anatomic location classify pneumonia. It is not uncommon to have acute viral and bacterial pneumonia concurrently.

B. Other types of pneumonia and pulmonary inflammation occur secondary to smoking, exposure to chemicals or fungi, near drowning, and recurrent aspiration with gastroesophageal reflux.

C. Hospitalization is recommended in severe cases of pneumonia.

D. The Pneumonia Severity Index Calculator is a tool to estimate pneumonia mortality and assist in determining whether the client should best be treated in the inpatient or outpatient setting. See www.mdcalc.com/psi-port-score-pneumonia-severity-index-cap.

INCIDENCE

A. Pneumonia is a leading cause of death in clients older than 65 years.

B. Bacterial pneumonia is more prevalent in the very old and very young.

C. A higher mortality rate occurs in persons with immunodeficiency, comorbid conditions, abnormal vital signs, and virulent pathogens.
D. The incidence rate also varies by pathogens.

PATHOGENESIS

A. Pneumonia results from inflammation of the alveolar space. Lobar pneumonia has four stages:
1. Vascular congestion and alveolar edema within the first 24 hours of infection.
2. Red hepatization (2–3 days), characterized by erythrocytes, neutrophils, and fibrin within the alveoli.
3. Gray hepatization (2–3 days), characterized by a gray-brown to yellow color secondary to exudate.
4. Resorption and restoration of the pulmonary architecture. A rub may still be auscultated due to the fibrinous inflammation.
B. Bacterial causes include *Streptococcus pneumoniae* (the most common pathogen), *Haemophilus influenzae* type b (the second most common pathogen), *Staphylococcus aureus, Legionella, Chlamydia trachomatis, C. pneumoniae, Mycoplasma pneumoniae, Pseudomonas, Klebsiella*, and *Pneumocystis jirovecii* pneumonia (PCP) in clients with HIV.

PREDISPOSING FACTORS

A. Advanced age.
B. Impaired mentation.
C. Smoking.
D. Chronic obstructive pulmonary disease (COPD).
E. Alcoholism.
F. Aspiration.
G. Heart failure.
H. Diabetes.
I. Heart disease.
J. Crowded conditions (dormitories, long-term care centers).
K. Immunodeficiency.
L. Congenital anomalies.
M. Abnormal mucus clearance.
N. Lack of immunization.
O. Measles.
P. Indoor air pollutants from cooking or heating with wood.
Q. History of pneumonia.
R. Lung cancer.
S. Malnutrition.
T. Medications: immunosuppressives, oral corticosteroids, Proton Pump Inhibitor, and H2 antagonist.

COMMON COMPLAINTS

A. Acute onset of the following symptoms:
1. Fever.
2. Shaking chills.
3. Dyspnea, rapid, labored breathing.
4. Cough.
5. Rust-colored sputum.

OTHER SIGNS AND SYMPTOMS

A. Increased respiratory rate (tachypnea).
B. Chest pain, localized.
C. Upper respiratory infection (URI) symptoms such as pharyngitis.
D. Headache.
E. Nausea.
F. Vomiting.
G. Vague abdominal pain.
H. Diarrhea.
I. Myalgia.
J. Arthralgias.
K. Anorexia.
L. Change in level of consciousness (LOC), including confusion.

SUBJECTIVE DATA

A. Determine the onset, duration, and course of illness.
B. Has the client had fever or shaking chills?
C. Has there been breathing trouble? Are the breathing problems interfering with eating and drinking?
D. If diabetic, obtain take-home blood sugar readings.
E. Is there a cough? Is the cough productive? What color is the sputum?
F. Are any other family members ill?
G. Has the client ever been hospitalized for pneumonia or respiratory distress before?
H. Review the history for any chronic or comorbid conditions, including malignancy.
I. Has the client ever been immunized for influenza or pneumonia?
J. Review all medications, including over-the-counter (OTC) and herbal products. Specifically review whether the client has been on any antibiotics in the past 3 months. Recent exposure to an antibiotic is a risk factor for antibiotic resistance. Continued or repeated use of that class of antibiotics is not recommended.
K. Ask about change in sexual partners (including the elderly) for risk of HIV or chlamydia infections.
L. Review immunizations.
M. Ask about recent travel.
N. Review occupational risk factors.

PHYSICAL EXAMINATION

A. Check temperature, blood pressure (BP), pulse, and weight.
1. In the elderly the BP can be low; this is a consideration for sepsis.
B. Inspect:
1. Observe overall appearance. Does the client appear ill? Consider the clinical presentation, age of the person, and history. Assess mental status.
2. Observe breathing pattern and the use of accessory muscles, grunting, retractions, and tachypnea.
3. Obtain a pulse oximetry to assess oxygen saturation. **An Oxygen saturation less than 92% is an indicator of severity and the need for oxygen therapy.**
4. Check the nail beds and lips for cyanosis.
5. Examine the eyes, nose, ears, and throat.
6. Examine skin for turgor and mucous membranes for dehydration.
C. Auscultate:
1. Auscultate heart.
2. Auscultate lungs for the following (auscultate the bases first in geriatric clients):
a. Crackles (present in 80% of clients), wheezes, and decreased breath sounds.
b. Whispered pectoriloquy (increased loudness of whisper during auscultation).
c. Egophony (client's "e" sounds like "a" during auscultation).
d. Bronchophony (voice sounds louder than usual).
3. Auscultate abdomen (usually hypoactive bowel sounds). ▶

D. Percuss:
1. Percuss chest to identify areas of consolidation or dullness.
E. Palpate:
1. Palpate chest for tactile fremitus (increased conduction when client says "99").
2. Palpate lymph nodes for adenopathy.
3. Palpate sinuses for tenderness. Sinusitis is a sign of *Mycoplasma* infection.

DIAGNOSTIC TESTS

A. The World Health Organization (WHO) defines pneumonia solely on the basis of clinical findings observed by inspection and timing of respirations.
B. Chest radiograph (CXR):
1. Infiltrates confirm diagnosis. False negatives result from dehydration, evaluation in first 24 hours, and infection.
2. Ordering a posterior, anterior, and lateral CXR ensures adequate visualization for diagnosis.
C. Complete blood count (CBC) with differential.
D. BUN, creatinine level, and glomerular filtration rate (GFR) if acute renal failure secondary to dehydration or sepsis is a concern.
E. Cultures:
1. Blood cultures if critically ill, immunocompromised, or for persistent symptoms.
2. Sputum cultures are reserved for very ill clients for unusual presentations.
F. Consider rapid viral testing.
G. Consider skin testing for tuberculosis (TB) for high-risk exposure risk.
H. Acute phase reactants: C-reactive protein greater than or equal to 48 may favor diagnosis of community acquired pneumonia over exacerbation of chronic lung disease. Procalcitonin level may help differentiate bacterial from nonbacterial pneumonia.
I. Consider metabolic panel to assess renal and liver functions.

DIFFERENTIAL DIAGNOSES

A. Pneumonia:
1. Bacterial pneumonia.
2. Viral pneumonia.
3. Aspiration pneumonia.
4. Chemical-induced pneumonia.
5. Fungal pneumonia (especially in immunocompromised or diabetes out of control).
B. Asthma.
C. Bronchitis/bronchiolitis.
D. Pertussis.
E. Heart failure.
F. Pulmonary embolus.
G. Empyema and abscess.
H. Aspiration of foreign body.

PLAN

A. General interventions:
1. Encourage rest during acute phase.
2. Encourage the client to avoid smoking/secondhand smoke.
3. A vaporizer may be used to increase humidity.
4. Encourage good handwashing or use of hand sanitizer.
5. Severity of symptoms and safety of home environment should be part of the assessment for inpatient versus outpatient treatment.
6. Chest physiotherapy is not prescribed for pneumonia.
B. Client teaching: *See Client Teaching Guide for this chapter, "Pneumonia, Bacterial."*
C. Dietary management:
1. Encourage a nutritious diet with increased fluid intake.
D. Pharmaceutical therapy:
1. *S. pneumoniae* is the most common bacterial pathogen, and resistance against antibiotics is a concern. Resistance is seen more commonly in:
a. Age greater than 65 years.
b. Use of a beta-lactam antibiotic or fluoroquinolones in the past 6 months.
c. Alcoholism.
d. Chronic illness/comorbidities.
e. Immunosuppression therapy or illness.
f. Exposure to a child at a day-care center.
2. The Pneumonia Severity Index Calculator should be used prior to initiation of antibiotic therapy; it is available at www.mdcalc.com/psi-port-score-pneumonia-severity-index-cap.
3. Treatment with antibiotics is empirical. Oral therapy should continue 7 to 10 days.
a. If antimicrobials have not been used in the past 3 months:
i. Amoxicillin 1g orally TID.
ii. **Macrolide antibiotics (azithromycin) in areas with less than 25% community resistance to macrolide.**
iii. Doxycycline.
b. If antimicrobials have been used in the past 3 months:
i. Respiratory quinolones.
ii. Augmentin or high-dose amoxicillin.
c. Chronic comorbidities:
i. Monotherapy with respiratory fluoroquinolones.
ii. Combination therapy with amoxicillin -clavulanate OR cephalosporin PLUS macrolide or doxycycline.
4. Administer acetaminophen (Tylenol) for fever.
5. Avoid cough suppressants. Suppression of a cough may interfere with airway clearance.
6. Vaccines:
a. Pneumococcal vaccine is recommended for anyone older than the age of 19 with chronic lung disease, including asthma; immunocompromised adults; and in the elderly 65 years and older.

FOLLOW-UP

A. Clients should know the signs of increasing respiratory distress and seek immediate medical attention.
B. Follow up by telephone in 24 hours.
C. If there is no improvement after 48 hours on antibiotics, the client is advised to call back.
D. Schedule a return visit in 2 weeks for evaluation, unless elderly and living alone: Follow up in person within a week.
E. Clients older than 60 years and those who smoke should follow up with a CXR in 4 to 6 weeks. However, if the client is younger than 60 years, a nonsmoker, and feels well at 6-week follow-up, there is no need to follow up with a CXR.

EMERGENT ISSUES/INSTRUCTIONS

A. Clients demonstrating the following should receive same-day ED evaluation:
1. Respiratory compromise.

2. Neurologic changes including obvious central nervous system involvement.
3. Profound dehydration or alterations in vital signs.

CONSULTATION/REFERRAL

A. Clients who are immunocompromised or have signs of toxicity or hypoxia may need hospitalization. Refer them to a physician.
B. If the client is in any level of respiratory distress, dehydrated, or hypoxemic, consult with or refer the client to a physician/hospital.
C. Poor prognostic signs that require referral are age older than 65 years, respiration rate greater than or equal to 30 breaths per minute, systolic BP less than 90 or diastolic BP less than 60, temperature greater than 101°F, altered mental status, extrapulmonary infection, and white blood cell (WBC) less than 4,000 or greater than 30,000.
D. Physician consultation is needed for suspected PCP.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. The annual U.S. rate of antepartum community-acquired pneumonia (CAP) is 0.5 to 1.5 per 1,000 pregnancies.
2. Perinatal mortality may increase slightly due to associated increase in prematurity. Pneumonia puts older mothers at high risk of maternal death.
3. The symptoms of bacterial pneumonia are the same in pregnancy.
4. CXRs are acceptable in pregnancy to diagnose pneumonia.
B. Geriatrics:
1. Mortality rates from pneumonia are highest in geriatric groups.
2. Geriatric clients may present initially with confusion only, not with typical signs and symptoms of younger clients, and unable to mount a fever.
3. Older adults may remain afebrile in 20% of cases or more.
4. Up to half may not exhibit leukocytosis, but 95% have a left shift.
5. Aspiration pneumonia is more common as people age. Right lower lobe infiltration may indicate aspiration.
6. Avoid overuse of antibiotics. Antibiotics are indicated:
a. In an otherwise healthy older adult if fever of 102°F or greater is present, and/or respiratory rate is greater than 25.
b. In the absence of fever, in older adults with COPD and increased cough/purulent sputum.
c. In afebrile older adults without COPD if there is a new/increased cough, a respiratory rate greater than 25, rigors, or new-onset delirium.
7. Geriatric red flags: Geriatric clients with bacterial pneumonia may have worsening of any of the following geriatric syndromes:
a. Weight loss related to anorexia with viral illness.
b. Delirium/confusion due to illness.
c. Falls related to orthostasis with viral illness.

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PNEUMONIA (VIRAL)

DEFINITION

A. Viral pneumonia is inflammation and consolidation of lung tissue due to a viral pathogen.
B. Hospitalization is recommended for clients who are significantly immunocompromised and the frail elderly.

INCIDENCE

A. Viral pneumonia accounts for a small portion of pneumonia cases in adult pneumonia. It is not uncommon to have concurrent viral and bacterial infections.
B. Elderly persons have the highest rate of influenza-associated hospitalizations.

PATHOGENESIS

A. Pneumonia results from inflammation of the alveolar space and may compromise air exchange. Viral pneumonia is caused by influenza viruses, parainfluenza virus, adenovirus, and respiratory syncytial virus (RSV).
B. Influenza is the most common cause of viral pneumonia in adults.
C. Viruses and bacteria are spread from a cough or sneeze.
D. Pneumonia can also be spread via blood.

PREDISPOSING FACTORS

A. Age extremes.
B. Frailty or immunocompromised conditions.
C. Exposure to viral illness.
D. Lack of immunization.

COMMON COMPLAINTS

A. Fever.
B. Cough.
C. Dyspnea.
D. Tachypnea.
E. Wheezing (more common in viral pneumonia).

OTHER SIGNS AND SYMPTOMS

A. Upper respiratory prodrome.
B. Poor appetite.
C. Malaise/lethargy.
D. Myalgia.
E. Muscle aches.
F. Headache.
G. Fatigue.
H. Chest pain/tightness.

SUBJECTIVE DATA

A. Determine onset, duration, and course of illness.
B. Has the client had fever, cough, and upper respiratory infection?
C. Have there been any flulike symptoms?
D. Has there been any labored breathing?
E. Has there been a cough? Is it a productive cough? What color is the sputum?
F. Are the breathing problems affecting the ability to eat or drink?
G. Has the client had nausea, vomiting, or diarrhea?
H. Review if the client is up to date on immunizations.
I. Review all medications, including over-the-counter (OTC) and herbal products.

J. If diabetic, ask about home blood glucose readings/control.
K. Is there localized pain in chest?
L. Ask about pneumonia history.
M. Ask about pneumonia and influenza vaccine history.

PHYSICAL EXAMINATION

A. Check temperature, blood pressure (BP), pulse, respirations, and pulse oximetry. **Observe the level of consciousness (LOC).**
B. Inspect:
1. Observe overall appearance. Is the client septic? Consider the clinical presentation, age of person, and history.
2. Observe respiratory pattern, grunting, nasal flaring, retractions, and use of accessory muscles.
3. Check pulse oximetry. **An Oxygen saturation less than 92% is an indicator of severity and the need for oxygen therapy.**
4. Check nail beds and lips for cyanosis.
5. Examine eyes, ears, nose, and throat.
C. Auscultate:
1. Auscultate heart.
2. Auscultate lungs for the following (auscultate the bases first in geriatric clients):
a. Crackles, decreased breath sounds.
b. Whispered pectoriloquy (the client's whispered sounds are louder than normal).
c. Egophony (client's "e" sounds like "a").
d. Bronchophony (voice sounds louder than usual).
D. Percuss:
1. Percuss chest for dull sound (consolidation).
E. Palpate:
1. Palpate lymph nodes for swelling.
2. Palpate chest for tactile fremitus (increased conduction when client says "99").
3. Palpate sinuses.

DIAGNOSTIC TESTS

A. Chest radiograph (CXR), which reveals interstitial, perihilar, or diffuse infiltrates.
B. Complete blood count (CBC) with differential.
C. Rapid viral tests per nasal swab.
D. Sputum Gram stain if indicated.

DIFFERENTIAL DIAGNOSES

A. Viral pneumonia.
B. Bacterial pneumonia.
C. Varicella pneumonia.
D. Herpes pneumonia.
E. Cytomegalovirus pneumonia.
F. Pertussis.
G. Asthma.
H. Bronchitis/bronchiolitis.
I. Sinusitis.
J. Foreign-body obstruction.
K. Aspiration.
L. Fungal pneumonia (especially in the immunocompromised).
M. Severe acute respiratory syndrome (SARS).
N. Middle East respiratory syndrome coronavirus (MERS-CoV).
O. Avian flu.
P. Pulmonary tuberculosis (TB).

PLAN

A. General interventions:
1. Instruct the client to rest during acute phase.
2. Avoid smoking or secondhand smoke.
3. Require respiratory isolation; may use facial masks.
4. Encourage good handwashing or use of hand sanitizer.
5. Chest physiotherapy is not prescribed for pneumonia.
B. Client teaching: *See Client Teaching Guide for this chapter, "Pneumonia, Viral."*
C. Dietary management:
1. Encourage fluids and nutritious diet.
D. Pharmaceutical therapy:
1. Antiviral agents have limited support for efficacy. Treatment should be targeted based on viral etiology.
a. Oseltamivir, zanamivir, peramivir, and baloxavir are recommended and started within 24 to 48 hours of onset of fever and symptoms.
b. Amantadine and rimantadine have high levels of resistance and are not recommended.
c. Acyclovir (Zovirax) for herpes viruses is administered as an intravenous (IV) infusion.
2. Clients with viral pneumonia who are superinfected with bacterial organisms require antibiotic therapy.
3. Avoid cough suppressants. Suppression of a cough may interfere with airway clearance.
4. Give acetaminophen (Tylenol) for fever.
5. Immunizations:
a. **Recommend the pneumonia and influenza vaccines for prevention.**
i. Administer pneumonia vaccine in clients 65 years and older, or beginning at the age of 19 if immunocompromised or chronic respiratory condition is present. Consider revaccination every 5 to 10 years in high-risk clients.
ii. Sequential administration of the pneumococcal conjugate vaccine (PCV13) and pneumococcal polysaccharide vaccine (PPSV23) is recommended (download the Centers for Disease Control and Prevention [CDC] guide at www.cdc.gov/vaccines/vpd/pneumo/downloads/pneumo-vaccine-timing.pdf). In the vaccine-naive client, give PCV13 first, then the PPSV23 12 months later.
iii. In older adults who received PPSV23 at the age of 65 or older, give PCV13 in 1 year or more.
iv. In older adults who received PPSV23 BEFORE the age of 65, give PCV13, then 5 years after the initial PPSV23 give a second PPSV23.
v. Give a onetime second dose of PPSV23 in persons with chronic renal failure or nephrotic syndrome, functional or anatomic asplenia, or immunocompromising conditions.

Administer yearly flu vaccine. Trivalent or quadrivalent influenza vaccines are available. Give the client the vaccine in late September, at least 6 weeks before the flu season. Yearly flu vaccination information is available on the CDC website.

FOLLOW-UP

A. Signs and symptoms may vary greatly according to the viral pathogen, severity of disease, and the client's age:
1. Instruct the client to return to the clinic if no improvement is seen after 48 hours on antiviral agents. ▶

2. Follow up by phone in 24 hours.
3. Consider follow-up at 2 weeks if bronchoconstriction is noted on exam.

EMERGENT ISSUES/INSTRUCTIONS

A. Clients demonstrating the following should receive same-day ED evaluation:
1. Respiratory compromise.
2. Neurologic changes including obvious central nervous system (CNS) involvement.
3. Profound dehydration or alterations in vital signs.

CONSULTATION/REFERRAL

A. Consult a physician if viral pneumonia is strongly suspected.
B. Consult a physician if the client is pregnant.
C. Consult a physician or transfer to the hospital if the client is in respiratory distress, dehydrated, or hypoxemic.
D. Consider consultation with a pulmonologist.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Ribavirin, a class X drug, is contraindicated in pregnancy.
2. Acyclovir is given in the third trimester at 10 mg/kg IV Q8H for 5 days.
3. The varicella zoster immune globulin (VZIG) may be considered in pregnancy.
4. The measles virus is a live attenuated virus and should not be given during pregnancy.
5. Pneumonia during pregnancy is associated with higher morbidity rates. Physician consultation is advised.
B. Geriatrics:
1. Mortality rates from pneumonia are highest in geriatric groups.
2. Geriatric clients may present initially with confusion only, not with typical signs and symptoms of younger clients, and unable to mount a fever.
3. Geriatric clients in communal living situations should adhere to vaccination guidelines for pneumococcus, influenza, and COVID-19.
4. Geriatric red flags: Geriatric clients with viral pneumonia may have worsening of any of the following geriatric syndromes:
a. Weight loss related to anorexia with viral illness.
b. Delirium/confusion due to illness.
c. Falls related to orthostasis with viral illness.

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TUBERCULOSIS

DEFINITION

A. Tuberculosis (TB) is an infectious disease. It is a granulomatous disease caused by *Mycobacterium tuberculosis*, *M. bovis*, and other mycobacteria. Humans are the only reservoirs for *M. tuberculosis*. TB may involve multiple organs, including the lungs, liver, spleen, kidney, eyes, brain, spine, and bone.
B. In most immunocompetent individuals, macrophages are successful in containing the bacilli, and the infection is self-limited and often subclinical. As many as 5% of adults with primary TB are asymptomatic, with approximately 41% of cases worldwide being undiagnosed. When the pulmonary macrophages are unable to contain the bacilli, it leads to clinically apparent infection-progressive primary TB.
C. Postprimary (reactivation) TB occurs when the initial infection is successfully contained by the pulmonary macrophages, with bacilli remaining viable within the macrophages. Infection results when the host's immune status (T cells) is compromised.
D. Clients with fever of unknown origin, failure to thrive, significant weight loss, or unexplained lymphadenopathy should be evaluated for TB.
E. The lungs are the most common site for the development of TB; 85% of symptomatic clients present with pulmonary complaints.

INCIDENCE

A. TB is a worldwide infection and is considered a global public health emergency by the World Health Organization (WHO). One-third of the world population are infected:
1. The WHO reports more than nine million new cases of TB every year.
2. TB can affect any age group. Over 60% of the cases are 25 to 40 years old.
3. TB is the leading cause of death in those infected with HIV.
4. The incidence of TB in the United States is 2.9 cases per 100,000 population.
5. Due to the high risk among immigrants, California, New York, Texas, and Florida accounted for half of the TB cases reported in the United States.
B. Postprimary TB is a significant cause of worldwide morbidity and mortality. Pulmonary morbidity results from a chronic cough, hemoptysis, fibrosis, superinfection, bronchial stenosis, repeated pulmonary infections, or empyema. Morbidity also arises from chronic TB osteomyelitis, chronic renal insufficiency, and central nervous system (CNS) TB.

PATHOGENESIS

A. Mycobacteria are non spore-forming, slow-growing bacilli. TB infection occurs by means of inhalation of airborne bacillus droplets from an infected host. The development of an infection depends on prolonged exposure (weeks) to an individual with active pulmonary TB. Bacilli travel through the pulmonary lymphatics or enter the vascular system and are disseminated to the brain, meninges, eyes, bones, joints, lymph nodes, kidneys, intestines, larynx, and skin. The incubation period from infection to a positive skin test reaction is 2 to 10 weeks, although disease may not occur for many years or may never occur. The risk of disease is greatest within 2 years following infection.

PREDISPOSING FACTORS

A. Exposure to someone with active disease, including household contact.
B. High-risk groups: minorities, birth in TB-endemic country, prisoners, nursing home residents, teachers, indigents, migrant workers, and healthcare providers.
C. HIV infection is one of the most significant risk factors for TB.
D. Steroid therapy, cancer chemotherapy, and hematologic malignancies increase the risk of TB. ▶

E. Tumor necrosis factor-alpha (TNF-alpha) antagonists used for rheumatoid arthritis, psoriasis, and other autoimmune disease are associated with a significant risk for TB. **Prior to beginning any TNF-alpha treatment, a TB skin test should be administered**.
F. Non-TB infections such as measles, varicella, and pertussis may activate quiescent TB.
G. Smoking.
H. Malnutrition.
I. Alcoholism.
J. Intravenous (IV) drug abuse.
K. Congenital TB (rare).
L. Poverty.

COMMON COMPLAINTS

A. Fever (usually low grade at onset but becoming marked with progression of disease).
B. Malaise.
C. Weight loss/difficulty gaining weight.
D. Cough.
E. Night sweats.
F. Chills.
G. Occasional hemoptysis.
H. Fatigue.
I. Anorexia.

OTHER SIGNS AND SYMPTOMS

A. Pulmonary TB: fatigue, irritability, and undernutrition, with or without fever and cough.
B. Glandular TB: chronic cervical adenitis.
C. Meningeal TB: fever and meningeal signs, and positive cerebrospinal fluid.
D. Skeletal TB: back pain or stiffness, lower extremity paralysis, and tuberculous arthritis, which usually involves only one joint (most often the hip or knee, followed by the ankle, elbow, wrist, and shoulder).
E. Genitourinary TB: flank pain, dysuria, frequent urination, pen-painful scrotal mass, prostatitis, orchitis, or epididymitis; in females symptoms mimic pelvic inflammatory disease.
F. Gastrointestinal TB: referable to the infected site including nonhealing ulcers in the mouth or anus, difficulty swallowing (esophageal disease), abdominal pain mimicking peptic ulcer disease, malabsorption, pain, diarrhea, or hematochezia.

SUBJECTIVE DATA

A. Determine onset, duration, and course of illness.
B. Review symptom history: fever, night sweats, chills, or cough.
C. Review history of weight loss.
D. Review exposure history to someone who has TB and the date of past screening.
E. What is the client's living situation?
F. Review travel history to endemic areas with TB, including China, Pakistan, the Philippines, Thailand, Indonesia, Bangladesh, and the Democratic Republic of Congo.
G. Review HIV status or need for testing.

PHYSICAL EXAMINATION

A. A complete physical examination is mandatory. Physical findings of pulmonary TB are not specific and are usually absent in mild or moderate disease.
B. Record temperature, respirations, pulse, blood pressure (BP), and weight. Compare weight with previous readings in the past 12 months.
C. Inspect:
  1. Observe overall appearance.
  2. Check skin for pallor.
  3. Inspect eyes, ears, nose, and throat. Evaluate for nonhealing ulcers in the mouth or anus.
D. Auscultate:
  1. Auscultate heart.
  2. Auscultate lungs and chest for the following:
    a. Rales in upper posterior chest.
    b. Bronchophony (voice sounds louder than usual).
    c. Whispered pectoriloquy: Client's whispered sounds are louder than normal.
E. Percuss:
  1. Percuss chest.
F. Palpate:
  1. Palpate for lymphadenopathy, usually anterior or posterior cervical and supraclavicular nodes. Less commonly involved lymph nodes include submandibular, axillary, and inguinal lymph nodes.
  2. Palpate to evaluate hepatosplenomegaly.
G. Perfom neurologic examination:
  1. Evaluate the presence of nuchal rigidity.
  2. Assess deep tendon reflexes.

DIAGNOSTIC TESTS

A. Diagnosis is based on a combination of purified protein derivative (PPD) skin testing, serum testing with interferon-gamma release assays (IGRAs), and sputum cultures. Bronchoscopy may be required to obtain specimens. Clients with primary TB may not undergo imaging; however, conventional chest radiograph (CXR) may be performed, and 15% of clients with primary TB have normal CXR findings.
B. Clients with progressive primary or postprimary TB may need a CT to evaluate parenchymal involvement, satellite lesions, bronchogenic spread, and miliary disease. MRI may be ordered to evaluate complications, such as the extent of thoracic wall involvement with empyema.
C. Tuberculin skin test using the Mantoux test is the recommended method. Dosage of 0.1 mL or 5 tuberculin units (TU) of PPD should be injected intradermally into the volar aspect of the forearm using a 27-gauge needle. A wheal should be raised and should measure approximately 6 to 10 mm in diameter. Skilled personnel should read the test in 48 to 72 hours after administration. Measure the amount of induration and not the erythema. Measure transverse to the long axis of the forearm (Table 12.3).
  1. Sputum culture with acid-fast smear; nasopharyngeal secretions and saliva are not acceptable.
D. The Food and Drug Administration has approved Quantiferon TB Gold as an alternative TB test for detecting both TB and latent TB infection.
E. Annual Mantoux tests for those at high risk are recommended. There is no need to repeat PPD once the client has reacted and had a positive PPD. A two-step test is recommended for initial screening only.
F. Anteroposterior (AP) and lateral CXR films: "Snowstorm" appearance indicates miliary TB; segmental consolidation and hilar adenopathy are common; pleural effusion may be present.
G. Complete blood count (CBC) with differential.
H. IGRA test.
I. HIV testing as indicated by risk factors to guide management.
J. Pregnancy test (if indicated) to guide management.

TABLE 12.3 INTERPRETATION OF MANTOUX TUBERCULIN SKIN TEST

Positive Reaction—Induration Size	Risk Factors
5 mm or more	Close contact with known or suspected TB; immunosuppressive conditions (e.g., HIV); on immunosuppressive medications; or an abnormal chest x-ray—consistent with active TB, previously active TB, or clinical evidence of the disease
10 mm or more	High-risk categories (e.g., homeless, HIV-infected, users of illicit drugs, residents in nursing homes, incarcerated, or institutionalized); travel histories to high-prevalence areas of world
15 mm or more	Any age without any risk factors for TB

TB, tuberculosis.

DIFFERENTIAL DIAGNOSES

A. TB.
B. Bronchiectasis.
C. Asthma.
D. Histoplasmosis.
E. Coccidioidomycosis.
F. Blastomycosis.
G. Malignancies.
H. Other pulmonary infections.
I. Aspiration pneumonia.

PLAN

A. General interventions:
1. Report all suspected and confirmed cases of TB to the local health department.
2. Directly observed therapy (DOT) is mandatory for treatment of clients with coexistent HIV disease, those with multidrug-resistant (MDR) TB, and those who may be noncompliant.
B. Client teaching:
1. Educate clients regarding compliance to therapy, adverse effect of medications, and follow-up care.
C. Pharmaceutical therapy:
1. The American Thoracic Society (ATS) and the Centers for Disease Control and Prevention (CDC) provide standard guidelines for therapy:
a. Refer to the CDC for up-to-date guidelines on treatment: www.cdc.gov/tb/publications/guidelines/treatment.htm. Medication protocols vary and include the following first-line agents: isoniazid (INH), rifampin (Rifadin), pyrazinamide (Tebrazid), and ethambutol (Etibi):
i. INH (Laniazid, Nydrazid) dosing:
1) Adults: 5 to 10 mg/kg PO; not to exceed 300 mg/d.
ii. Rifampin (Rifadin) dosing:
1) Adults: 600 mg PO every day.
iii. Pyrazinamide (Tebrazid) dosing:
1) Adults: 15 to 30 mg/kg PO every day; not to exceed 2 g/d.
iv. Ethambutol (Myambutol) dosing:
1) Adults with no previous anti-TB therapy: 15 mg/kg PO every day.
2) Adults with previous therapy: 25 mg/kg PO every day.
b. Compliance with drug regimen is most important.
c. Drug resistance averages 5% to 10% nationally and is increasing.
d. The Bacillus Calmette-Guerin (BCG) vaccine is available in TB-endemic countries for prevention of childhood TB-related meningitis and miliary TB. BCG is a live vaccine. BCG does not prevent infection with *M. tuberculosis*.

FOLLOW-UP

A. Regular follow-up every 4 to 8 weeks to ensure compliance and to monitor the adverse effects and response of the medications.
B. Repeat CXR may be performed after 2 to 3 months of therapy to observe the response to treatment of clients with pulmonary TB.
C. Consider monitoring liver enzymes monthly in the following clients:
1. Severe or disseminated TB.
2. Concurrent or recent hepatic disease or hepatobiliary tract disease from other causes.
3. Those receiving high doses of INH (10 mg/kg/d) in combination with rifampin, pyrazinamide, or both drugs.
4. Those who are pregnant or within the first 6 weeks postpartum.
5. Clinical evidence of hepatotoxic effects.

CONSULTATION/REFERRAL

A. An infectious disease consultation for management of affected clients.
B. Reportable illness to the county health department.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Manage TB on a case-by-case basis with a physician.
2. Chemotherapy must be started immediately after the first trimester to protect both mother and fetus:
a. First-line agents recommended by the American Academy of Pediatrics include INH, rifampin, and ethambutol.
b. Streptomycin is contraindicated in pregnancy.
3. All pregnant persons on INH therapy should receive pyridoxine.
4. Pregnant persons are at increased risk for INH-induced hepatotoxicity.
5. The mother who has current disease but is noncontagious at delivery does not require separation from their infant. These mothers can also breastfeed.
6. The mother who has current disease and is contagious at delivery requires separation from their infant until the mother is noncontagious.
B. Geriatrics:
1. Geriatrics, no matter the ethnic group or gender identity, represent the largest reservoir of TB infection, especially in developed nations. The institutionalized geriatric client is at particular risk for both reactivation of latent TB and development of new infection. ▶

2. Baseline acute/chronic disease processes, malnutrition, and normal changes associated with aging make the diagnosis of TB difficult. Clinical features may be atypical, nonspecific, and mimic concomitant age-related diseases.

3. Treatment of TB in the geriatric population is difficult due to adverse drug reactions and drug-to-drug interactions.

4. Treatment priorities include treatment of associated chronic diseases, minimization of invasive procedures, limitation of polypharmacy, and appropriate nutrition support.

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CLIENT TEACHING GUIDE

ASTHMA

PROBLEM Asthma is a chronic condition with wheezing, coughing, breathlessness, and chest tightness.

CAUSE The most common cause is inflammation that results from exercise or exposure to environmental irritants, allergens, furry animals, cockroaches, dust mites, pollens and molds, cold air, or viral respiratory infections.

PREVENTION

A. You have asthma and so are encouraged to get the flu vaccine every year.
B. Always keep your quick-relief (rescue) inhaler with you.
C. Asthma is more difficult to control in obesity. Weight reduction, even 5% to 10% of weight loss, can improve asthma control.
D. You may be able to prevent frequent recurrences of asthma by following these asthma trigger avoidance strategies:
1. Dust mite allergens: Wash bedding weekly in hot water and dry it in a hot dryer. Encase pillows and mattresses in airtight covers. Remove carpets, especially from your bedroom. Avoid use of fabric-covered furniture, especially for sleeping.
2. Cockroach allergens: Clean your house thoroughly. Use poison bait or traps. Do not leave food or garbage exposed.
3. Animal fur allergens: Avoid keeping house pets, or at least do not allow them in sleeping areas.
4. Smoke allergens: Avoid smoking, contact with tobacco smoke, smoke from wood-burning stoves or fireplaces, and unvented stoves or heaters.
5. Outdoor pollens and molds: Keep windows closed when pollen or mold counts are high.
6. Indoor mold: Reduce dampness in your home by using a dehumidifier. Clean damp areas often. Remove carpets that are laid on concrete.
7. Other irritants: Avoid perfumes, cleaning agents, and sprays.

TREATMENT PLAN You should also be given the *Client Teaching Guides "Asthma Action Plan and Peak Flow Monitoring"* and *"How to Use a Metered-Dose Inhaler."*

Activity:
A. If cold air causes symptoms, wear a scarf over your mouth and nose if you must go outside during the winter.
B. Avoid vigorous exercise if this causes asthma symptoms. Learn to recognize activities that trigger your breathing problems.
C. Make an asthma action plan to follow. Copy the plan and place it on the refrigerator, take a copy of your action plan to school, and give each coach a copy of your asthma action plan.
D. Color-code your inhalers with tape or markers; for example, use green tape for quick-relief inhalers and blue tape for long-acting inhalers.

Diet: There are no diet restrictions unless you have found a food that causes an allergic reaction and trouble with breathing.

Medications: Cough medicines should not be used for asthma symptoms.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. A peak flow reading below 60% of your personal best number that does not return to the yellow or green zone after taking your medication.
B. Other: _____

Phone: _____

RESOURCE A Patient's Guide to Aerosol Drug Delivery: http://www.copd-alert.com/AerosolDrug.pdf

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CLIENT TEACHING GUIDE

ASTHMA ACTION PLAN AND PEAK FLOW MONITORING

A peak flow meter is a device that measures how well air moves out of your lungs. This measurement is referred to as your "peak expiratory flow" (PEF).

HOW TO USE A PEAK FLOW METER

A. Place the indicator at the bottom of the numbered scale.
B. Stand up.
C. Take a deep breath, filling your lungs as deeply as possible.
D. Place the mouthpiece in your mouth and close your lips around it. Do not put your tongue inside the hole.
E. Blow into the mouthpiece as hard and fast as you can. It is important to give this your best effort.
F. Write down the number on the indicator. If you cough or make a mistake, do not record that number—do it over again.
G. Repeat steps A through F two more times.
H. Write down the highest number of the three attempts. This is your PEF.

CALCULATING YOUR PERSONAL BEST PEAK FLOW NUMBER

This number is the highest peak flow number you can achieve over a 2- to 3-week period when your asthma is under good control (when you do not have any symptoms). To find this number, take peak flow readings.

A. Twice daily for 2 to 3 weeks.
B. When you wake up and between noon and 2 p.m.
C. Before and after taking your quick-relief medication.
D. Or as directed by your healthcare provider.

THE PEAK FLOW ZONE SYSTEM

Once you have determined your personal best peak flow number, your healthcare provider can give you the numbers that let you know what medications to take based on your PEF. The numbers are set up like a traffic light system (red, yellow, and green).

Green zone (80%–100% of your personal best number): signals good control. No asthma symptoms are present, and you should take your medication as usual.

Yellow zone (50%–80% of your personal best number): signals caution. You may be having an episode of asthma that requires an increase in your medications.

Red zone (below 50% of your personal best number): signals a medical alert. You must use your "fast" inhaler to help open up your airways right away and call your healthcare provider immediately if your peak flow number does not return to the yellow or green zone and stay there.

Use the following Asthma Action Plan, which specifies what medications you should take when you are in each zone, and also use the self-assessment diary provided in Table 12.4.

ASTHMA ACTION PLAN

A. My personal best PEF is _____.
B. When I am in the ***green zone***, PEF above _____, I should continue to take my regularly scheduled asthma medications. They are:
1. _____
2. _____
3. _____

C. When I am in the ***yellow zone***, PEF between _____ and _____, I should add the following to my regularly scheduled medications:
1. _____
2. _____
3. _____

D. When I am in the ***red zone***, PEF below _____, I should immediately take the following rescue medication and contact my healthcare provider:

(*continued*)

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CLIENT TEACHING GUIDE

ASTHMA ACTION PLAN AND PEAK FLOW MONITORING (*CONTINUED*)

TABLE 12.4 ASTHMA DIARY SELF-ASSESSMENT

Symptom Codes: Rate Your Symptoms 1 (Mild); 2 (Moderate); or 3 (Severe)							
My Personal Best PEF Is _____							
Date	**PEAK FLOW ZONES Green = Good control Yellow = Caution Red = Emergency**			**Symptoms (Use Codes) W = Wheeze C = Cough S = Shortness of Breath**	**Quick–Relief Medication (Include the Number of Times Needed for Relief)**	**Anti-Inflammatory Medication**	**Additional Medications or Activity**
Monday	a.m.	p.m.	Zone				

PEF, peak expiratory flow.

1. _____
2. _____
3. _____

Rescue Medication: _____

E. Other Directions: _____

_____

_____

RESOURCE American Academy of Allergy, Asthma & Immunology (AAAAI): www.aaaai.org

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CLIENT TEACHING GUIDE

PNEUMONIA BACTERIAL

PROBLEM Pneumonia is a lung infection that causes fluid to collect in the air sacs. You may have a fever, cough, or trouble breathing.

CAUSE Respiratory bacteria or viruses cause pneumonia.

PREVENTION

A. Although the flu vaccine will not keep you from getting bacterial pneumonia, a flu vaccine is recommended every year.
B. There are two types of pneumonia vaccines. People older than 65 years of age, people who smoke, younger people with chronic lung disease, and people with other medical conditions may receive vaccines to prevent pneumococcal pneumonia.
C. You may be given one of the pneumonia vaccines at the same time as you get the flu vaccine.
D. Avoid contact with people with respiratory illnesses.

TREATMENT PLAN

A. Use a cool-mist humidifier. Clean the humidifier daily.
B. Do not smoke, and avoid smoke-filled rooms.
C. Cover your mouth when you cough and cover your nose when you sneeze.
D. Use tissues when you blow your nose. Throw away tissues as soon as they are used.
E. If no tissue is available, do the "elbow sneeze" into the bend of your arm/sleeve of your shirt.
F. Wash your hands frequently with soap and water or use hand sanitizer.

Activity: Rest during the early phase of the illness.

Diet: Eat a nutritious diet. Drink 8 to 10 glasses of water a day.

Medications:

A. Do not use cough suppressants if your cough produces sputum. Use them only for a dry, nonproductive cough.
B. Acetaminophen (Tylenol) may be used for fever or body aches.
C. Antibiotics are given for pneumonia that is caused by bacteria. Take your medication as directed.
D. Finish all of your antibiotics even though you may feel better.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Increased difficulty breathing.
B. Fever after 48 hours on an antibiotic.
C. Blood in your sputum.
D. Worsening discomfort or fatigue.
E. Trouble keeping your antibiotic down.
F. Diabetes and your blood sugars are elevated, or you notice ketones in your urine while you are sick.
G. Other: _____

Phone: _____

Follow-up appointment on: _____

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CLIENT TEACHING GUIDE

BRONCHITIS, ACUTE

PROBLEM Acute bronchitis is a lung infection followed by a productive cough.

CAUSE Respiratory viruses cause bronchitis.

PREVENTION

A. Although the flu shot does not prevent acute bronchitis, you are encouraged to take the flu vaccine every year.
B. Avoid exposure to other people with respiratory illnesses.
C. Do not smoke, and avoid secondhand smoke and other smoke-filled environments.
D. Avoid air pollutants, such as wood smoke, solvents, and cleaners.
E. Cover your nose and mouth when you sneeze or cough.
F. Use tissues when you blow your nose.
G. Throw away all tissues as soon as they are used. If no tissue is available, do the "elbow sneeze" into the bend of your arm/sleeve of your shirt.
H. Use good handwashing techniques with soap and water or hand sanitizer.

TREATMENT PLAN

A. Humidity and mist may be helpful.
B. Always clean the humidifier daily to prevent bacteria from growing.
C. Twenty minutes several times a day in a steamy bathroom may provide relief.

Activity: Rest is important since you have been diagnosed with bronchitis; increase activity as tolerated when the fever subsides.

Diet: Eat a nutritious diet. Drink 8 to 10 glasses of water daily.

Medications:

A. Acetaminophen (Tylenol) may be used to relieve discomfort.
B. For a nonproductive cough, take cough suppressants if recommended. You may be prescribed a cough medicine or be told the best kind to buy in the drugstore.
C. Since a virus almost always causes acute bronchitis, **antibiotics will rarely be needed to get better.**

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. No improvement after 48 hours.
B. Worsening symptoms.
C. High fever, chills, chest tightness or pain, or shortness of breath.
D. Symptoms that last longer than 3 weeks.
E. Diabetes and your blood sugars are elevated, or you notice ketones in your urine while you are sick.
F. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

BRONCHITIS, CHRONIC

PROBLEM Chronic bronchitis is an upper respiratory infection followed by a productive cough. To be diagnosed with chronic bronchitis, you need to have had the symptoms 3 months for 2 years in a row.

CAUSE Both viruses and bacterial infections cause chronic bronchitis.

PREVENTION

A. A yearly flu vaccine is recommended because the flu may lead to bronchitis.
B. There are two pneumonia vaccines. Pneumonia vaccines are recommended for people older than 65 years of age, younger people who smoke, and for people with chronic respiratory conditions and other medical conditions.
C. You may be given one of the pneumonia vaccines at the same time as you get the flu vaccine.
D. Avoid exposure to others with respiratory illnesses.
E. Do not smoke, and avoid secondhand smoke and smoke-filled environments.
F. Avoid other air pollutants, such as wood smoke, solvents, and cleaners.
G. Use good handwashing techniques and use hand sanitizers.
H. Use tissues for the mucus coughed up. Dispose of the tissues after use.
I. Cover your mouth when you cough. If you do not have a tissue, the "elbow sneeze" into the bend of your arm/into your sleeve will prevent you from spreading your illness.

TREATMENT PLAN

A. Humidity and mist may be helpful.
B. Always clean the humidifier daily to prevent bacterial growth.
C. Twenty minutes several times a day in a steamy bathroom may provide relief.
D. If continuing to smoke, do your best to reduce the amount.

Activity: Rest during the early stage of the illness, then increase activity as tolerated when the fever subsides. It is not uncommon to feel tired for several weeks.

Diet: Eat a nutritious diet. Drink 8 to 10 glasses of water daily.

Medications:

A. Acetaminophen (Tylenol) may be used to relieve fever and discomfort.
B. You may be prescribed an inhaler to help your breathing.
C. You may be prescribed steroids to help with the inflammation of your lungs. The steroids may be given by an inhaler or as a pill.
D. It is very important that you use the inhaler properly so that the medicine can go into your lungs. A teaching sheet on how to use an inhaler is available.
E. You may also be prescribed an antibiotic for a bacterial infection. Take all of your antibiotics even if you feel better.
F. You may be prescribed a cough suppressant to take at night to help you rest. However, coughing up the mucus is very important to clear out your "windpipes."

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. No improvement after 48 hours.
B. Worsening symptoms.
C. High fever, chills, chest tightness or pain, or shortness of breath.
D. Symptoms that last longer than 3 weeks after taking all of your antibiotics.
E. Diabetes and your blood sugars are elevated, or you notice ketones in your urine while you are sick.
F. Other: _____

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CLIENT TEACHING GUIDE

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

PROBLEM Chronic obstructive pulmonary disease (COPD) is a chronic, progressive, debilitating disease of the lungs that does not have a cure. Most people have a combination of emphysema and chronic bronchitis. Persons with COPD usually have some of the following symptoms: cough (usually productive), shortness of breath at rest or with exertion, wheezing, decreased energy level, and weight loss.

CAUSE COPD is most commonly associated with cigarette smoking. COPD is also caused by long-term exposure to pulmonary irritants in the environment (e.g., coal dust). Repeated respiratory infections may also contribute to the development of COPD.

PREVENTION

A. Avoid smoking and exposure to secondhand smoke.
B. Avoid exposure to environmental irritants, including pollution, household cleaning products, and smoke from fires.

TREATMENT PLAN

A. Stopping smoking is the most important treatment. Talk to your healthcare provider about support for stopping. It is never too late to stop smoking. You should also be given the *Client Teaching Guide "Nicotine Dependence."*
B. Get the flu vaccine every year.
C. There are two pneumococcal vaccines; you may need to be given both vaccines.
D. You may be given one of the pneumonia vaccines at the same time as you get the flu vaccine.
E. Reduce your exposure to lung irritants and extremely hot and cold air temperatures.
F. Begin an exercise program with your healthcare provider's approval. Walking is a good aerobic exercise. Begin with a pace that is tolerable and easy to maintain, then increase the duration and intensity of the exercise as tolerated. Stop if you experience shortness of breath or chest pain. A realistic goal may be 5 to 10 minutes a day, eventually increasing to 30 to 40 minutes a day.
G. Use a spacer/holding chamber to help you inhale all of your medicine. Spacers help you place more of your medicine in your lungs instead of at the back of your throat and mouth. Keep your spacer clean. You should also be given the *Client Teaching Guide "How to Use a Metered-Dose Inhaler."*
H. Use slow, deep breathing or pursed-lip breathing when you are short of breath. Breathe out like you are blowing out a candle.
I. Ask your healthcare provider if you are a candidate for low-flow oxygen treatment when shortness of breath occurs at night and causes insomnia and restlessness.
J. If you are prescribed theophylline to help control your respiratory systems, your blood level needs to be monitored.

Activity: Group activities together, such as planning shopping with going to the post office. Schedule rest periods throughout the day. Exercise programs should help increase activity tolerance.

Diet: Good nutrition is important. Six small, high-calorie meals a day are suggested. Avoid excessive intake of carbohydrates, especially simple carbohydrates like candy, soda, and potato chips. Milk and milk products increase the production of mucus. Ask your healthcare provider to refer you to a dietitian if nutritional problems persist.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. Your mucus changes color or increases in amount, or if the consistency is becoming thicker.
B. Your wheezing or shortness of breath is getting worse after you start your medication.
C. You are having trouble walking or talking because of your shortness of breath.
D. Other: _____

Phone: _____

You should have a follow-up appointment in 3 months unless you worsen before then.

(*continued*)

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CLIENT TEACHING GUIDE

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (*CONTINUED*)

If you are prescribed theophylline, your blood level needs to be monitored whenever you have a new illness, worsening chronic illness, or change in medications that may alter theophylline clearance (e.g., fever >102°F sustained for ≥24 hours, hepatitis). Call 911 and get emergency medical help for severe headache, high blood sugars, severe vomiting, irregular heartbeats, seizures, or confusion.

RESOURCE A Patient's Guide to Aerosol Drug Delivery: http://www.copd-alert.com/AerosolDrug.pdf

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CLIENT TEACHING GUIDE

COMMON COLD

PROBLEM The common cold is swelling of the mucous membranes of the respiratory tract. Most people complain of feeling tired and have a runny or stopped-up nose, a sore throat, hoarseness, and watery and/or red eyes. You may have a low-grade fever or no fever at all.

CAUSE A virus usually causes the common cold.

PREVENTION

A. The flu vaccine is recommended to be taken every year.
B. Colds are spread from one person to another through hand-to-hand contact and contact with air droplets from sneezing, coughing, and talking.
C. Practice good handwashing techniques with soap and water or hand sanitizers.
D. Do not drink from the same glass after others.
E. Cover your mouth and nose when you sneeze or cough.
F. Use tissues when you blow your nose. Dispose of them and then wash your hands.
G. If no tissue is available, do the "elbow sneeze" into the bend of your arm/shirt sleeve (away from your open hands). Always wash your hands.

TREATMENT PLAN

A. Using a humidifier for your bedroom or inhaling steam helps keep the mucous membranes of your nose from drying.
B. Discuss using saline nose drops.
C. Secondary infections of the respiratory tract (sinuses, lungs) may occur. If these do occur, then antibiotic therapy may be needed.
D. Zinc preparations are not recommended for an acute cough caused by the common cold.

Activity: There are no activity restrictions. Frequent rest periods or naps can help with fatigue.

Diet: Eat well-balanced meals and snacks. Drink extra liquids (10–12 glasses a day). Warm fluids, such as tea and soups, can increase the rate of mucus flow and provide some symptom relief.

Medications: Antibiotics are not prescribed for a cold, but they may be prescribed for a secondary infection. All cold medications are available over the counter. Please consider the side effects of these products, as side effects can be greater than any benefit. If you choose to use them, take as directed.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. You experience pain that is getting worse in your ears, sinuses, throat, neck, or chest.
B. You have green or yellow nasal drainage.
C. Your temperature is higher than 100.4°F.
D. You are a diabetic and your blood sugars are elevated, or you notice ketones in your urine while you are sick.
E. Other: _____

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CLIENT TEACHING GUIDE

COUGH

PROBLEM Coughing is an important defense mechanism your body uses to clear your airways of mucus and inhaled particles.

CAUSE A cough is often associated with other respiratory symptoms and may be a sign of infection. Coughing is often related to environmental or chemical irritants such as smoking.

PREVENTION

A. The flu vaccine is recommended each year; however, the flu vaccine does not prevent a cough.
B. Coughing cannot be prevented, but you do have some voluntary control over it.
C. Occasionally, medications can cause a cough. Therefore, review all medications with your healthcare provider.

TREATMENT PLAN

A. Stop smoking, including exposure to secondhand smoke. At the minimum, maintain a smoke-free bedroom.
B. Using a room humidifier may be helpful. Keep your humidifier clean—it can grow bacteria.
C. Change heating and air conditioning filters often to reduce environmental irritants.
D. Coughing for several minutes may tire you, so you may need extra rest.

Activity: No activity restrictions.

Diet: Drink at least 10 to 15 glasses of liquids a day.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. You have difficulty breathing.
B. You cough up blood.
C. You develop other symptoms besides coughing, such as green sinus drainage and/or a sore throat.
D. You cannot sleep due to coughing.
E. You develop a fever over 101°F.
F. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

EMPHYSEMA

PROBLEM Emphysema is a chronic lung disease and is incurable. Emphysema can only be managed; the goal of treatment is to improve the activities of daily living and the quality of life by preventing symptoms and by preserving optimal lung function.

CAUSE Cigarette smoking increases the risk of chronic obstructive pulmonary disease (another name for several lung diseases) by about 30 times. *You should also be given* the *Client Teaching Guide "Nicotine Dependence."*

PREVENTION Emphysema cannot be prevented once lung changes have taken place.

TREATMENT PLAN

A. Stop smoking—it causes more lung irritation, mucus/sputum production, and coughing. It is never too late to quit smoking.
B. Get a flu shot every year.
C. There are two types of pneumonia vaccinations to reduce your risk for pneumonia.
D. One of the pneumonia shots can be given at the same time as the flu shot.
E. If you are prescribed theophylline to help control your respiratory systems, your blood level needs to be monitored.
F. Eliminate other lung irritants, such as wood smoke, secondhand smoke, hairspray, paint, bleach, and other chemicals found at home. Avoid sweeping and dusting, and stay indoors when air pollution or pollen counts are high.
G. Pulmonary rehabilitation may be ordered. Exercising is a very important component of pulmonary rehabilitation, as well as learning breathing techniques.
H. Report respiratory infections to your healthcare provider as soon as possible.
I. Use postural drainage: Lean over the side of the bed, rest your elbows on a pillow placed on the floor, and cough as someone gently pounds on your back.
J. Stay indoors during extremely hot or cold weather. If you must be outside in the cold, cover your nose and face. Use an air conditioner in hot weather.
K. Avoid people who have respiratory illnesses; also avoid crowds and poorly ventilated areas.
L. Oxygen therapy may be ordered if you have trouble breathing.
M. Use community resources such as Meals on Wheels, a handicap parking tag, or parking stickers.
N. You may be asked to talk to a social worker.

Activity:
A. Pace yourself to avoid shortness of breath.
B. Follow a daily exercise plan. Start with three to four times a day, each lasting 5 to 15 minutes. Start at half-speed and build up.
C. Sexual dysfunction can occur due to lack of physical energy and trouble breathing. Find other ways to show affection, such as kissing, hugging, or massage.

Diet:
A. If you do not have congestive heart failure, drink 3 L of fluid a day—equal to one-and-a-half large soda bottles.
B. Avoid dairy products; they increase mucus/sputum production.
C. Eat five to six small meals a day. Big meals feel like pressure on your stomach and lungs.
D. Avoid foods that cause gas and stomach discomfort.
E. Use oxygen during meals, if necessary; take your time eating, rest between bites, and avoid hard-to-chew foods because eating may tire you. Rest before and after eating if you have shortness of breath.
F. Eat a high-protein diet with a good balance of vitamins and minerals.
G. Avoid excessively hot or cold foods and drinks that may start an irritating cough.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

(*continued*)

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CLIENT TEACHING GUIDE

EMPHYSEMA (*CONTINUED*)

Use of a spacer/chamber device improves aerosol delivery to your lungs and reduces side effects. ***You should also be given the Client Teaching Guide "How to Use a Metered-Dose Inhaler."***

You Need to Notify the Office If:

A. You have trouble breathing.
B. You develop an infection (signs are fever, change in sputum, sinus drainage).
C. Your inhaler does not help your symptoms.
D. Your symptoms do not improve within 48 hours of starting medication.
E. Other: _____

Phone: _____

You should have a follow-up appointment in 3 months unless you are worsening.

If you are prescribed theophylline, your blood level needs to be monitored whenever you have a new illness, worsening chronic illness, or a change in medications that may alter theophylline clearance (e.g., fever >102°F sustained for ≥24 hours, hepatitis). Call 911 and get emergency medical help for severe headache, high blood sugars, severe vomiting, irregular heartbeats, seizures, or confusion.

RESOURCE A Patient's Guide to Aerosol Drug Delivery: http://www.copd-alert.com/AerosolDrug.pdf

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CLIENT TEACHING GUIDE

HOW TO USE A METERED-DOSE INHALER

USING AN INHALER To receive the proper dose from your inhaler, you must use good technique. Your healthcare practitioner may provide you with a drug-free practice inhaler. Practice the following steps until you are comfortable administering your inhalant:

A. Shake the inhaler well immediately before each use.

B. Using a spacer helps to deliver more medication to your lung (not just the back of your mouth).

C. Remove the cap from the mouthpiece. Hold the inhaler upright. Make sure the medication canister is firmly inserted into the plastic holder (actuator).

D. The first time you use your new inhaler (or if it has been 1 month or longer since the your last use), test-spray four times into the air.

E. Breathe out through your mouth to the end of a normal breath.

F. Position the mouthpiece about 1 to 2 in. in front of your open mouth. Or you may close your lips in a tight seal around the mouthpiece.

G. Open your mouth widely (unless you are using the second method) and position your head in a neutral position.

H. While breathing in slowly and deeply, firmly depress the container once.

I. Continue breathing in slowly until your lungs are full.

J. Once you have breathed in fully, hold your breath for 10 seconds or as long as you can.

K. If you need a second puff of the same medication, wait a minimum of 1 minute before repeating steps A through J. If you are using a different inhaler for the second puff, wait at least 5 minutes before using the second inhaler.

OTHER TIPS

A. If you are taking a steroid inhalant, rinse your mouth and throat with water after each dose.

B. When you are short of breath, use your bronchodilator ("rescue medicine") first; then wait about 5 minutes before using your steroid inhaler. The rescue inhaler opens your airways so more of the steroid medication reaches your lungs.

C. Keep the inhaler clean. Once a week, remove the medication canister from the actuator and wash the actuator in warm, soapy water. Rinse and allow to air-dry. Replace the medication canister in the holder and re-cap the mouthpiece.

D. Always check the expiration date on your inhaler and make sure to refill your prescription before the medication expires.

E. Color-code your inhalers with tape or markers; for example, use green tape for quick-relief inhalers and blue tape for long-acting inhalers.

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INFLUENZA (FLU)

PROBLEM Influenza (flu) is an acute, self-limiting, febrile illness of the respiratory tract. You are contagious for 24 to 48 hours before feeling the symptoms, and you are contagious up to 7 days after symptoms begin. Coughing and sneezing spread the flu.

CAUSE There are many flu viruses. Stress, excessive fatigue, poor nutrition, recent illness, crowded places, and immunosuppression from drugs or illness lower your resistance to these viruses.

PREVENTION

A. Although the flu vaccine neither prevents nor causes the flu, the flu vaccine is recommended every year.
B. Avoid unnecessary contact with sick persons, including in crowded areas.
C. To keep the flu from spreading:
  1. Cover your mouth when coughing or sneezing.
  2. Use tissues when you blow your nose. Dispose of them and then wash your hands.
  3. If no tissue is available, do the "elbow sneeze" into the bend of your arm/shirt sleeve (away from your open hands).
  4. Do not share drinking glasses.
  5. Wash your hands with soap and water or use hand sanitizer.

TREATMENT PLAN

A. Rest.
B. Drink lots of fluids.
C. Run cool-mist vaporizer.
D. Take tepid sponge baths in warm water to prevent chilling and shivers.
E. Gargle with warm saltwater for a sore throat.
F. Use warm compress or heating pad on low for aching muscles.

Activity: Stay in bed for at least 24 hours after your fever is gone.

Diet: You may not be hungry, but you do not need to be on a special diet for the flu. Drink plenty of liquids (at least 10 glasses a day).

Medications:
A. Antibiotics do not help the flu since it is a virus.
B. Special medications shorten the flu. They must be started within 2 days of contracting the flu.

You Have Been Prescribed to Shorten Your Flu Symptoms: _____

You Need to Take: _____

You Have Been Prescribed for Your Fever: _____

You Need to Take: _____

You Have Been Prescribed for Your Respiratory Symptoms: _____

You Need to Take: _____

You Have Been Prescribed a Cough Suppressant: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. Thick, green nasal drainage.
B. Ear pain.
C. Increase in fever or cough.
D. Shortness of breath or chest pain.

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CLIENT TEACHING GUIDE

INFLUENZA (FLU) (*CONTINUED*)

E. Blood when you cough.
F. Neck pain or stiffness.
G. New or unexplained symptoms.
H. No need to urinate (longer than 8 hours while awake without the need to urinate).
I. Diabetes and your blood sugars are elevated, or you notice ketones in your urine while you are sick.
J. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

PNEUMONIA, VIRAL

PROBLEM Viral pneumonia is an infection of the lung that causes fluid in the air sacs. You may have fever, cough, or difficulty breathing.

CAUSE Respiratory viruses cause viral pneumonia and bacteria.

PREVENTION

A. Although the flu vaccine does not prevent pneumonia, a yearly flu shot is recommended.
B. There are two types of pneumonia vaccines. The pneumonia vaccines help prevent pneumonia caused by bacteria. If you are older than 65 years, smoke, and have chronic respiratory illness or certain medical conditions, the pneumonia vaccines are recommended.
C. You may be given one of the pneumonia vaccines at the same time as you get the flu vaccine.
D. Avoid contact with people with respiratory illnesses.

TREATMENT PLAN

A. Use a cool-mist humidifier and clean it daily.
B. Take deep breaths and cough frequently to clear secretions from the lungs.
C. Avoid smoking and exposure to secondhand smoke.
D. Cover your mouth when you cough and cover your nose when you sneeze.
E. Use tissues when you blow your nose. Throw away tissues as soon as they are used.
F. If no tissue is available, do the "elbow sneeze" into the bend of your arm/sleeve of your shirt.
G. Practice good handwashing techniques with soap and water or use of hand sanitizers.

Activity: Rest frequently during the early phase of the illness. Fatigue may continue for up to 6 weeks.

Diet: Eat a nutritious diet. Drink to 10 glasses of water a day.

Medications:

A. Take acetaminophen (Tylenol) for fever, discomfort, and headache.
B. Do not take cough suppressants. It is important for you to cough and get up any mucus.
C. Antibiotics are not given for a viral infection. If you have a bacterial infection, then you may be put on an antibiotic.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Increased difficulty breathing.
B. Fever over 101°F or fever that persists after 48 hours of antibiotics.
C. Worsening discomfort.
D. Shortness of breath.
E. Blood in sputum.
F. Nausea, vomiting, or diarrhea.
G. Diabetes and your blood sugars are elevated, or you notice ketones in your urine while you are sick.
H. Other: _____

Phone: _____

You should be seen for a follow-up appointment on: _____

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CLIENT TEACHING GUIDE

SLEEP APNEA

PROBLEM You have been diagnosed with sleep apnea. During sleep, your tongue and throat relax, causing less air to go down into your lungs. When you sleep and snore, you have short periods when you stop breathing and may wake up gasping for breath. Your bed partner may shake you awake because of your loud snoring or may notice that you have stopped breathing. The sleep apnea makes you very tired, leading you to take daytime naps.

CAUSES

A. Being overweight and having a "thick neck" can cause sleep apnea.
B. As you get older, you are more likely to have sleep apnea.
C. After menopause, females may start having sleep apnea.
D. If you have not had your tonsils removed, they may be part of the problem as your tongue and throat relax.
E. Alcohol and some medicines cause sleep apnea.
F. Allergies may also cause some sleep apnea.

PREVENTION

A. There is no way to prevent sleep apnea once you have it.
B. One way to help your sleep apnea is to lose weight.

TREATMENT PLAN

A. Review all of your medicines and any herbal products with your healthcare provider.
B. You may be told to sleep with your head up on two pillows, use a tennis ball under your pillow, or even sleep with a backpack under your pillow. This holds your head up higher and prevents your tongue from making your windpipe smaller.
C. You may also need a mouthpiece to help your tongue from relaxing when you sleep. Your dentist will need to help you find the best one.
D. You may be sent to a lung specialist to help with your treatment.
E. You may be told you need to have a sleep study to check out and measure how often you stop breathing at night.
F. After your sleep study, your lung specialist may order you to have a sleep machine called continuous positive airway pressure (CPAP) or biphasic positive airway pressure (BiPAP).
    1. The sleep machine uses nose tubing, a nasal mask, or a full face mask.
    2. The machine is small and portable.
    3. Your CPAP machine can be taken through the airport.
    4. You will have a supplier send you more tubing, machine filters, and breathing tubes/masks on a regular schedule.
G. Continuing to use the prescribed treatment for sleep apnea is important to your overall health.

Activity:
A. You will continue to need to sleep with your head higher, use the mouthpiece, or use the sleep machine to keep from having sleep apnea.
B. Exercise; losing even a small amount of weight will help with sleep apnea.
C. Stop smoking. You should be given the *Client Teaching Guide "Nicotine Dependence."* **It is never too late to quit smoking**.
D. Avoid alcohol, especially near your bedtime.

Diet: There is no special diet for sleep apnea, but losing weight helps.

Medications: There are no medicines for sleep apnea.

You Need to Notify the Office If:
A. Your bed partner complains that your snoring or sleep apnea has worsened.
B. You want to make an appointment to discuss having a sleep study.
C. You are having difficulty with your sleep machine or other equipment.
D. Other: _____

Phone: _____

RESOURCE National Sleep Foundation: www.sleepfoundation.org.

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CHAPTER 13

CARDIOVASCULAR GUIDELINES

Debbie A. Gunter and Laura A. Petty

ACUTE MYOCARDIAL INFARCTION

DEFINITION

A. Acute myocardial infarction (MI) is a prolonged lack of myocardial oxygenation leading to necrosis of a portion of the heart muscle. It is caused by atherosclerotic coronary artery disease (CAD), which alone or in association with other factors causes complete blockage of one of the coronary arteries.

INCIDENCE

A. The estimated annual incidence of heart attack in the United States is 605,000 new attacks and 200,000 recurrent attacks. The average age at the first heart attack is 66 years for males and 70 years for females. Approximately every 42 seconds, an American will have a heart attack. Despite a marked decrease in incidence and mortality during the past three decades, MI continues to be the leading cause of death in this country, accounting for one-fourth of all fatalities. More females die from heart disease than males.

PATHOGENESIS

A. Abrupt coronary artery occlusion is the primary cause of most MIs. Occlusions can result from atherosclerotic plaque, intracoronary thrombus formation, or arterial spasm.

PREDISPOSING FACTORS

A. Hypercholesterolemia: increased low-density lipoprotein (LDL) and decreased high-density lipoprotein (HDL).
B. Hypertriglyceridemia.
C. Premature familial onset of coronary heart disease (CHD), formerly called CAD, before the age of 55.
D. Smoking.
E. Hypertension (HTN).
F. Obesity.
G. Sedentary lifestyle.
H. Diabetes mellitus.
I. Aging.
J. Stress.

COMMON COMPLAINTS

A. Primary complaint: pain somewhere in the chest, often described as worst pain ever experienced.
B. Nausea.
C. Vomiting.
D. Diaphoresis.
E. Indigestion.

OTHER SIGNS AND SYMPTOMS

A. Pain in abdomen, arm, back, jaw, and neck.
B. Chest heaviness or tightness.
C. Anxiety.
D. Cough.
E. Dyspnea.
F. HTN or hypotension.
G. Weakness, light-headedness, syncope.
H. Pallor.
I. Orthopnea.
J. Fatigue.
K. Malaise.

POTENTIAL COMPLICATIONS

A. Arrhythmias.
B. Heart failure (HF).
C. Cardiogenic shock.
D. Rupture of left ventricular (LV) papillary muscle.
E. Ventricular septal rupture.
F. Pericarditis or Dressler syndrome.
G. Ventricular aneurysm.
H. Thromboembolism.
I. Death.

SUBJECTIVE DATA

A. Ask the client if they were participating in some type of activity and what it was that brought about or preceded the episode of chest pain.
B. Ask the client to describe the duration of pain and what time of day symptoms began.
C. Ask the client to describe pain, for example, crushing, stabbing, or burning.
D. Ask the client where sensation began and in what direction it radiates.
E. Identify the degree of pain by using a pain scale of 0 to 10, with 0 being no pain.
F. Ask the client to list all medications currently being taken, particularly substances not prescribed and illicit drugs such as cocaine.

PHYSICAL EXAMINATION

A. Clients presenting with acute chest pain should be quickly assessed for the need to call emergency services/911 for immediate transport to the hospital.
B. Check pulse, respirations, blood pressure (BP), and pulse oxygenation.
C. Inspect:
1. Inspect general appearance, noting dyspnea and weakness.
2. Inspect skin for pallor and diaphoresis.
3. Inspect legs for edema.
4. Inspect chest wall for visible pulsations.
5. Inspect neck for jugular vein distension.
6. Observe nail beds for signs of cyanosis and note capillary filling time. ▶

D. Palpate:
1. Palpate abdomen for organomegaly.
2. Palpate peripheral pulses in legs.
3. Palpate femoral pulses.
E. Auscultate:
1. Auscultate carotid arteries.
2. Auscultate abdomen.
3. Conduct a complete heart exam, checking for dysrhythmias.
4. Conduct a complete lung exam.
F. Mental status:
1. Assess for confusion and anxiety.

DIAGNOSTIC TESTS

A. EKG shows inverted T waves, ST-segment elevation, and Q waves. **One normal EKG initially does not always rule out MI; perform serial EKGs if MI is suspected.**
B. Laboratory testing:
1. Cardiac biomarkers/enzymes.
2. Troponin level: Troponin is a protein that is released when necrosis of the cardiac muscles occurs.
3. Creatine kinase (CK): Creatine kinase-muscle/brain (CK-MB) levels increase 3 to 12 hours after the chest pain begins, peak at 24 hours, and return to normal in 48 to 72 hours.
4. Myoglobin: Urine myoglobin levels rise 1 to 4 hours after the chest pain begins.
5. Complete blood count (CBC).
6. Chemistry profile.
7. Lipid profile.
8. C-reactive protein (CRP) and inflammatory markers.
C. Cardiac imaging:
1. Coronary angiogram.

DIFFERENTIAL DIAGNOSES

A. Acute MI.
B. Unstable angina pectoris.
C. Aortic dissection.
D. Pulmonary embolism (PE).
E. Pericarditis.
F. Esophageal spasm.
G. Pancreatitis.
H. Biliary tract disease.

PLAN

A. General interventions:
1. Educate the client and the family regarding the signs and symptoms of an acute MI.
2. Long-term care and treatment should be reinforced at each client visit.
B. Client teaching:
1. Educate the client about modifying controllable risk factors, such as keeping diabetes and HTN under control, diet, exercise, and smoking cessation.
2. If known CHD is present:
a. Instruct the client on signs and symptoms of an acute MI.
b. Advise the client to have a plan in seeking medical attention or dialing 911 if signs and symptoms occur.
c. Advise the client to carry nitroglycerin at all times and to take the nitroglycerin at the first sign of chest pain. If there is no relief after 5 minutes, 911 should be called. Nitroglycerin may be repeated every 5 minutes times three doses.
d. Encourage CPR training for family members and close friends.
e. Exercise regimen: Encourage routine exercise for the client most days of the week, such as walking, treadmill use, and so forth, once released by the cardiologist.
f. Advise smoking cessation as indicated. Encourage support groups, classes, and smoking cessation aids as indicated. *See Client Teaching Guide for Chapter 25, "Nicotine Dependence."*
C. Dietary management:
1. Counsel the client on nutrition and low-fat, low-cholesterol, and low-sodium diets. Recommend Dietary Approaches to Stop Hypertension (DASH) diet and lifestyle changes. Provide dietary handouts on the DASH diet and a low-fat/low-cholesterol/low-sodium diet. See Appendix B for the DASH diet.
E. Pharmaceutical therapy:
1. When MI is suspected, give aspirin 160 to 325 mg (four 81-mg baby aspirin) chewed or swallowed as soon as possible. Enteric-coated aspirin delays absorption and is therefore not recommended.
2. Instruct the client on how to take sublingual nitroglycerin tablets and other medications.
3. Nitrates: Give nitroglycerin sublingually 0.2 to 0.6 mg every 5 minutes for ischemic chest pain in the absence of hypotension.
4. If pain persists after three doses of nitroglycerin:
a. Morphine sulfate intravenously (IV) 2 to 4 mg repeating every 5 minutes until pain resolves. Dose may be increased to 2 to 8 mg per dose as tolerated. Monitor side effects such as nausea/vomiting, dizziness, hypotension, and respiratory depression.
5. Oxygen therapy: 2 to 4 L per nasal cannula.
6. If there are no contraindications (bradycardia, HF, second- or third-degree heart block, asthma, shock), beta-blockers may be started IV during the acute phase and changed to oral therapy during the course of treatment.
7. Fibrinolytic therapy may be used for clients with suspected MI with ST-elevation myocardial infarction (STEMI) or non-ST-elevation myocardial infarction (NSTEMI) with left bundle branch block.
8. After an MI, if the client is not currently on a statin, a statin should be started.

FOLLOW-UP

A. Follow-up is determined by client's needs, severity of acute MI, and whether complications are present.

EMERGENT ISSUES/INSTRUCTIONS

A. If MI is suspected, refer client for immediate hospitalization.
B. Administer four aspirin 81 mg swallowed or chewed while waiting for emergency medical service (EMS).

CONSULTATION/REFERRAL

A. According to the American College of Cardiology/American Heart Association (ACC/AHA) guidelines for management of a client who is a candidate for reperfusion who is seen at a percutaneous coronary intervention (PCI)-capable hospital, this client should be sent to the cath lab for primary PCI in less than or equal to 90 minutes. If the client is at a facility that is non-PCI capable, then a transfer should be made to a PCI facility as soon as possible and in less than or equal to 120 minutes. If the time lapse will be greater than 120 minutes, then it is recommended to administer fibrinolytic therapy within 30 minutes of arrival.
B. Follow up with cardiologist as scheduled when discharged from the hospital.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. The approach to older adults with MI is similar to that of younger adults with the following considerations:

a. NSTEMI is more common than STEMI in older adults. Recurrent MI and post-MI HF are more common in the geriatric population.

b. **Geriatric adults are more likely to complain of dyspnea, fatigue, and dizziness, than typical coronary type chest pain, and may present with altered mental status or shortness of breath.**

c. Even when classic ischemic precordial discomfort is present, it tends to be less severe and less well-defined.

d. Older clients are more likely to have "silent" MIs, often delaying diagnosis and hospital admission for this cohort.

2. Pharmacotherapy for geriatric clients post-MI is similar to treatment strategies aimed toward younger individuals with the following considerations, taking into account normal age-related declines in renal function:

a. An older adult (over age 65) treated for an acute coronary syndrome (MI or angina) should discontinue nonsteroidal anti-inflammatory drugs (NSAIDs). NSAIDs (other than aspirin) as these are independent risk factors for cardiovascular disease.

b. **Aspirin is no longer recommended as primary prevention for older adults.** Unless contraindicated, older adults who have had an MI should take aspirin, a beta-blocker, and an angiotensin-converting enzyme inhibitor (ACEI).

c. Long-acting nitrates (e.g., isosorbide mononitrate) or long-acting calcium channel blockers (CCBs) may be used for chronic angina if beta-blockers are contraindicated.

3. Beers criteria precautions:

a. Use nitrates with caution due to an increased risk for syncope.

b. Avoid nondihydropyridine CCBs in HF with reduced ejection fraction due to increased risk of fluid retention and exacerbation of HF.

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ARRHYTHMIAS

DEFINITION

A. Arrhythmias are abnormal heart rhythms. The following are the common types:

1. Bradycardia: heart rate less than 60 beats per minute (bpm); impulse originates in the sinoatrial (SA) node.

2. Tachycardia: heart rate greater than 100 to 160 bpm; impulse originates in the SA node.

3. Supraventricular tachydysrhythmias (SVTs): heart rate greater than 100 bpm; originates in the following:

a. Atrioventricular nodal reentrant tachycardia (AVNRT), which is an intranodal reentry by means of fast and slow conduction pathways within the atrioventricular (AV) junction.

b. Orthodromic atrioventricular reentrant tachycardia (AVRT), which is a tachycardia across accessory pathways associated with preexcitation.

4. Atrial fibrillation (AF): chaotic electrical activity caused by rapid discharges from numerous ectopic foci in atria. Atrial rate is difficult to count. There are three types of AF:

a. Paroxysmal AF occurs in clients who usually have normal sinus rhythm (NSR). There may be an episode of faulty electrical signals and rapid heart rate. This usually starts suddenly and stops on its own. Symptoms can be mild or severe and usually stop in less than 24 hours, but may last for several days.

b. Chronic AF occurs in clients who have a permanent fibrillation rather than brief episodes of symptoms.

c. Premature ventricular contractions (PVCs) are impulses that form within the Purkinje network (see the "Atrial Fibrillation" section of this chapter).

INCIDENCE

A. SVTs are the most common cardiac arrhythmias presenting to healthcare providers.

B. AVNRT accounts for 60% to 70% of all SVTs.

C. AVRT accounts for 30% to 40%.

D. AF is the most common cardiac tachydysrhythmia, affecting approximately 2% of the general population. Prevalence increases with age to 5% of those older than age 69. The presence of AF is associated with a fivefold increase in risk of morbidity, a twofold increase in mortality, and an increased incidence of embolic stroke.

E. PVCs are common and their frequency increases with age.

PATHOGENESIS

A. Bradycardia: Dominance of the parasympathetic nervous system, with excessive vagal stimulation to the heart, causes decreased heart rate of sinus node discharge.

B. Tachycardia: Dominant sympathetic nervous system stimulation of the heart or vagal inhibition results in positive chronotropic, dromotropic, and inotropic effects.

C. AVRT: The most classic form of this SVT is Wolff–Parkinson–White (WPW) syndrome. Reentry occurs in a loop using atrial myocardium, the AV node–His–Purkinje system, ventricular myocardium, and an accessory AV connection. During sinus rhythm, antegrade conduction through the accessory connection depolarizes the myocardium earlier than would occur by conduction through the AV node–His–Purkinje system, preexcitation is present, and a delta wave (slurring of initial deflection of the QRS complex) is usually seen on the surface 12-lead EKG. ▶

D. AVNRT: The most common form is antegrade conduction, which occurs through a pathway with a short effective refractory period (ERP) and a longer conduction time. This pathway is often referred to as the slow pathway.

E. AF: Multiple, rapid impulses from many foci depolarize the atria in a totally disorganized manner. In the chaos, no P waves, no atrial contraction, loss of atrial kick, and a totally irregular ventricular response occur. The atria quiver, which leads to formation of mural thrombi and potential embolic events.

F. PVCs: These originate in the ventricles as a result of increased irritability in those cells.

PREDISPOSING FACTORS

A. Bradycardia:
- **1.** Increased vagal tone.
- **2.** Decreased sympathetic drive.
- **3.** Ischemia to SA node.
- **4.** Drugs: digitalis, propranolol, sedatives, propylthiouracil (PTU) or Tapazole, aminophylline, caffeine, alcohol, nicotine, and sympathomimetics.
- **5.** Normal variant in athletes.
- **6.** Normal body response to insult.
- **7.** Atrial enlargement.
- **8.** Acute myocardial infarction (MI).
- **9.** Congestive heart failure (CHF).
- **10.** Rheumatic heart disease.
- **11.** Hypertensive heart disease.
- **12.** Thyroid disease.
- **13.** Hypothermia.
- **14.** Electrolyte abnormality.
- **15.** Acidosis.
- **16.** Infection.

B. Tachycardia:
- **1.** Decreased vagal tone.
- **2.** Increased sympathetic tone.
- **3.** MI.

C. SVT:
- **1.** Digitalis toxicity.
- **2.** Catecholamines.

D. AF:
- **1.** Myocardial ischemia.
- **2.** Thyrotoxicosis.

E. PVC:
- **1.** Stress.
- **2.** Myocardial ischemia.

COMMON COMPLAINTS

A. Symptoms may not be present; however, the client may note irregular heartbeat.

B. Palpitations.

C. Chest discomfort.

D. Shortness of breath (SOB).

E. Dizziness.

F. Diaphoresis.

G. Weakness.

H. Syncope.

I. Nausea.

SUBJECTIVE DATA

A. Obtain an accurate health and medical history.

B. Explore precipitating factors, such as emotional stress, alcohol or drug use, and hot tub or bath use.

C. Inquire about the onset and duration of symptoms, also noting the age of the client when symptoms began.

D. Explore whether the client has had concomitant weight loss, mood changes, and tremor, which are often associated with hyperthyroidism.

E. Carefully determine the number of previous episodes of palpitations or symptoms and what treatment, if any, was initiated.

F. Review all medications including prescription, over-the-counter (OTC), and herbal products.

PHYSICAL EXAMINATION

A. Check pulse, respirations, blood pressure (BP), and weight.
- **1.** Sinus bradycardia: pulse rate decreased.
- **2.** EKG: normal.
- **3.** Sinus tachycardia:
  - a. Pulse regular.
  - b. Systolic blood pressure (SBP) constant.
- **4.** AF:
  - a. Pulse irregular.
  - b. SBP changing.
- **5.** AVNRT:
  - a. Pulse regular; AV block usual.
  - b. SBP constant; electrical alternans rare.
- **6.** AVRT:
  - a. Pulse regular; AV block not present.
  - b. SBP constant; electrical alternans common, especially at high heart rates.
- **7.** PVC: pulse diminished or absent.

B. Inspect:
- **1.** General appearance:
  - a. Is the client in respiratory distress? Note SOB, chest pain, dyspnea.
  - b. Does the client look apathetic? This is a sign of a thyroid problem.
- **2.** Inspect the skin for flushing or pallor.
- **3.** Examine the eyes, noting lid lag.
- **4.** Assess the neck for jugular vein distension or thyromegaly:
  - a. With sinus tachycardia, neck vein pulsation is normal.
  - b. With AF, neck vein pulsation is irregular; assess for thyromegaly.
  - c. AVNRT: Assess the neck veins for "frog sign," in which the atria contract against closed AV valves, producing rapid, regular, expansive venous pulsation in the neck, resembling the rhythmic puffing motion of a frog.
  - d. AVRT: Assess for frog sign.

C. Auscultate:
- **1.** Auscultate the neck for carotid artery bruits and the heart for abnormal heart sounds. Heart rhythm may be regular or irregular, depending on type of dysrhythmia. Have the client perform a vagal maneuver (Valsalva maneuver). If the vagal maneuver responds to rapid heart rate and the cycle is broken, it is likely the client has AVRT. If it does not respond, it is possible the client has AVNRT.
  - a. Sinus bradycardia:
    - i. Rate less than 60 bpm.
    - ii. Rhythm regular.
  - b. Sinus tachycardia:
    - i. Rate equals 160 to 200 bpm.
    - ii. Rhythm regular; gradual onset and cessation.
    - iii. Constant loudness of first heart sound.
  - c. AF:

▶

i. Rate: Atrial rate is nonmeasurable and ventricular rate is variable, usually rapid at onset.
ii. Rhythm: Atrial and ventricular rhythms are irregular.
iii. Loudness of first heart sound changes.

d. AVNRT:
i. AV block is usually present.
ii. Loudness of first heart sound is constant.

e. AVRT:
i. AV block is not present.
ii. Constant loudness of first heart sound.

f. PVC:
i. Rate depends on underlying rhythm.
ii. Rhythm: Prematurity interrupts regularity of the rhythm.

DIAGNOSTIC TESTS

A. EKG.
B. Drug screen:
1. Digitalis.
2. Aminophylline.
3. Illicit drugs.
C. Electrolytes.
D. Arterial blood gas (ABG) if indicated.

DIFFERENTIAL DIAGNOSES

A. Multifocal atrial tachycardia.
B. Sinus tachycardia with multiple premature atrial contractions.
C. Atrial flutter.
D. Ventricular tachycardia.
E. AV blocks.

PLAN

A. General interventions:
1. Remove as many predisposing factors as possible.
2. Stop smoking.
B. Client teaching:
1. Teach relaxation techniques.
2. Teach the client and their family signs of hemodynamic compromise, including rapid heart rate, unexplained weight gain, worsening dyspnea on exertion, and decreased exercise tolerance.
3. Teach and reassure the client about long-term medication therapy and its side effects.
4. Educate the client and their family regarding safety, dietary restrictions, and complications that may occur (bleeding) with the use of anticoagulant therapy.
5. Discuss the need for a pacemaker/defibrillator or surgical ablation.
C. Pharmaceutical therapy:
1. Initial treatment is usually prescribed by a physician.
2. Selection of treatment modality should be based on the underlying pathophysiology.
3. For reentrant cases (AVNRT, AVRT), agents that block the reentrant circuit are more effective.
a. Digitalis.
b. Beta-blockers.
c. Calcium channel blockers (CCBs).
4. Episodes caused by increased automaticity are treated with the following:
a. Quinidine gluconate (Quinaglute).
b. Procainamide HCl (Procan SR).
c. Disopyramide phosphate (Norpace CR).
5. Chronic AF is treated with anticoagulants such as warfarin sodium (Coumadin).
a. Start therapy as soon as possible if a history of underlying heart disease is present.
b. Evaluate prothrombin time (PT)/international normalized ratio (INR) on a regular basis to monitor for therapeutic response to warfarin sodium treatment.

FOLLOW-UP

A. Clients who have their first episode of AF should return to the clinic within 24 to 48 hours for reevaluation.
B. Clients on antiarrhythmic agents should have liver enzymes measured during the first 4 to 8 weeks of therapy.
C. Clients with risk factors for developing cardiac complications to therapy should have EKG during the first weeks of therapy and every 3 to 6 months thereafter.
D. Monitor clients on digoxin carefully for digitalis toxicity.
E. Caution the clients regarding interactions of medications with digitalis.

CONSULTATION/REFERRAL

A. Consult a physician when questions arise about the difference between a narrow and wide QRS complex.
B. Consult a physician if the client has an abnormal EKG pattern, refractory AF, suspicion of WPW syndrome, or sick sinus syndrome.

EMERGENT ISSUES/INSTRUCTIONS

A. Clients with chest pain and/or hemodynamic instability should be referred to an ED or call 911 immediately.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Digitalis is safe during pregnancy.
C. Geriatrics:
1. Over 50% of AF occurs in the percentage (6%) of the population older than 75 years of age.
2. The prevalence of AF increases with age.
3. Advanced age and female sex are potent risk factors for stroke.
4. Use the CHA_2DS_2-VASc risk calculator to estimate the risk of stroke in nonvalvular AF. In older adults with a CHA_2DS_2-VASc score less than 2, it is unlikely a stroke reduction benefit can be achieved with the use of oral anticoagulants. The risk of treatment may outweigh the benefit for those with a low CHA_2DS_2-VASc score. A CHA_2DS_2-VASc risk calculator is available at www.globalrph.com/chad2.htm.
5. Prior to prescribing anticoagulant therapy, it is important to assess bleeding risk in older adults. The HAS-BLED (Hypertension, Abnormal liver/ renal function, Stroke history, Bleeding history or predisposition, Labile INR, Elderly, Drug/alcohol usage) bleeding risk score can be used specifically when considering the use of warfarin (not validated in other anticoagulant therapy). It assesses the 1-year risk of a major bleed in clients with AF. A HAS-BLED risk calculator is available at www.globalrph.com/has-bled-score.htm.
6. Direct thrombin inhibitor anticoagulants provide alternatives to warfarin but are not without limitations. Most are related to renal function and an increased risk of bleeding:
a. Pradaxa (dabigatran) is listed in the Beers criteria to be used with caution in adults aged 75 or older and those with creatinine clearance (CrCl) less than 30 due to an association with an increased risk for ▶

gastrointestinal (GI) bleeds (refer to Appendix C, Table C.1).

b. Eliquis (apixaban): Use half the regular dose (2.5 mg BID) in those aged 80 and older who weigh less than 60 kg. In those with a CrCl greater than 1.5, use the half-regular dose if *either* over the age of 80 *or* if weight is less than 60 kg. Beers criteria: AVOID use in those aged 65 and older who have a CrCl less than 25 mL/min.

7. The routine use of mechanical interventions and devices in older AF clients remains to be clearly established. Left atrial appendage occlusion may be considered in clients with AF who are at increased risk of stroke and have a contraindication to anticoagulation.

8. Lenient rate control with concomitant anticoagulation is a reasonable therapeutic strategy in older individuals with limited symptoms.

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ATHEROSCLEROSIS AND HYPERLIPIDEMIA

DEFINITION

A. Atherosclerosis is a systemic disease characterized by lipid deposition and smooth muscle cell migration and proliferation in the intima of the larger arteries. Atheromatous changes lead to thrombotic stroke, peripheral vascular disease (PVD), atherosclerosis cardiovascular disease (ASCVD), and myocardial infarction (MI).

B. Hyperlipidemia is an elevation in serum lipoproteins and a major risk factor in the development of cardiovascular disease (CVD). The two main lipids in the blood are cholesterol and triglyceride. Cholesterol is a relatively insoluble lipid that is necessary for cell membrane formation, steroid and bile salt production, and the development of nerve sheaths. Cholesterol is composed of three clinically significant components: high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and very-low-density lipoprotein (VLDL). Triglyceride is found in VLDL particles, but its role in atherosclerosis is not clear.

C. The atherosclerotic buildup of lipids, cholesterol, calcium, and cellular debris within the intima of the blood vessels causes plaque formation, vascular remodeling, and acute and chronic obstruction of the lumen of the blood vessels, which in turn decreases blood flow, causing myocardial ischemia and decreased oxygen to other vital organs.

D. In 2019, the American College of Cardiology (ACC) and the American Heart Association (AHA) published a revised and comprehensive set of guidelines on the primary prevention of CVD. A web-based calculator is available to help estimate 10-year and lifetime risks for ASCVD in individuals without a history of ASCVD (tools.acc.org/ASCVD-Risk-Estimator-Plus/#!/calculate/estimate/).These risk tools are used to drive conversations on client risk factors for ASCVD, potential benefits, negative aspects of risk, and client preferences regarding initiation of relevant therapies. The assessment of ASCVD risk factors is recommended every 4 to 6 years in adults 20 to 79 years of age who are free from ASCVD. Long-term and lifetime risk information may be used to motivate therapeutic lifestyle changes (TLCs) and to encourage adherence to these changes and pharmacologic therapies.

INCIDENCE

A. Atherosclerosis begins in childhood with the development of fatty streaks. The incidence of atherosclerotic diseases increases with age. CVD causes one in four deaths reported each year in the United States. The annual direct cost of CVD is estimated at $273 billion and the overall cost of CVD is estimated at $444 billion annually.

B. The leading risk factors for CVD are hypertension (HTN), high cholesterol, and smoking.

1. HTN can increase arterial wall tension, potentially leading to disturbed repair processes and aneurysm formation.

2. Cigarette smoking is associated with an increase in multiple inflammatory markers, including C-reactive protein (CRP), interleukin-6, and tumor necrosis factor.

PATHOGENESIS

A. Atherosclerosis is in part attributed to the deposition of cholesterol and lipoproteins in arterial smooth muscle cells. Dietary factors, obesity, drugs, and genetic defects in lipoprotein particle metabolism influence lipid and lipoprotein concentrations in blood.

B. Primary hyperlipoproteinemias are either due to single-gene disorders transmitted by simple dominant or recessive mechanisms, or to multifactorial disorders with complicated inheritance patterns.

C. Secondary hyperlipoproteinemias (such as in thyroid disease and diabetes mellitus) occur as part of a constellation of abnormalities in certain metabolic pathways. The association between atherosclerosis, CVD, and hypercholesterolemia is well-documented. HDL-C comprises about one-fourth of the total serum cholesterol and acts as a scavenger, removing cholesterol from peripheral tissues and returning it to the liver, which produces a favorable cardioprotective effect. Elevated HDL-C levels are desirable. HDL-C levels more than 60 mg/dL are a negative risk factor for CVD; those below 35 mg/dL are a major risk factor for CVD.

D. LDL-C constitutes 70% of the total serum cholesterol. It is the most atherogenic cholesterol subgroup. LDL-C particles interact with platelets, damaged arterial endothelium, and smooth muscle cells in the process of plaque formation. LDL-C levels of 160 mg/dL or greater are associated with an increased number of cardiac events.

E. VLDL accounts for a small amount of total serum cholesterol and is responsible for carrying triglycerides from the liver. Its role in atherogenesis is uncertain, but an inverse relationship has been observed between VLDL and HDL-C.

PREDISPOSING FACTORS

A. High-risk factors for CVD events (CVD risk equivalent):

1. Clinical CVD.

2. Symptomatic carotid artery disease.

3. Peripheral arterial disease (PAD).

4. Abdominal aortic aneurysm.

B. Presence of major risk factors (other than LDL-C):

1. Age is the strongest risk factor for the development of CVD.

a. Age older than 45 years for males and older than 55 years for females.
b. Older adults experience a higher morbidity and mortality.

2. Cigarette smoking.
3. Low HDL-C level, less than 40 mg/dL.
4. Family history of early CVD: MI or sudden cardiac death younger than 55 years in the father or other male first-degree relative, and before age 65 in the mother or other female first-degree relative.
5. HTN (blood pressure [BP] >140/90 mmHg or on antihypertensive medication).
6. Sedentary lifestyle.
7. Obesity.
8. Metabolic syndrome.
9. Diabetes.
10. Chronic inflammation.

HDL-C of greater than 60 mg/dL is equal to "negative" risk factor and removes one risk factor from the total count.

COMMON COMPLAINTS

A. There are no complaints or symptoms associated with atherosclerosis and hyperlipidemia. Most lipid abnormalities are detected by routine laboratory testing or as part of a cardiovascular evaluation.

SUBJECTIVE DATA

A. Ask the client if there is a history of CVD.
B. Discuss their medical history, including predisposing factors for CVD.
C. Have the client list current medications, including over-the-counter (OTC) and herbal products.
D. Have the client discuss their current diet and exercise routine.
E. Explore the client's social habits, including use of alcohol and tobacco.

PHYSICAL EXAMINATION

A. Check pulse, respirations, BP, height, and weight. Calculate body mass index (BMI) at each subsequent visit. An adult BMI calculator and teen BMI calculator are located at www.cdc.gov/healthyweight/assessing/bmi.
B. Inspect:
1. Perform funduscopic exam: Examine eyes for premature arcus cornealis, which is a gray opaque line around the cornea caused by lipoid degeneration, and for lipemia retinalis, which is a pale retina with white blood vessels caused by excess serum lipids due to VLDL of more than 2,000 mg/dL or alcoholism.
2. Inspect skin for xanthomas, which appear as red-brown or yellow papules, nodules, or plaque, caused by lipid deposits from high VLDL. Tendinous xanthomas are found on Achilles tendons, patellae, and hands.
3. Inspect joints for Achilles tendonitis and arthritis.
C. Palpate:
1. Palpate abdomen for hepatomegaly or splenomegaly.
2. Palpate neck and thyroid.
D. Auscultate:
1. Perform a complete heart exam.
2. Perform a complete vascular exam.

DIAGNOSTIC TESTS

A. Laboratory testing:
1. Lipid profile and lipoprotein analysis (Table 13.1).
2. Complete blood count (CBC).
3. Complete metabolic panel (CMP).
4. Thyroid function studies to exclude disorders of the thyroid.
5. CRP.
6. Hemoglobin A1C (if appropriate).
B. Tests/imaging:
1. Treadmill stress test.
2. Nuclear stress test.
3. Echocardiogram.
4. Ultrasound.
5. CT.
6. Coronary angioplasty.

DIFFERENTIAL DIAGNOSES

A. Assess client for the following secondary causes of ASCVD and hyperlipidemia:
1. Atherosclerosis.
2. Hyperlipidemia.
3. Diabetes mellitus.
4. Hypothyroidism.
5. Nephrotic syndrome.
6. Porphyria.
7. Obesity.
8. Obstructive liver disease.
9. Diuretic use.

TABLE 13.1 ATP III CLASSIFICATION OF LDL, TOTAL, AND HDL CHOLESTEROL (MG/DL)

Determine Lipoprotein Levels. Obtain Complete Lipoprotein Profile After 9- to 12-Hour Fast.	
LDL Cholesterol: Primary Target of Therapy	
<100	Optimal
100–129	Near optimal/above optimal
130–159	Borderline high
160–189	High
>190	Very high
Total Cholesterol	
<200	Desirable
200–239	Borderline high
>240	High
HDL Cholesterol	
<40	Low
>60	High
Triglycerides	
<149	Good
150–199	Borderline
>200	High
>500	Very high

ATP, Adult Treatment Panel; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

PLAN

A. General interventions:

1. TLCs, including exercise, diet, and weight management, are recommended for all clients.

2. Increased physical activity. The 2013 ACC/AHA guidelines on lifestyle management outline the newest physical activity recommendations advising adults to engage in 40 minutes of aerobic physical activity three to four times a week. The aerobic exercise should involve moderate-to-vigorous intensity to reduce BP, LDL-C, and non-HDL-C. Refer to Chapter 3 for further exercise recommendations from the *2008 Physical Activity Guidelines for Americans* for key guidelines for adults 18 to 64 years, older adults, people with chronic medical conditions, and adults with disabilities.

B. Dietary management:

1. Advise the client that diet modification is the first line of therapy for hyperlipidemia.

2. Explain the cholesterol-lowering diet. Give dietary recommendation sheets. See Appendix B for low-fat/low-cholesterol and the Dietary Approaches to Stop Hypertension (DASH) diets.

C. Client teaching: *See Client Teaching Guide for Chapter 25, "Nicotine Dependence."*

1. Weight reduction:

a. Explain that weight reduction in clients who are more than 20% over ideal body weight can lower LDL-C and triglyceride levels.

2. Other key dietary recommendations include the following:

a. Reduce intake of saturated fats and trans fats. Aim for 5% to 6% of calories from saturated fat.

b. Increase intake of poly and monounsaturated fats.

c. Increase intake of soluble fiber (psyllium supplement).

d. Limit intake of alcohol: one drink per day for females and two drinks per day for males.

e. Increase intake of plant stanols and sterols (1 oz of Promise Activ or Benecol spread per day).

f. Increase intake of omega-3 fatty acids from marine sources (salmon or tuna twice a week or supplements).

g. Follow the DASH, Mediterranean, or AHA diet.

h. Lower sodium intake. Consume no more than 2,400 mg/d of sodium. Further reduction to 1,500 mg/d of sodium is associated with greater reduction in BP.

D. Pharmaceutical therapy: **Use clinical judgment when deciding potential benefits, possible side effects, and costs of drug treatment**:

1. Drug of choice: HMG-CoA reductase inhibitors (statins):

a. Statins suppress the activity of the key enzyme in cholesterol synthesis in liver; they are highly effective in lowering LDL-C but can cause liver toxicity, myositis, rhabdomyolysis, and low.

b. There are four major groups that benefit from statins:

i. Individuals with presence of clinical ASCVD, including acute coronary syndromes, history of MI, stable or unstable angina, coronary or other arterial revascularization, stroke, transient ischemic attack (TIA), or PAD.

ii. Individuals with primary elevations of LDL-C greater than 190 mg/dL.

iii. Individuals 40 to 75 years of age with diabetes and LDL-C of 70 to 189 mg/dL and without clinical ASCVD (refer to Chapter 24 for statin therapy for diabetes).

iv. Individuals without clinical ASCVD or diabetes who are 40 to 75 years of age with an LDL-C of 70 to 189 mg/dL and an estimated 10-year ASCVD risk of 7.5% or higher.

c. **ASCVD events are reduced by using the maximum tolerated statin intensity in those groups shown to benefit. The expert panel defines intensity of statin therapy on the basis of the average expected LDL-C response to a specific statin and dose. The intensity levels are "high-intensity," "moderate-intensity," and "lower-intensity" statin therapy. Full statin treatment recommendations for primary and secondary prevention as well as the high/moderate/low-intensity statin therapy from the 2013 ACC/AHA guidelines on the treatment of cholesterol are available at www.ahajournals.org/doi/10.1161/JAHA.116.004909.**

d. Monitor liver function tests (LFTs) before therapy begins and 4 to 6 weeks after starting drug therapy. Then check at 6- to 12-month intervals or more frequently, if necessary.

e. Discontinue medications if abnormal lab values or adverse symptoms appear.

f. Use caution when using statins and other medications. Avoid concomitant drugs such as erythromycin, nicotine, azole antifungals, clofibrate, and gemfibrozil.

g. The use of statins in older adults over age 75 remains controversial. See the "Individual Considerations" section on geriatrics.

2. Category X drugs:

a. **Statins are category X drugs and are contraindicated in pregnancy.**

3. Nonstatin drug therapy (see Table 13.2 for nonstatin drugs that affect cholesterol):

a. Bile acid sequestrants bind bile acids in the gastrointestinal (GI) tract; lower moderately elevated LDL-C by 20%; and may cause constipation, bloating, and poor absorption of other drugs. Bile acid sequestrant therapy is not recommended if triglycerides are greater than 300 mg/dL.

b. Nicotinic acid or niacin: broad-spectrum lipid regulating agent. Niacin has been documented to exhibit anti-inflammatory properties (reduction of lipoprotein-associated phospholipase A2 and CRP). Discuss possibilities of flushing. Niacin is contraindicated in chronic liver disease and severe gout. The niacin-treated subjects in the Atherothrombosis Intervention in Metabolic Syndrome With Low HDL/High Triglycerides: Impact on Global Health (AIM-HIGH) clinical trial had a trend toward increased stroke incidence.

c. Fibric acid derivatives are highly effective in lowering triglycerides, and lower VLDL-C, cause modest reduction in LDL-C, and raise HDL-C.

i. The combination of niacin with other lipid-lowering drugs has been shown to reduce progression and promote regression of coronary and carotid atherosclerosis and improve clinical outcomes.

ii. Fibrates may cause GI distress, rash, pain, blurred vision, anemia, and gallstones.

iii. Fibrates may inhibit insulin and oral hypoglycemic absorption and potentiate oral anticoagulants. ▶

TABLE 13.2 NONSTATIN DRUGS AFFECTING LIPOPROTEIN METABOLISM

Drug Class	Agents and Daily Doses	Lipid/Lipoprotein Effects	Side Effects	Contraindications
Bile acid sequestrants	Cholestyramine (4–16 g), colestipol (5–30 g), colesevelam (1,875–3,750 mg)	LDL-C ↓15%–30% HDL-C ↑3%–5% TG no change or increase	GI distress Constipation Decreased absorption of other drugs	Absolute: • Dysbetalipoproteinemia • TG >400 mg/dL Relative: • TG >200 mg/dL
Nicotinic acid	Immediate-release (crystalline) nicotinic acid (1.5–3 g), extended-release nicotinic acid (Niaspan; 500 mg–2 g), sustained-release nicotinic acid (1–2 g)	LDL-C ↓5%–25% HDL-C ↑15%–35% TG ↓20%–50%	Flushing Hyperglycemia Hyperuricemia (or gout) Upper GI distress hepatotoxicity	Absolute: • Chronic liver disease • Severe gout Relative: • Diabetes • Hyperuricemia • Peptic ulcer disease
Fibric acid	Gemfibrozil (600 mg BID), fenofibrate (145–200 mg), clofibrate (500–2,000 mg)	LDL-C ↓5%–20% (may be increased in patients with high TG) HDL-C ↑10%–20% TG ↓20%–50%	Dyspepsia Gallstones Myopathy	Absolute: • Severe renal disease • Severe hepatic disease
PCSK9 inhibitors.	Alirocumab (75–150 mg Q 2 weeks or 300 mg Q 4 weeks), evolocumab (140 mg Q 2 weeks or 420 mg Q 4 weeks)	LDL-C ↓43%–64%	Arthralgias Headache Limb pain Fatigue	Absolute: • Prior sensitivity reaction to either medication
Cholesterol absorption inhibitors	Ezetimibe (Zetia) (10 mg Q day)	LDL-C ↓13%–20%	Diarrhea Back pain Abdominal pain	Absolute: • None Serious: • Cyclosporine

GI, gastrointestinal; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides.

FOLLOW-UP

A. Measure client's total cholesterol 4 weeks after initiation of diet and then at 3- to 4-month intervals.

B. If initiating medication therapy, obtain baseline blood work, including fasting lipid profile with LFTs and a CBC. Recheck tests 4 to 6 weeks after starting drug therapy. Then check at 6- to 12-month intervals or more frequently, if necessary.

CONSULTATION/REFERRAL

A. Refer to a dietitian for nutritional counseling for dietary modifications.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. The use of statin therapy for primary prevention of ASCVD in those over age 75 remains controversial. The use of the 10-year ASCVD risk calculator almost always exceeds the guideline-recommended treatment threshold in this population. However, there is little evidence that statins provide primary prevention in older adults.

2. The risk of side effects when using statins increases with age (myalgias, elevated liver enzymes, cognitive impairment).

3. The American Diabetes Association recommends statins as primary prevention in older adults with diabetes, even in those with no previous CVD who are over age 75.

4. There is compelling evidence supporting the use of statins for secondary prevention in individuals older than 75. Much the same as younger adults, adults over age 75 with clinical ASCVD have been shown to have a reduction in the risk for secondary events when a statin is used.

5. Because of a number of barriers, the rates of participation in physical activity are low among many older adults. Older adults who are isolated, frail, have mobility limitations or disabilities, and have fewer resources available may be particularly vulnerable to the effects of inactivity. Chronic pain and arthritis could represent additional barriers to physical activity among populations who are already at high risk of poor health outcomes associated with low levels of physical activity.

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ATRIAL FIBRILLATION

DEFINITION

A. Atrial fibrillation (AF) is the irregular and rapid heart rhythm caused by abnormal electrical impulses. These impulses make the heart's upper chambers (the atria) beat chaotically and out of sync with the heart's lower chambers (the ventricles),p resulting in poor circulation of blood throughout the body.

B. Classification of AF:

1. Paroxysmal AF:

a. Also called intermittent AF.

b. AF that is self-terminating usually within 48 hours or with intervention within 7 days of onset.

2. Persistent AF:

a. AF that is sustained greater than 7 days.

b. Usually requires pharmacologic and/or cardioversion to restore sinus rhythm.

3. Long-standing persistent AF:

a. Continuous AF for greater than 12 months.

4. Permanent AF:

a. Persistent AF in which rhythm control strategies are no longer effective (such strategies are not utilized to restore sinus rhythm).

5. Nonvalvular AF:

a. The term used to reference clients with paroxysmal, persistent, or permanent AF who do not have valvular heart disease (e.g., rheumatic mitral stenosis, a mechanical or prosthetic heart valve, or mitral valve prolapse [MVP]).

6. "Lone" AF:

a. The term used historically to refer to younger clients without clinical or echocardiographic evidence of cardiopulmonary disease, hypertension (HTN), or diabetes.

INCIDENCE

A. AF is the most common heart arrhythmia. The prevalence of AF in adults who are ≥65 years of age increased 5% per year from 1993 to 2007. More than 750,000 hospitalizations in the United States annually list AF as the primary admitting diagnosis. An estimated 150,000 deaths per year in the United States are attributed to AF. Clients with AF are hospitalized twice as often and are three times more likely to have multiple admissions. As of 2017, an estimated 2.7 million and 6.1 million American adults have AF. It is estimated that by 2030 more than 12 million Americans will have AF. AF is more common in males than in females and more common in Caucasians than other ethnicities.

PATHOGENESIS

A. Multiple impulses travel throughout the atria, yielding continuous electrical activity and an atrial rate in excess of 300 beats per minute (bpm). The impulses enter the atrioventricular node in a completely random manner. A small percentage of the impulses are conducted to the ventricle, which results in a lower ventricular rate, usually 100 to 180 bpm, and an irregularly irregular rhythm. This leads to ineffective atrial contractions, a decrease in cardiac output, and an increased risk of thrombus formation.

PREDISPOSING FACTORS

A. Increased age:

1. Approximately 1% of all AF clients are less than 60 years of age.

2. Approximately one-third of all AF clients are older than 80 years.

B. Ischemic heart disease (IHD).

C. HTN.

D. Rheumatic heart disease.

E. Valvular heart disease:

1. Mitral valve stenosis.

2. Mitral regurgitation.

3. Tricuspid regurgitation.

4. Left ventricular hypertrophy (LVH).

F. Heart failure (HF).

G. Myocardial infarction (MI).

H. Hyperthyroidism.

I. Obstructive sleep apnea (OSA).

J. Obesity.

K. Pericarditis.

L. Myocarditis.

M. Electrocution.

N. Pneumonia.

O. Pulmonary embolism (PE).

P. Cardiothoracic surgery.

Q. Diabetes mellitus.

R. Smoking.

S. Excessive alcohol use.

T. Recent cardiac surgery, including heart transplantation.

U. Pulmonary diseases (related to hypoxia):

1. Chronic obstructive pulmonary disease (COPD).

2. Bronchitis, acute or chronic.

3. Asthma.

4. Emphysema.

V. Electrolyte imbalances:

1. Hyperkalemia and hypokalemia.

2. Hypercalcemia and hypocalcemia.

3. Hypermagnesemia and hypomagnesemia.

W. Factors that can trigger episodes of AF:

1. Physical or emotional stress.

2. Nicotine.

3. Caffeine.

4. Alcohol.

5. Exercise.

COMMON COMPLAINTS

A. Palpitations.

B. Angina.

C. Fatigue.

D. Dyspnea at rest or on exertion.

E. Vertigo or dizziness.

F. Disorientation.

G. Confusion.

H. Syncope.

I. Headache.

J. Urinary frequency or urgency.

K. Anxiety.

L. Asymptomatic presentation, most often seen in the elderly and in clients with permanent AF.

POTENTIAL COMPLICATIONS

A. Stroke:

1. The risk of stroke increases four to five times during an episode of AF.

2. An AF-related stroke is more likely to be severe than a non-AF-related stroke.

B. HF:

▶

1. The risk of HF increases three times in the presence of AF.

C. Dementia:

1. The risk of dementia increases two times in the presence of AF.

D. Mortality:

1. Overall mortality increases two times in the presence of AF.

2. There is increased mortality in individuals who have other cardiovascular conditions or procedures, specifically HF, MI, coronary artery bypass surgery (CAB), stroke, and HTN.

E. PE.

F. Peripheral emboli:

1. These may present as an ischemic extremity or ischemic bowel.

SUBJECTIVE DATA

A. Ask the client what activity brought about or preceded the episode.

B. Have the client describe the duration of symptoms and what time of day the symptoms began.

C. Ask the client to describe their symptoms.

D. Ask the client whether any previous episodes have occurred.

E. Ask the client to list all medications, over-the-counter (OTC), and herbal products currently being taken or recently stopped:

1. Medications with links to AF:

a. Common complications with AF:

i. Theophylline (theophylline anhydrous; Theo-24, Elixophyllin).

ii. Digoxin (Lanoxin).

iii. Quinidine (quinidine gluconate; Nuedexta).

iv. Tricyclic antidepressants (TCAs).

b. Rare complications with AF:

i. Donepezil hydrochloride (Aricept).

c. Questionable complications with AF:

i. Bisphosphonates (alendronate, risedronate, etidronate).

F. Ask client to quantify their smoking history, alcohol history, and caffeine intake.

PHYSICAL EXAMINATION

A. Clients presenting with acute cardiovascular episode should be quickly assessed for the need to call emergency services/911 for immediate transport to the hospital.

B. Early screening is key. Current guidelines strongly suggest that all clients who present with breathlessness, palpitations, syncope, chest discomfort, and/or stroke symptoms have their pulse checked for irregularities and a 12-lead EKG.

C. Check vital signs: blood pressure (BP), pulse, and respirations. Count heart rate for 1 full minute.

1. Check orthostatic BP: sitting, standing, and lying down.

D. Inspect:

1. Inspect overall physical appearance, noting any distress.

2. Inspect the neck: Check jugular vein distension and pulsations:

a. **Provoking maneuvers (e.g., carotid massage) should only be performed by a cardiologist.**

3. Inspect extremities: Note edema, pallor, and cyanosis.

4. Perform a funduscopic exam: Note hemorrhage, exudates, and papilledema to determine the presence of a malignant HTN.

E. Palpate:

1. Palpate extremities for peripheral pulses in the arm and groin; determine rate and regularity.

2. Assess capillary refill.

3. Palpate carotid arteries for thrills and heaves.

F. Auscultate:

1. Auscultate heart: While client is sitting, standing, and in left lateral recumbent positions, note normal and extra heart sounds (S3 and S4).

a. S4 is not present during AF.

2. Auscultate neck for carotid bruits.

3. Auscultate lungs: Note the presence of wheezing and crackles.

G. Additional areas for physical examination:

1. Assess for focal neurologic deficits (orientation, unilateral weakness, dysarthria).

DIAGNOSTIC TESTS

A. Complete blood count (CBC), basic metabolic panel (BMP; including electrolytes, blood glucose, blood urea nitrogen [BUN], creatinine), magnesium, and liver function tests (LFTs).

B. Thyroid profile and lipid profile.

C. Brain natriuretic peptide (BNP) and N-terminal pro-brain natriuretic peptide (NT-proBNP).

D. Cardiac profile (including troponin, creatine phosphokinase [CPK] test, creatine kinase-muscle/brain [CK-MB]).

E. Serum drug levels, digoxin, amiodarone, and quinidine (if applicable).

F. International normalized ratio (INR), if applicable.

G. Creatinine clearance (CrCl).

H. 12-lead EKG, required to confirm the AF diagnosis.

I. Two-dimensional echocardiogram.

J. Transesophageal echocardiogram (TEE), most sensitive and specific test to detect left atrial (LA) thrombi and identify features associated with an increased risk of LA thrombus formation and subsequent systemic embolism.

K. Chest x-ray.

L. Exercise stress test or thallium stress test, if exercise-induced arrhythmia or coronary artery disease (CAD) is suspected.

M. Holter monitoring.

N. Evaluation of sleep apnea.

DIFFERENTIAL DIAGNOSES

A. AF.

B. MI.

C. IHD.

D. HF.

E. Mitral stenosis.

F. HTN.

G. Hyperthyroidism.

H. Digitalis intoxication.

I. Acute infections.

PLAN

A. General interventions:

1. The goal of therapy is to improve the client's quality of life by reducing morbidity and prolonging survival.

B. Client teaching: *See Client Teaching Guide for this chapter, "Atrial Fibrillation."*

1. Encourage weight loss, smoking cessation, and stress management. ▶

2. Educate clients about the adverse effects of their anticoagulant and antiarrhythmic medications.

3. Instruct clients taking anticoagulants, including Coumadin (warfarin), to take steps to lessen their risk of falls. Some examples include well-fitting footwear, removing rugs from the home, nightlights between the bed and bathroom, and grab bars in the bathroom.

4. Educate clients with implanted defibrillators and pacemakers about their susceptibility for external electrical fields and avoidance of exposure.

5. Prevention:

a. Control other chronic medical conditions, that is, HTN, diabetes, HF, pulmonary diseases, and hyperlipidemia.

C. Dietary management:

1. Counsel client on proper nutrition, specifically a low-fat, low-cholesterol, and low-sodium diet. Give diet handouts.

2. Educate clients taking Coumadin (warfarin) regarding dietary modifications to prevent variations in INR levels.

D. Pharmaceutical therapy:

1. Anticoagulant therapy:

a. Note: The CHA_2DS_2-VASc score evaluates the risk of ischemic stroke in clients with AF. To calculate a CHA_2DS_2-VASc score, go to clincalc.com/cardiology/stroke/chadsvasc.aspx. In general, the lower the score, the stronger the consideration for oral anticoagulation. The one exception to this is when the client has a score of 1 due to gender alone.

b. Goal of therapy: prevention of thromboembolism:

i. Warfarin sodium (Coumadin, Jantoven): Beers criteria caution: AVOID warfarin in older clients also taking amiodarone due to risk of elevated INRs and subsequent bleeds. AVOID warfarin in older adults who use nonsteroidal anti-inflammatory drugs (NSAIDs) due to risk of gastrointestinal (GI) bleed (refer to Appendix C, Tables C.3 and C.4):

1) Doses: 1, 2, 2.5, 3, 4, 5, 6, 7.5, and 10 mg tablets.

2) A warfarin dose calculator is located at www.globalrph.com/warfarin_calc.htm.

3) Review all medications that may affect the anticoagulant effects of warfarin. GlobalRPH is a resource with a list of medications that both increase and decrease the INR (www.globalrph.com/warfarin_drug_interactions.htm). Because antibiotics can have a significant effect on both decreasing and increasing the INR (especially Bactrim, Cipro, and erythromycin), INRs should be checked at least twice weekly through the duration of antibiotic therapy.

4) The incidence of anticoagulation with warfarin often declines as age advances due to the increased risk of falls, bleeding, and other adverse events.

ii. Dabigatran (Pradaxa): Beers criteria caution (age 65 and older): AVOID use in older adults with CrCl less than 30 due to increased risk of bleeds:

1) Pradaxa 75, 110, and 150 mg tablets:

a) Dosage indications based on CrCl:

i) CrCl greater than 30 mL/min: 150 mg, taken BID.

ii) CrCl of 15 to 30 mL/min: 75 mg, taken BID.

iii. Rivaroxaban (Xarelto): Beers criteria caution (age 65 and older): Reduce dose in older adults with CrCl 30 to 50 and AVOID in older adults with CrCl less than 30:

1) Xarelto 2.5, 10, 15, and 20 mg tablets:

a) Dosage indications based on CrCl:

i) CrCl greater than 50 mL/min: 20 mg with evening meal.

ii) CrCl of 15 to 50 mL/min: 15 mg with evening meal.

iv. Apixaban (Eliquis): Beers criteria caution (age 65 and older): AVOID in older adults with CrCl less than 25:

1) Eliquis 2.5 and 5 mg tablets:

a) Dosage indications: The recommended dose is 2.5 mg, taken BID in clients who fit any of the following criteria:

i) 80 years of age or older.

ii) Weight greater than or equal to 60 kg (132 lbs).

iii) Serum creatinine greater than or equal to 1.5 mg/dL.

2. Antiplatelet therapy:

a. Goal of therapy: prevention of thromboembolism; modest preventive effect:

i. Clopidogrel bisulfate (Plavix):

1) 75 mg (daily dose) and 300 mg (single loading dose).

ii. Aspirin:

1) 81 and 325 mg tablets:

2) Beers criteria caution:

a) Use aspirin with caution in older adults over the age of 80 with no history of cardiovascular disease (CVD): There is no clear evidence that aspirin provides primary prevention for cardiac events, but there is a risk for GI bleeds.

b) In adults 65 and older, avoid use of greater than 325 mg due to risk of GI bleed, unless other alternatives are not effective and the client can take a gastroprotective agent.

c) In adults older than 75 years, or adults older than 65 taking corticosteroids, anticoagulants, or antiplatelet agents, there is an increased risk of upper GI bleeds. The use of proton pump inhibitors (PPIs) reduces but does not eliminate risk. The risk of GI bleed increases with duration of use of NSAIDs, including high-dose aspirin (ASA). At 1 year, the risk is 2% to 4% and this increases with each subsequent year.

3. Combined anticoagulant and antiplatelet therapies:

a. Prescription of both antiplatelet medications (Plavix and aspirin) in addition to an anticoagulant is referred to as triple therapy. Triple therapy is commonly used to prevent complications when two or more of the following conditions are present:

i. AF.

ii. Mechanical valve prosthesis.

iii. Drug-eluting coronary stent.

b. Triple therapy is linked to an increase in bleeding complications ranging from mild to life-threatening.

4. Heart rate control therapy:
   a. Goal of therapy: varies based on client age but typically involves achieving a ventricular rate between 60 and 80 bpm at rest and 90 and 115 bpm during moderate exercise.
   b. Beta-blockers:
      i. Acebutolol (acebutolol hydrochloride; Sectral): 200 and 400 mg capsules.
      ii. Atenolol (Tenormin): 25, 50 (scored), and 100 mg tablets.
      iii. Betaxolol (betaxolol hydrochloride; Betoptic, Betoptic S): 10 and 20 mg tablets, all scored.
      iv. Bisoprolol (bisoprolol fumarate; Zebeta): 5 (scored) and 10 mg tablets.
      v. Metoprolol (metoprolol tartrate; Lopressor, Toprol XL): 50 and 100 mg tablets, all scored.
      vi. Nadolol (Coreg, Corgard): 20, 40, and 80 mg tablets, all scored.
      vii. Propranolol (propranolol hydrochloride; Hemangeol, Inderal LA, Inderal XL, InnoPran XL): 10, 20, 40, 60, and 80 mg tablets, all scored.
      viii. Sotalol (sotalol hydrochloride; Betapace, Sorine): 80, 120, and 160 mg tablets, all scored.
      ix. Timolol (timolol maleate; Blocadren): 5, 10 (scored), and 20 mg (scored) tablets.
   c. Calcium channel blockers (CCBs):
      i. Amlodipine (amlodipine besylate; Norvasc): 2.5, 5, and 10 mg tablets.
      ii. Diltiazem (diltiazem hydrochloride; Cardizem): 30, 60 (scored), 90 (scored), and 120 mg (scored) tablets.
      iii. Felodipine: 2.5, 5, and 10 mg tablets.
      iv. Isradipine: 2.5 and 5 mg tablets.
      v. Nicardipine (nicardipine hydrochloride):
         1) 20 and 30 mg capsules.
         2) Cardene SR: 30, 45, and 60 mg capsules.
      vi. Nifedipine (Procardia): 10 and 20 mg capsules.
      vii. Nimodipine: 30 mg capsules.
      viii. Nisoldipine (Sular): 8.5, 10, 17, 20, 25.5, 30, 34, and 40 mg tablets.
      ix. Verapamil (verapamil hydrochloride; Calan Tarka): 40, 80 (scored), and 120 mg (scored) tablets.
   d. Digoxin (Lanoxin):
      i. Beers criteria caution: Digoxin should be avoided as first-line therapy for AF and should not be prescribed in daily doses greater than 0.125 mg: 0.125 and 0.25 mg tablets, all scored.
5. Heart rhythm control therapy:
   a. Goal of therapy: maintenance of a normal rhythm and suppression of AF:
      i. Sodium channel blockers:
         1) Disopyramide (Norpace): 100 and 150 mg capsules.
         2) Quinidine (quinidine gluconate): 200 and 300 mg tablets, all scored.
      ii. Potassium channel blockers:
         1) Amiodarone (amiodarone hydrochloride; Cordarone, Pacerone, Nexterone).
            a) Beers criteria caution: Amiodarone should be avoided as first-line therapy for AF unless the client has HF or substantial LVH: 200 mg tablet, scored.
         2) Dofetilide (Tikosyn): 125, 250, and 500 mcg capsules.
         3) Dronedarone (Multaq).
            a) Beers criteria caution: Dronedarone should be avoided in clients with permanent AF or with severe of recently decompensated HF: 400 mg tablet.
         4) Sotalol (sotalol hydrochloride; Betapace): 80, 120, and 160 mg tablets, all scored.

E. Surgical therapies:
   1. Ablation therapy:
      a. Goal of therapy: prevention of recurrent AF.
      b. Preferred clinical characteristics include the following:
         i. Symptomatic paroxysmal AF.
         ii. Failure of one or more antiarrhythmic medications.
         iii. Normal to mildly dilated atria.
         iv. Normal to mildly reduced ventricular function.
         v. Absence of severe pulmonary disease.
      c. The long-term efficacy of ablation therapy requires further study, especially with regard to clients with HF and structural heart disease.

FOLLOW-UP

A. AF clients should be comanaged with a physician.
B. AF that is resistant to routine therapy should always be followed by a cardiologist.
C. Lab monitoring as indicated by the client's anticoagulant and antiarrhythmic medications:
   1. CrCl for clients taking Pradaxa and Xarelto.
   2. INR for clients taking Coumadin. Multiple medications affect the anticoagulant property of Coumadin.
D. Follow-up is determined by client's needs, frequency of AF reoccurrence, and the presence of other medical conditions.
E. After defibrillator or pacemaker placement, monitor clients utilizing regular follow-up appointments and EKGs to identify failure of implanted device, thromboembolism, lead dislodgment, infection, and complicating arrhythmias.

EMERGENT ISSUES/INSTRUCTIONS

A. Clients should go to the ED if they have any of the following symptoms:
   1. Palpitations or a fluttering in their chest.
   2. Chest pain.
   3. Weakness or extreme tiredness.
   4. Shortness of breath (SOB) at rest or with activity.
   5. Dizziness.
   6. Disorientation.
   7. Confusion.
   8. Passing out or losing consciousness.
   9. Severe headache.
   10. Frequent urination or a compelling urge to urinate.
   11. Anxiety or panic symptoms.
   12. Sudden weakness in an arm or leg.
   13. Sudden change in vision.
   14. Trouble speaking or understanding others.
   15. If taking an anticoagulant and have any of the following symptoms:
      a. Nosebleed that will not stop with pressure.
      b. Coughing up blood.
B. Clients should contact the office if they are taking an anticoagulant medication and have any of the following symptoms:
   1. Vomiting or other illness causes them to miss more than one dose of their medications.
   2. Vomit that is bright red or dark and that looks like coffee grounds or grape jelly. ▶

3. Blood in their bowel movements that looks black or tarry in color.
4. Heavy periods.

C. Clients should contact the office if they are experiencing any new symptom(s) not present at their last office visit.

CONSULTATION/REFERRAL

A. If you suspect an acute cardiovascular episode, refer client for immediate hospitalization to initiate thrombolytic therapy, cardioversion, hypertensive management, and additional diagnostic testing.

B. Refer to cardiology as indicated by client's clinical situation, specifically the frequency of AF reoccurrence and the presence of other complex medical conditions.

C. AF that is resistant to routine therapy should always be followed by a cardiologist.

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CAROTID ARTERY STENOSIS

DEFINITION

A. Carotid artery stenosis is the narrowing of one or both of the carotid arteries.

B. Classification of carotid artery stenosis:
1. Moderate:
   a. Less than 69% narrowing.
2. Severe:
   a. 70% to 99% narrowing.

INCIDENCE

A. Moderate carotid artery stenosis:
1. Prior to age 70: male, 4.8%; female, 2.2%.
2. After age 70: male, 12.5%; female, 6.9%.

B. Severe carotid artery stenosis:
1. All ages: both sexes: 1.7%.

C. Severe asymptomatic carotid artery stenosis:
1. After age 80: male, 3.1%; female, 0.9%.

PATHOGENESIS

A. As the human body ages, the artery walls become thicker and less flexible (atherosclerosis). The plaques, also known as atheromas, can develop on the interior artery wall. Over time, the buildup of plaque reduces blood flow to the brain. The arterial narrowing also increases the risk of thrombus formation and subsequent stroke risk.

PREDISPOSING FACTORS

A. Nonmodifiable factors:
1. Age:
   a. Male: increased risk prior to age 75.
   b. Female: increased risk after age 75.
2. Personal or family history of any of the following:
   a. Stroke.
   b. Angina.
   c. Myocardial infarction (MI).
   d. Peripheral arterial disease (PAD).

B. Modifiable factors:
1. Hypertension (HTN).
2. Hypercholesterolemia.
3. Diabetes mellitus.
4. Obesity.
5. Tobacco abuse.
6. Sedentary lifestyle.

COMMON COMPLAINTS

A. Asymptomatic carotid artery stenosis:
1. Often there are no symptoms.

B. Symptomatic carotid artery stenosis:
1. Focal neurologic symptoms, acute onset:
   a. Monoparesis or hemiparesis.
   b. Sensory alterations:
      i. Paresthesias.
   c. Speech disturbance:
      i. Expressive aphasia.
      ii. Receptive aphasia.
      iii. Changes in speech pattern or vocal quality (dysprosody).
   d. Visual changes:
      i. Blurred vision.
      ii. Blindness in one eye, partial or complete.
      iii. Visual field loss: specifically contralateral homonymous hemianopsia.
      iv. Dimming of vision.
   e. Constructional apraxia (i.e., inability to perform purposeful movements).
2. Vertigo and syncope are symptoms not generally caused by carotid artery stenosis.

POTENTIAL COMPLICATIONS

A. Cerebrovascular accident (CVA).

B. Transient ischemic attack (TIA).

C. Death.

D. Comorbidities that increase complication risk:
1. Age: older than 80 years.
2. New York Heart Association (NYHA) III or IV heart failure (HF).
3. Left ventricular ejection fraction (LVEF): less than 30%.
4. Class III or IV angina pectoris.
5. Coronary artery disease (CAD): left main coronary artery.
6. CAD: multivessel.
7. Planned cardiac surgery within 30 days.
8. History of MI within 4 weeks.
9. Severe chronic lung disease (e.g., chronic obstructive pulmonary disease [COPD], bronchitis, emphysema, asthma).

SUBJECTIVE DATA

A. Ask client to describe any new neurologic symptoms.
  1. Further determine the frequency, intensity, and duration of the symptoms.
B. Ask client to list all medications currently being taken, including over-the-counter (OTC) substances.

PHYSICAL EXAMINATION

A. Clients presenting with acute neurologic symptoms indicative of a stroke (e.g., paresis, speech or swallowing difficulty, visual field loss) should be quickly assessed for the need to call emergency services/911 for immediate transport to the hospital.
B. Check vital signs: blood pressure (BP), pulse, and respirations.
C. Inspect:
  1. Inspect overall physical appearance, noting any distress.
  2. Eyes: Assess papillary reflex bilaterally and perform a funduscopic exam, noting arterial occlusions or ischemic retinal damage.
D. Auscultate:
  1. Neck: carotid bruits, bilaterally:
    a. Studies have shown that carotid bruits have a poor predictive value in asymptomatic clients but are more indicative of CAD. However, the presence of a bruit should still be documented.

DIAGNOSTIC TESTS

A. Noninvasive testing:
  1. Carotid Doppler/duplex ultrasound.
  2. Carotid magnetic resonance angiogram (MRA).
  3. CT angiography (CTA).
B. Invasive testing:
  1. Cerebral angiography with contrast:
    a. **The gold standard for determination of carotid artery stenosis.**

DIFFERENTIAL DIAGNOSES

A. Monoparesis:
  1. TIA.
  2. CVA/stroke.
  3. Multiple sclerosis.
  4. Separated shoulder.
  5. Fractured limb.
B. Hemiparesis:
  1. TIA.
  2. CVA/stroke.
  3. Multiple sclerosis.
  4. Spinal tumor.
C. Sensory alterations (paresthesias):
  1. TIA.
  2. CVA/stroke.
  3. Peripheral neuropathy.
  4. Cervical spinal stenosis.
  5. Lead poisoning.
  6. Lyme disease.
  7. Wernicke syndrome.
  8. Multiple sclerosis.
D. Speech disturbance (aphasia):
  1. TIA.
  2. CVA/stroke.
  3. Myasthenia gravis.
  4. Brain tumor.
E. Speech disturbance (speech pattern or vocal quality; e.g., dysprosody):
  1. TIA.
  2. CVA/stroke.
  3. Brain tumor.
F. Visual changes:
  1. TIA.
  2. CVA/stroke.
  3. Retinal detachment.
  4. Ocular migraine.
  5. Cataracts.
  6. Giant cell arteritis.
  7. HTN.
  8. Macular degeneration.
  9. Sarcoidosis.
  10. Glaucoma.
  11. Brain tumor.
G. Constructional apraxia:
  1. TIA.
  2. CVA/stroke.
  3. Brain tumor.

PLAN

A. General interventions:
  1. The goal of therapy is to improve the client's quality of life by reducing morbidity and prolonging survival.
  2. The estimated risk of stroke within 5 years when utilizing medical management is 11.8% versus the risk of perioperative death, which is 6.4%.
B. Client teaching:
  1. Educate client about modifying controllable risk factors such as HTN, diabetes, hypercholesterolemia, obesity, tobacco abuse, and sedentary lifestyles.
  2. If known carotid artery stenosis is present:
    a. Make sure client is aware of signs and symptoms of TIA and stroke.
    b. Make sure client knows to seek medical attention or dial 911 if signs and symptoms occur.
C. Dietary management:
  1. Counsel client regarding appropriate change to their diet (e.g., low-fat, low-cholesterol, and low-sodium diet if applicable).
  2. Reinforce teaching with nutrition/diet handouts.
  3. Consider referral to a registered dietitian.
D. Pharmaceutical therapy:
  1. Antiplatelet therapy:
    a. Aspirin:
      i. Taken prior to carotid endarterectomy (CEA) surgery and for at least 3 months postoperatively.
      ii. Dosage: 81 to 325 mg/d.
      iii. Use caution when prescribing higher dose aspirin therapy in older adults. See geriatrics discussion under the "Individual Considerations" section.
  2. Additional pharmaceutical therapies:
    a. Hypertensive therapy:
      i. BP goal: less than 140/90.
    b. Hyperlipidemia therapy:
      i. Low-density lipoprotein cholesterol (LDL-C) goal (primary): less than 100 mg/dL.
      ii. LDL-C goal (secondary): less than 70 mg/dL.
    c. Tobacco cessation therapy.
E. Surgical therapies:
  1. CEA:
    a. Goal of therapy: stroke risk reduction.

b. Preferred clinical characteristics:
i. Recent TIA or CVA within 6 months and ipsilateral stenosis between 70% and 99% (severe):
1) CEA is recommended.
2) Morbidity/mortality: less than 6%.
ii. Recent TIA or CVA within 6 months and ipsilateral stenosis between 50% and 69% (moderate):
1) CEA is recommended after analysis of other factors that include age, gender, and comorbidities.
2) Morbidity/mortality: less than 6%.
iii. Ipsilateral stenosis less than 50%:
1) No indication for CEA surgery.
c. Pharmaceutical therapy and CEA:
i. Aspirin:
1) Recommendations: Take prior to CEA surgery and for at least 3 months postoperatively unless contraindicated.
2) Dosage: 81 to 325 mg/d.
ii. Other available antiplatelet agents do not have standardized dosage or frequency regimens established for postoperative CEA clients.

2. Carotid angioplasty/stenting (CAS):
a. Goal of therapy: stroke risk reduction.
b. Preferred clinical characteristics:
i. Symptomatic clients with stenosis less than 70% as determined by noninvasive imaging or less than 50% as determined by conventional contrast angiography.
ii. Symptomatic clients with stenosis greater than 70% in which surgical access via CEA would be difficult or history indicates an increased complication risk (e.g., radiation-induced stenosis and restenosis of a prior CEA).
iii. Ipsilateral stenosis less than 50%:
1) No indication for CAS surgery.
c. Pharmaceutical therapy and CAS:
i. Acetaminophen (aspirin):
1) Recommendations: Take prior to CEA surgery and for at least 3 months postoperatively unless contraindicated.
2) Dosage: 81 to 325 mg/d.
ii. Clopidogrel (Plavix):
1) Recommendations: 300 mg loading dose on operative day and 75 mg/d after stenting.
iii. Other available antiplatelet agents do not have standardized dosage or frequency regimens established for postoperative CAS clients.

3. Contraindications to carotid surgery:
a. Chronic total occlusion of the stenosed artery.
b. Clients with severe disability related to prior CVA.

FOLLOW-UP

A. Follow-up with cardiology and vascular surgery is determined by client's needs and the presence of other medical conditions.

EMERGENT ISSUES/INSTRUCTIONS

A. Clients presenting with acute neurologic symptoms indicative of a stroke (e.g., paresis, speech or swallowing difficulty, visual field loss) should be quickly assessed for the need to call emergency services/911 for immediate transport to the hospital.

CONSULTATION/REFERRAL

A. Referral to a registered dietitian is indicated for additional teaching regarding dietary modifications.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. Beers criteria: aspirin cautions:
a. Use aspirin with caution in older adults over age 80 with no history of cardiovascular disease (CVD): There is no clear evidence that aspirin provides primary prevention for cardiac events, but there is a risk for gastrointestinal (GI) bleeds. The routine use of aspirin as primary prevention is not recommended.
b. In adults 65 and older, avoid use of greater than 325 mg of aspirin due to risk of GI bleed, unless other alternatives are not effective and the client can take a gastroprotective agent.
c. In older adults more than 75 years old, or adults older than 65 taking corticosteroids, anticoagulants, or antiplatelet agents, there is an increased risk of upper GI bleeds. The use of proton pump inhibitors (PPIs) reduces but does not eliminate risk.
d. The risk of GI bleed increases with duration of use of nonsteroidal anti-inflammatory drugs (NSAIDs), including high-dose aspirin (ASA). At 1 year, the risk is 2% to 4%, and this increases with each subsequent year.

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CHEST PAIN

DEFINITION

A. Chest pain is a localized sensation of distress or discomfort that may or may not be associated with actual tissue damage.

INCIDENCE

A. Chest pain is one of the most common complaints of adult clients. Causes can range from minor disorders to life-threatening diseases, so every client must be assessed carefully.

PATHOGENESIS

A. Cardiac etiology: ischemia, atherosclerosis, inflammation, or valvular problems due to angina, myocardial infarction (MI), pericarditis, endocarditis, dissecting aortic aneurysm, or mitral valve prolapse (MVP; see Table 13.3).
B. Musculoskeletal etiology: muscle strain and inflammation due to costochondritis, chest wall syndrome, cervicodorsal arthritis, or intercostal myositis.

TABLE 13.3 COMPARISON OF COMMON CHEST PAIN ETIOLOGIES

Condition	Pain Findings	Associated Symptoms	Precipitating Factors	Relieving Factors	Physical Findings	Diagnostic Tests	Treatment Modalities
Cardiac stable angina	Substernal, tight, and dull pressure, usually lasts longer than 15 minutes		Exertion, cold, emotional stress	Rest, nitroglycerin, Valsalva maneuver	Sinus tachycardia, bradycardia, xanthomas, signs of HF	Resting EKG, stress EKG, cardiac enzymes, echo, angiogram	ASA, BB, nitrates, CCB
Prinzmetal angina (variant)	Substernal, achy, tight, and dull pressure		Often occurs at rest; may awaken from sleep	Nitroglycerin	Same as stable angina	Resting EKG, angiogram	ASA, nitrates, CCB
MI	Precordial, substernal, severe, crushing, squeezing, lasts >15 minutes	Dyspnea, sweating, dizziness, radiation to neck/arm/jaw, N&V, cough, fever, unstable V/S	Oxygen	Not relieved by nitroglycerin	S3 or S4 murmur, tachycardia, bradycardia pericardial friction rub, hyper/hypotension	EKG, serial CK enzymes, echo, radionuclide studies	Analgesia, reperfusion, prevention, and treatment complications limit infarcts Thrombolytic therapy for acute MI
MVP	Usually not substernal, often knifelike, may last 1–3 hours	Palpitations, fatigue, light-headed, arrhythmia, syncope		Recumbent position, BB, nitroglycerine	Midsystolic click and/or murmur, thin body status, SOB	Echo, EKG	Usually none, BB if palpitations or ventricular ectopy becomes disabling
Hypertrophic cardiomegaly	Similar to angina	Dyspnea with exertion, arrhythmia, light-headed, syncope	May be increased by nitroglycerin, exertion	BB, squatting	Systolic murmur, increased upright position, Valsalva maneuver, more forceful PMI	EKG, CXR, echo, Doppler, cardiac catheterization	BB, CCB, possible pacing and myomectomy, exercise restriction
Pericarditis	Retrosternal, sharp or dull, sudden onset, long duration, radiates to one side of the trapezius	Fever, myalgia, anorexia, anxiety, recent viral infection		Sitting up, leaning forward	Friction rub, SVT1, tachypnea, crackles, signs of cardiac tamponade	EKG, echo, CBC, ESR	Hospitalize, rule out purulent process, analgesics

(*continued*)

TABLE 13.3 COMPARISON OF COMMON CHEST PAIN ETIOLOGIES (*CONTINUED*)

Condition	Pain Findings	Associated Symptoms	Precipitating Factors	Relieving Factors	Physical Findings	Diagnostic Tests	Treatment Modalities
Endocarditis	Usually dull, retrosternal, may radiate to back	Fever, night sweats, joint pain, back pain, weight loss, headache, murmur			Systolic–diastolic murmur, petechiae, Osler modes, Roth spots, neck vein distension, pleural or pericardial rub, pain in extremities, splenomegaly, hematuria	CBC, ESR, blood cultures, echo	Hospitalization, for antibiotic therapy
GI esophageal spasm	May be identical to angina		Alcohol or cold liquids	May be relieved by nitroglycerin		Esophageal manometry	Nitroglycerin, anticholinergics, esophageal dilation
Esophagitis	Burning, tightness	Heartburn, water brash	Overeating, alcohol, recumbent position	Antacids	May have slight to moderate epigastric tenderness	Esophagoscopy	Lifestyle modification, antacid, H2 blocker, PPI, promotility agent
Musculoskeletal costochondritis	Sharp, sometimes pleuritic, parasternal costochondral pain		Sneezing, cough on deep inspiration, twisting motions, reaching overhead		Erythema at sites of tenderness, positive pinpoint tenderness at costochondral junctions	CXR to rule out other causes	NSAIDs, ASA, ibuprofen, naproxen, heat application

ASA, aspirin; BB, beta-blocker; CBC, complete blood count; CCB, calcium channel blocker; CK, creatine kinase; CXR, chest x-ray; ESR, eosinophilic sedimentation rate; GI, gastrointestinal; HF, heart failure; MI, myocardial infarction; MVP, mitral valve prolapse; NSAIDs, nonsteroidal anti-inflammatory drugs; N&V, nausea and vomiting; PMI, point of maximal impulse; PPI, proton pump inhibitor; SOB, shortness of breath; SVT, supraventricular tachycardia; V/S, vital signs

C. Neurologic etiology: nerve inflammation and/or compression due to herpes zoster and nerve root compression.
D. Gastrointestinal (GI) etiology: structural defects, inflammation, or infection due to gastroesophageal reflux disease (GERD), hiatal hernia, esophageal spasm, pancreatitis, cholecystitis, or peptic ulcer disease (PUD).
E. Pleural etiology: inflammation, distension, or compression of pleural membranes due to pneumonia, pulmonary embolus, pulmonary hypertension (HTN), spontaneous pneumothorax, and lung and mediastinal tumors.
F. Psychogenic etiology: stress due to anxiety, depression, or panic disorders.

PREDISPOSING FACTORS

A. These vary depending on the etiology of pain.

COMMON COMPLAINTS

A. Primary complaint: pain somewhere in the chest.
B. "Levine sign": placing the fist to the center of the chest to demonstrate pain.
C. Fatigue.
D. Cough.
E. Indigestion.
F. Dyspnea.
G. Syncope.
H. Palpitations.
I. Profound fatigue.

OTHER SIGNS AND SYMPTOMS

A. Pain may be typical of angina and MI.
B. Musculoskeletal pain may be relieved by position change, aggravated by body movement, reproducible, or caused by injury or trauma.
C. Neurologic pain is associated with skin lesion if herpes zoster is the causative agent.
D. GI pain may be associated with meals, certain positions, belching, or an acid "brash" taste in mouth, or it may be referred to other sites.
E. Pleural pain is usually accompanied by cough, upper respiratory infection (URI) symptoms, or shortness of breath (SOB).
F. Psychogenic pain or pressure along with SOB and dizziness may be associated with a specific event or time.

SUBJECTIVE DATA

A. How long has the client had chest pain?
B. Has the client ever been treated for chest pain? What treatments, tests, and medications (such as nitroglycerin) were used?
C. What precipitates and relieves the client's chest pain?
  1. Precipitating factors: exertion, taking a deep breath, eating, cold, stress, and sexual intercourse.
  2. Alleviating factors: resting, eating, taking an antacid, taking nitroglycerin, and positional change.
D. Inquire about the characteristics of pain.
  1. Location: neck, throat, chest, epigastric area, and shoulder.
  2. Radiation: neck, throat, shoulder, lower jaw, and upper extremity.
    a. **Radiation to one or both arms is a predictor of acute MI.**
    b. **Chest pain that radiates between the scapulae may be due to aortic dissection.**
  3. Quality: squeezing, pressure, strangling, fullness, heavy weight, tightening, constriction, and ripping/tearing (acute aortic dissection).
  4. Intensity: abrupt onset, gradually getting worse, dull, or insidious.
  5. Duration: seconds, minutes, hours, or years.
  6. Frequency: intermittent, occurs every morning/evening.
E. Are other associated symptoms present?
F. Discuss any risk factors the client may have for cardiac disease: smoking, hyperlipidemia, HTN, sedentary lifestyle, diabetes, and family history.
G. Review medical history as noted earlier.
H. Review all medications, including prescription (such as sildenafil), over-the-counter (OTC), and herbal products.
I. Review recreational/illicit drug use.
J. Inquire about any new physical labor if musculoskeletal etiology is suspected.
K. Has the client had any trauma (including domestic violence)?
L. Has the client had a recent infection?

PHYSICAL EXAMINATION

A. Check temperature (if infection is suspected), pulse, respirations, blood pressure (BP), and pulse oximetry.
B. Inspect:
  1. Inspect general appearance:
    a. Appearance of discomfort/distress.
    b. Any appearance of respiratory distress.
    c. Evaluate jugular venous distention (JVD).
    d. Note client position: sitting, lying, squatting. Relief of chest pain with recumbency suggests MVP; relief with squatting suggests hypertrophic cardiomyopathy. Noncardiac chest pain may be present along with cardiac chest pain.
  2. Inspect skin for diaphoresis, jaundice, pallor, herpes zoster lesions, rash, or cyanosis.
  3. Inspect the chest wall for herpes zoster lesions or signs of trauma.
  4. Inspect eyes by performing funduscopic exam.
  5. Inspect legs for signs of phlebitis: unilateral swelling, cyanosis, venous stasis, and diminished pulses.
  6. Inspect neck for enlarged thyroid and lymph nodes, midline trachea, and JVD.
C. Palpate:
  1. Palpate chest wall for tenderness and swelling. Chest pain present in only one body position is usually not cardiac in origin.
  2. Palpate abdomen for masses, tenderness, bounding pulses, organomegaly, and ascites.
  3. Palpate femoral and distal pulses.
D. Auscultate:
  1. Auscultate carotid arteries for bruits.
  2. Auscultate lungs for crackles, wheezes, equal breath sounds, and pleural rub.
  3. Auscultate abdomen for bruits and bowel sounds.
  4. Auscultate heart for murmurs, rubs, clicks, irregularities, or extra sounds.
E. Perform neurologic exam:
  1. Perform this exam if neurologic etiology is suspected.

DIAGNOSTIC TESTS

A. Testing depends on information collected in the exam. A normal physical exam, EKG, and/or lab test results in a client with chest pain does not rule out coronary heart disease (CHD). Typical tests include the following:
  1. EKG.
  2. Chest radiography, whenever diagnosis of chest pain is not clear.
  3. Echocardiogram.

▶

4. Stress test.
5. Cardiac catheterization.
6. Barium tests.
7. Endoscopy to rule out GI etiology.
8. Esophageal pH, low.
9. Lab tests:
   a. Troponin I or T.
   b. Myoglobin.
   c. Creatine kinase (CK).
   d. Creatine kinase-muscle/brain (CK-MB) isoenzyme: may be useful if the initial troponin determination is abnormal for suspected reinfarction.
   e. C-reactive protein (CRP).
   f. B-type natriuretic peptide (BNP) for clinical findings/risk of heart failure.
   g. D-dimer for suspected venous thrombotic event (deep vein thrombosis [DVT] or pulmonary embolism [PE]).

DIFFERENTIAL DIAGNOSES

A. Cardiac causes:
1. CHD:
   a. Acute MI: chest pain lasting more than 15 minutes.
   b. Unstable angina pectoris.
   c. Stable angina pectoris.
   d. Prinzmetal, or variant, angina.
2. Valvular heart disease:
   a. MVP.
   b. Aortic stenosis.
3. Cardiomyopathies:
   a. Hypertrophic.
   b. Dilated.
   c. Restrictive.
4. Pericarditis.
5. Endocarditis.
6. Aortic dissection.

B. Noncardiac causes:
1. Pulmonary causes:
   a. Pneumonia.
   b. Pleurisy.
   c. PE.
   d. Pulmonary HTN.
   e. Pneumothorax.
   f. Tracheobronchitis.
   g. Lung cancer.
2. GI causes:
   a. GERD.
   b. Esophageal spasm.
   c. PUD.
   d. Pancreatitis.
   e. Cholecystitis.
   f. Flatulence.
3. Rheumatology causes:
   a. Fibromyalgia.
   b. Costochondritis.
   c. Arthritis.
4. Chest wall causes:
   a. Rib fracture.
   b. Muscle strain.
   c. Cervical or thoracic spine disease.
   d. Metastatic bone disease.
   e. Breast conditions.
5. Neurologic causes:
   a. Herpes zoster.
   b. Postherpetic pain syndrome.
   c. Nerve root compression.
6. Psychogenic causes:
   a. Panic disorder.
   b. Generalized anxiety.
   c. Depression.
   d. Somatoform disorders.

PLAN

A. General interventions:
1. Direct management toward primary disorder causing the symptom.

C. Client teaching:
1. Teach the client about medications.
2. Encourage CPR training for the client's family and/or close friends for clients with coronary artery disease (CAD).
3. Explain to the client and their family when and how to call 911 and the importance of going to the ED immediately so that thrombolytic therapy can be considered. A positive response to nitroglycerin does not confirm the presence of CAD.

D. Pharmaceutical therapy:
1. Cardiac pain: nitroglycerin.
2. GI pain: H2 blocker and proton pump inhibitors (PPIs).
3. Musculoskeletal pain: nonsteroidal anti-inflammatory drugs (NSAIDs).
4. Psychogenic pain:
   a. Selective serotonin reuptake inhibitors (SSRIs).
   b. Tricyclic antidepressants (TCAs).
   c. Benzodiazepines.

FOLLOW-UP

A. Follow clients with CAD for an indefinite period of time to detect the recurrence or progression of disease.
B. Other follow-up depends on the etiology of chest pain.

EMERGENT ISSUES/INSTRUCTIONS

A. Refer clients exhibiting signs or symptoms of acute MI or aortic dissection to the closest ED and/or call 911.
B. Refer clients with hemodynamic instability to the closest ED and/or call 911.

CONSULTATION/REFERRAL

A. Consult a physician when chest pain is cardiac in origin. If a cardiac origin is found in a pregnant client, schedule a cardiology consultation as soon as possible for comanagement.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Evaluate chest pain in the same manner as in nonpregnant clients.
2. Rule out pregnancy-induced hypertension (PIH) and HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome when a third-trimester client presents with upper epigastric/chest pain.

B. Geriatrics:
1. Frail elderly clients often do not present with the "typical" symptom complex of chest pain. Frequently, the only symptoms of acute MI are lethargy, decreased level of consciousness (LOC), crackles, congestive heart failure (CHF), persistent cough, or hypotension.
2. The following signs and symptoms may be the primary presentation in the frail older person for many acute illness states:
   a. Confusion.
   b. Self-neglect.

▶

c. Falling.
d. Incontinence.
e. Apathy.
f. Anorexia.
g. Dyspnea.
h. Fatigue.

3. Prescribe medications for frail elderly clients based on their creatinine clearance and then slowly taper upward to the desired effect.

4. Assess for polypharmacy (considered to be greater than five to nine medications consumed on a daily basis) and medication interactions; side effects may account for or worsen many of the symptoms and geriatric syndromes described previously (refer to the "Polypharmacy" and "Deprescribing" sections of Chapter 3).

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CHRONIC VENOUS INSUFFICIENCY AND VARICOSE VEINS

DEFINITION

A. Peripheral vascular disease (PVD) is a general term that encompasses all occlusive or inflammatory diseases that occur within the peripheral arteries, veins, and lymphatics. These conditions include peripheral arterial disease (PAD), deep vein thrombosis (DVT), superficial thrombophlebitis, lymphedema, and chronic venous diseases. Chronic venous diseases include chronic venous insufficiency (CVI) and varicose veins.

INCIDENCE

A. CVI:

1. It is estimated that six to seven million adults in the United States have CVI. CVI is twice as common in females as in males.

2. Peak incidence is seen in females over 50 years of age.

3. CVI is the most common cause of the 2.5 million leg ulcerations in the United States.

B. Varicose veins:

1. More than 24 million Americans have varicose veins. They are usually thought to be more common in females; however, there is a higher incidence in males, especially African American males.

PATHOGENESIS

A. CVI: Venous insufficiency is caused by dysfunctional valves that allow valvular reflux and subsequently venous hypertension (HTN) and impaired circulation. In CVI, persistent ambulatory venous HTN leads to obstruction of venous flow, which produces local tissue anoxia, inflammation, and at times even tissue necrosis. This process eventually causes subcutaneous fibrosing panniculitis and additional venous and lymphatic outlet obstruction.

B. Varicose veins: Varicose veins are a form of CVI. The same dysfunctional valves that cause valvular reflux and subsequently venous HTN in CVI also cause varicose veins. This influx of volume and pressure causes the vessel walls to weaken, causing the veins to dilate, elongate, twist, and bulge.

PREDISPOSING FACTORS

A. CVI:

1. Age >30 years.
2. Sex, being female.
3. Prolonged standing or sitting.
4. History of DVT.
5. Stature, more common in tall persons.
6. Obesity.
7. Sedentary lifestyle.
8. Family history.
9. History of multiple pregnancies.
10. HTN.

B. Varicose veins:

1. Genetics:
a. Risk increases to 90% if both parents have varicose veins.
b. If one parent is affected, the risk increases by 25% for males and 62% for females.
2. Age.
3. Pregnancy.
4. Prolonged standing.
5. Restrictive clothing.
6. Obesity.
7. Ligamentous laxity:
a. History of hernia(s).
b. Flat feet.
8. Smoking.
9. History of superficial or deep vein clots.

COMMON COMPLAINTS

A. CVI:

1. Extremity edema.
2. Pain worse when standing, usually dull, aching, or cramping.
3. Pain improved with elevation.
4. Itching sensation.
5. Feeling of heaviness in extremity.
6. Hyperpigmentation.
7. Thickening and hardening of the skin.
8. Ulcerations.

B. Varicose veins:

1. Pain, usually burning, aching, or itching.
2. Blue veins that protrude above the surface of the skin.
3. Leg fatigue.
4. Edema.
5. Symptoms worsening toward the end of the day.
6. Leg heaviness.

POTENTIAL COMPLICATIONS

A. CVI:

1. Cellulitis.
2. Peripheral neuropathy.
3. Varicose veins.
4. Abscess.
5. Ulceration.
6. Stasis dermatitis.
7. DVT.

▶

B. Varicose veins:
- **1.** Stasis dermatitis.
- **2.** Stasis ulceration.
- **3.** Petechial hemorrhage.
- **4.** Chronic edema.
- **5.** Superficial thrombophlebitis.
- **6.** Hyperpigmentation.
- **7.** Eczema.
- **8.** DVT.

SUBJECTIVE DATA

A. Ask client when the symptom(s) were first noticed.
B. Have client describe duration of symptoms.
C. Ask client to describe pain, for example, crushing, stabbing, or burning.
D. Ask the client what makes the symptoms better and what makes them worse.
E. Have client rate pain on a scale of 0 to 10, with 0 being no pain.
F. Ask client to list all medications currently being taken, particularly substances not prescribed and illicit drugs such as cocaine.
G. Review recent history of invasive procedures or surgery.
H. Review medical history for heart disease, diabetes, HTN, and DVT.
I. Ask client if they have an increase in the size of lower extremities after being on their feet. Discern if one leg is worse than the other.

PHYSICAL EXAMINATION

A. CVI:
- **1.** Check vital signs.
  - a. Check blood pressure (BP) and document resting heart rate, respirations, temperature (if indicated), height, and weight.
- **2.** Inspect:
  - a. Inspect extremity for edema, hyperpigmentation, erythema, difference in temperature.
  - b. Inspect and document any varicosities.
- **3.** Palpate:
  - a. Palpate distended veins, noting tenderness.
  - b. Perform the cough impulse test to determine turbulent retrograde flow.
  - c. Perform the tap test to determine if the great saphenous vein is distended with blood.
- **4.** Auscultate:
  - a. Auscultate the heart: rate, rhythm, heart sounds, murmur, and gallops.
  - b. Auscultate lungs: lung sounds in all fields.
- **5.** Clinical, Etiology, Anatomy, and Pathophysiology (CEAP) clinical classification of CVI made based on the following:
  - a. Class C0: no visible signs of venous disease.
  - b. Class C1: telangiectasis or reticular veins (e.g., small visible blood vessels with no protrusion).
  - c. Class C2: visible varicose veins.
  - d. Class C3: edema without skin changes noted.
  - e. Class C4: skin discoloration/pigmentation.
  - f. Class C5: healed venous ulceration.
  - g. Class C6: active venous ulceration.

B. Varicose veins:
- **1. Clients presenting with any of the following should be quickly assessed for the need to call emergency services/911 for immediate transport to the hospital:**
  - a. **A bleeding varicosity with eroded surrounding skin.**
  - b. **A varicosity that has bled and is at risk for bleeding again.**
  - c. **An ulceration that is worsening and/or painful despite treatment.**
- **2.** Check vital signs.
  - a. Check BP and document resting heart rate, respirations, temperature (if indicated), height, and weight.
- **3.** Inspect:
  - a. Inspect skin for superficial veins that are raised above the skin's surface; client should be standing.
  - b. Inspect extremity for edema, hyperpigmentation, and eczema.
- **4.** Palpate:
  - a. Palpate distended veins, noting tenderness.
- **5.** Auscultate:
  - a. Auscultate heart: rate, rhythm, heart sounds, murmur, and gallops.
  - b. Auscultate lungs: lung sounds.
- **6.** CEAP clinical classification of varicose veins made based on the following:
  - a. Class C0: no visible signs of venous disease.
  - b. Class C1: telangiectasis or reticular veins (e.g., small visible blood vessels with no protrusion).
  - c. Class C2: visible varicose veins.
  - d. Class C3: edema without skin changes noted.
  - e. Class C4: changes in skin and subcutaneous tissue.
    - i. Class C4A: pigmentation or eczema.
    - ii. Class C4B: lipodermatosclerosis (panniculitis) or atrophie blanche.
  - f. Class C5: healed venous ulceration.
  - g. Class C6: active venous ulceration.

DIAGNOSTIC TESTS

A. CVI:
- **1.** Trendelenburg test.
- **2.** Perthes test.
- **3.** Doppler ankle–brachial index (ABI).
- **4.** Duplex venous ultrasound.

B. Varicose veins:
- **1.** Trendelenburg test.
- **2.** Perthes test.
- **3.** Duplex venous ultrasound.

DIFFERENTIAL DIAGNOSES

A. CVI:
- **1.** DVT.
- **2.** Ulceration.
- **3.** Infection.
- **4.** Peripheral artery disease (PAD).
- **5.** Varicose veins with risk of hemorrhage.

B. Varicose veins:
- **1.** Arthritis.
- **2.** Peripheral neuritis.
- **3.** Nerve root compression.
- **4.** Telangiectasia.
- **5.** DVT.
- **6.** Inflammatory liposclerosis.

PLAN

A. General prevention of CVI and varicose veins:
- **1.** Avoid prolonged standing or sitting. ▶

2. Exercise on a regular basis.
3. Encourage smoking cessation, weight loss, and exercise, if applicable.
4. Encourage strategies to better manage other chronic medical conditions that directly affect the progression of PAD, that is, diabetes, dyslipidemia, obesity, and HTN.

B. CVI:
1. Nonpharmaceutical therapy:
   a. Extremity elevation.
   b. Compression stockings.
   c. Exercise.
   d. Venous ulcerations treated with wound care and compression therapy.
      i. Compression therapy is the primary mode of management and has demonstrated venous ulcer healing rates as high as 97%.
2. Pharmaceutical therapy:
   a. Diuretics: management of edema, short-term administration:
      i. Hydrochlorothiazide.
   b. Antiplatelet: may increase the speed of healing to ulcerations:
      i. Aspirin: 325-mg tablet.
         1) Beers criteria caution:
            a) Use aspirin with caution in adults older than age 80 with no history of cardiovascular disease (CVD): There is no clear evidence that aspirin provides primary prevention for cardiac events, but there is a risk for gastrointestinal (GI) bleeds.
            b) In adults 65 and older, avoid use of greater than 325 mg due to risk of GI bleed, unless other alternatives are not effective and the client can take a gastroprotective agent.
            c) In adults older than 75 years, or adults older than 65 taking corticosteroids, anticoagulants, or antiplatelet agents, there is an increased risk of upper GI bleeds. The use of proton pump inhibitors (PPIs) reduces but does not eliminate risk. The risk of GI bleed increases with duration of use of nonsteroidal anti-inflammatory drugs (NSAIDs), including high-dose aspirin (ASA). At 1 year, the risk is 2% to 4% and this increases with each subsequent year.
   c. Systemic antibiotics: management of infection in persons demonstrating an increase in pain, erythema, or increase in size of ulceration.
3. Surgery:
   a. Venous ablation, for clients who continue to be symptomatic after 6 months of nonpharmacologic therapies. The types of ablation are chemical, thermal, and mechanical.

C. Varicose veins:
1. Client teaching: *See Client Teaching Guides for this chapter, "Chronic Venous Insufficiency" and "Varicose Veins."*
   a. If prolonged standing is required, shift weight from one leg to the other.
   b. Do not sit with legs dependent.
2. Nonpharmaceutical therapy:
   a. Extremity elevation.
   b. Compression stockings.
   c. Exercise.
3. Surgery:
   a. Radiofrequency ablation.
   b. Endovenous laser therapy.
   c. Phlebectomy.
   d. Sclerotherapy and foam sclerotherapy.
   e. Vein ligation and vein stripping.
   f. Valvuloplasty, valvuloconstruction, valve transplantation, and valve transposition.
   g. Subfascial endoscopic perforator surgery, Linton operation, and percutaneous ablation of perforators.

FOLLOW-UP

A. Follow-up is determined by client's needs, frequency and intensity of symptoms, and the presence of other medical conditions.
B. PVD manifesting persistent symptoms should always be followed by a cardiologist.

EMERGENT ISSUES/INSTRUCTIONS

A. Clients should go to the ED if they have any of the following symptoms:
1. **A bleeding varicosity with eroded surrounding skin.**
2. **A varicosity that has bled and is at risk for bleeding again.**
3. **An ulceration that is worsening and/or painful despite treatment.**
4. **A change in color of a foot or hand.**

B. Clients should contact the office if they are experiencing any of the following:
1. **Fever over 101°F.**
2. **Increased redness, pain, tenderness to touch, swelling, and/or warmth.**
3. **New wound or sore on the affected arm or leg.**
4. **If taking an antiplatelet therapy and have any of the following symptoms:**
   a. **Vomiting or other illness causes them to miss more than one dose of their medications.**
   b. **Vomit that is bright red or dark and looks like coffee grounds or grape jelly.**
   c. **Blood in their bowel movements that looks black or tarry in color.**

C. Clients should contact the office if they are experiencing any new symptom(s) not present at their last office visit.

CONSULTATION/REFERRAL

A. If you suspect acute limb ischemia (ALI), refer client for immediate hospitalization to obtain diagnostic testing to determine the presence of a thrombus and restore circulation to the affected extremity.
B. If chronic limb ischemia (CLI) has led to ulceration and/or superimposed infection, hospitalization is indicated to initiate wound care consultation and diagnostic testing to determine the degree of arterial occlusion.
C. Referral to a cardiologist is indicated in the presence of persistent PVD symptoms.
D. Referral to a podiatrist becomes necessary to trim toenails and assess client for properly fitting shoes.
E. Referral to pain management is indicated if pain is resistant to treatment.

INDIVIDUAL CONSIDERATIONS

A. Nonambulatory clients:
1. Using rocking chairs is a possible substitute for persons unable to participate in a walking program.

B. Geriatrics:

1. Compression stockings and elevating the legs are first-line treatments for lower extremity venous insufficiency in older adults.

2. Use of diuretics solely to reduce ankle edema requires a careful consideration of risks versus benefits. If the edema is not linked with serious harmful symptoms like rapid weight gain indicating decompensated congestive heart failure (CHF), respiratory compromise, or lower extremity infection such as cellulitis, the treatment itself may contribute to more harmful effects such as hypotension, incontinence, or dehydration.

3. Use caution when prescribing aspirin therapy in older adults. See the "Atrial Fibrillation" section of this chapter, under "Pharmaceutical Therapy"; see also Appendix C, Table C.1.

4. Be alert to signs and symptoms of depression related to immobility and pain.

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DEEP VEIN THROMBOSIS

DEFINITION

A. Peripheral vascular disease (PVD) is a general term that encompasses all occlusive or inflammatory diseases that occur within the peripheral arteries, veins, and lymphatics. PVD includes deep vein thrombosis (DVT). DVT is a condition in which a thrombus forms in one or more veins. DVT greatly increases the risk of pulmonary embolism (PE).

INCIDENCE

A. The Centers for Disease Control and Prevention (CDC) estimates that as many as 900,000 Americans are diagnosed with a first-time DVT or PE annually and between 60,000 and 180,000 deaths annually are directly or indirectly attributed to DVT or PE.

B. The Vascular Disease Foundation states that approximately 5% of the world's population will have a DVT during their lifetime.

PATHOGENESIS

A. Changes within the venous system precipitate the formation of a DVT. These changes are formally called the Virchow triad. This triad includes hypercoagulability, venous stasis, and injury to the vessel wall. At least two of the triad must be present for a DVT to form. Essentially, an injury to the vessel wall causes inflammation that attracts platelets, especially in a state of altered coagulation. The thrombus that forms spreads in the direction of blood flow and additional layers of platelets are added to the thrombus as time progresses. As it grows, the vessel becomes further occluded and the client becomes symptomatic.

PREDISPOSING FACTORS

A. Age 60 years and older.
B. Hip or femur fracture.
C. Recent surgery, especially cardiac or extremity surgery.
D. Prolonged inactivity/immobility.
E. Pregnancy.
F. Medication:
  1. Hormone replacement therapy (HRT).
  2. Oral contraceptives.
  3. Tamoxifen.
G. Smoking.
H. Obesity.
I. Cancer.
J. Inherited hypercoagulable conditions.

COMMON COMPLAINTS

A. Note: Symptoms of a DVT are usually unilateral and have a sudden onset.
  1. Extremity edema.
  2. Extremity pain.
  3. Increased temperature of extremity.
  4. Change in color or extremity.
  5. Asymptomatic, depending on the size and location of the thrombus.

POTENTIAL COMPLICATIONS

A. PE.
B. Arterial embolism with arteriovenous shunting.
C. Myocardial infarction (MI).
D. Chronic venous insufficiency (CVI).
E. Postphlebitic syndrome.
F. Phlegmasia cerulea dolens.

SUBJECTIVE DATA

A. Ask client when the symptom were first noticed.
B. Have client describe duration of symptoms.
C. Ask client to describe pain (e.g., crushing, stabbing, or burning).
D. Ask the client what makes the symptoms better and what makes them worse.
E. Have client rate pain on a scale of 0 to 10, with 0 being no pain.
F. Ask client to list all medications currently being taken, particularly substances not prescribed and illicit drugs such as cocaine.
G. Review recent history of invasive procedures or surgery.

PHYSICAL EXAMINATION

A. Clients presenting with acute shortness of breath (SOB) and/or chest pain should be quickly assessed for the need to call emergency services/911 for immediate transport to the hospital.
B. Clients presenting with symptoms of DVT that include cyanosis of the distal extremity should be quickly assessed for the need to call emergency services/911 for immediate transport to the hospital.
C. Check vital signs.

▶

1. Check blood pressure (BP) and document resting heart rate, respirations, height, and weight.

D. Inspect:
1. Assess for signs of erythema, increased temperature, and edema.
2. Assess for a Homans sign (i.e., calf pain with forced plantar flexion).
3. Assess for Moses or Bancroft sign (i.e., pain when calf muscle is compressed forward against the tibia).
4. Assess Lisker sign (i.e., pain upon tibial percussion).

E. Palpate:
1. Palpate pulses distal to affected area, noting symmetry.
2. Palpate capillary refill.
3. Palpate extremity for tenderness. Do not perform deep palpation.

F. Auscultate:
1. Auscultate heart: rate, rhythm, heart sounds, murmur, and gallops.
2. Auscultate lungs: lung sounds in all fields.

DIAGNOSTIC TESTS

A. Serum laboratory testing:
1. D-dimer.
2. Complete blood count (CBC) with differential.
3. Coagulation panel (prothrombin time [PT], partial thromboplastin time [PTT], international normalized ratio [INR]).
4. Testing for idiopathic DVT: Add factor V Leiden, homocysteine, G20210A prothrombin, factor VIII, lupus anticoagulant, protein C and S levels, anticardiolipin antibodies, and antithrombin.

B. Compression ultrasound.

C. MRI, if thrombus is suspected in the pelvic veins or vena cava.

D. Venography, not utilized often due to expense and risk of phlebitis.

DIFFERENTIAL DIAGNOSES

A. DVT.
B. Cellulitis.
C. Fracture.
D. Lymphedema.
E. Congestive heart failure (CHF).
F. Vein compression (caused by enlarged lymph nodes or mass).
G. Filariasis.
H. Allergic reaction, localized.
I. Compartment syndrome.

PLAN

A. Client teaching: *See Client Teaching Guide for this chapter*, "*Deep Vein Thrombosis*":
1. Clients taking Coumadin should be educated regarding foods that are high in vitamin K.
2. Avoid prolonged standing or sitting.
3. Avoid crossing the legs.
4. Gradually resume normal activity.
5. Avoid immobility.
6. Exercise on a regular basis.
7. Stop smoking.
8. Wear compression stockings frequently.
9. Encourage strategies to better manage other chronic medical conditions that directly affect the progression of peripheral arterial disease (PAD), that is, diabetes, dyslipidemia, obesity, and hypertension (HTN).

B. Nonpharmaceutical therapy:
1. Compression stockings.

C. Pharmaceutical therapy:
1. Thrombolytics: administered in the inpatient setting.
2. Anticoagulants:
   a. Heparin, intravenous (IV): administered in the inpatient setting.
   b. Warfarin sodium (Coumadin; Jantoven):
      i. Doses: 1, 2, 2.5, 3, 4, 5, 6, 7.5 , and 10 mg tablets.
      ii. See the "Atrial Fibrillation" section for specifics on Coumadin therapy.
3. Low-molecular-weight heparin (LMWH):
   a. Enoxaparin sodium (Lovenox):
      i. Doses (multidose vial): 300 mg/3 mL.
      ii. Doses (prefilled syringe): 30 mg/0.3 mL, 40 mg/0.4 mL, 60 mg/0.6 mL, 80 mg/0.8 mL, 100 mg/mL, 120 mg/0.8 mL, and 150 mg/mL.
   b. Dalteparin sodium (Fragmin):
      i. Doses (multidose vial): 95,000 IU/9.5 mL.
      ii. Doses (by injection): 2,500 IU/0.2 mL, 5,000 IU/0.2 mL, 7,500 IU/0.3 mL, 10,000 IU/0.4 mL, 10,000 IU/mL, 12,500 IU/0.5 mL, 15,000 IU/0.6 mL, and 18,000 IU/0.72 mL.
4. Novel anticoagulants:
   a. Rivaroxaban (Xarelto):
      i. 2.5, 10, 15, and 20 mg tablets.
      ii. Dosage indications based on creatinine clearance (CrCl).
         1) CrCl >50 mL/min: 20 mg with evening meal.
         2) CrCl of 15 to 50 mL/min: 15 mg with evening meal.
   b. Apixaban (Eliquis):
      i. 2.5 and 5 mg tablets.
      ii. Dosage indications:
         1) Recommended dose is 2.5 mg, taken BID in clients who fit any of the following criteria:
            a) 80 years of age or older.
            b) Weight ≥60 kg (132 lbs).
            c) Serum creatinine ≥1.5 mg/dL.
   c. Edoxaban (Savaysa):
      i. 15, 30, and 60 mg tablets.
      ii. Dosage indications:
         1) CrCl between 15 and 50 mL/min: 30 mg, once daily.
         2) CrCl >95 mL/min: Do not prescribe.
   d. Betrixaban (Bevyxxa):
      i. 40 and 80 mg.
      ii. Initial dose of 160 mg, then maintenance dose of 80 mg daily, with the duration of therapy being 35 to 42 days.
5. Thrombin inhibitors:
   a. Bivalirudin (Angiomax): 250 mg per vial, add 5 mL of sterile water for injection; recommended IV bolus of 0.75 mg/kg followed by an infusion of 1.75 mg/kg/hr.
   b. Argatroban (Acova): 2 mcg/kg/min in constant infusion until PTT is 1.5 to 3 times the initial baseline PTT value.
   c. Dabigatran etexilate mesylate (Pradaxa):
      i. 75, 110, and 150 mg tablets.
      ii. Dosage indications based on CrCl:
         1) CrCl >30 mL/min: 150 mg, taken BID.
         2) CrCl of 15 to 30 mL/min: 75 mg, taken BID.
   d. Thrombate III (antithrombin III).
6. Specific factor Xa inhibitor:

a. Fondaparinux sodium (Arixtra):
i. Doses: 2.5 mg/0.5 mL, 5 mg/0.4 mL, 7.5 mg/0.6 mL, and 10 mg/0.8 mL by injection.

D. Surgery:
1. Insertion of vena cava filter to prevent PE.
2. Venous thrombectomy.

FOLLOW-UP

A. Follow-up is determined by client's needs, frequency and intensity of symptoms, and presence of other medical conditions.
B. DVT manifesting persistent symptoms should always be followed by a cardiologist.
C. Clients taking anticoagulants are best followed by an anticoagulant clinic and/or cardiologist.

EMERGENT ISSUES/INSTRUCTIONS

A. Clients should go to the ED if they have any of the following symptoms:
1. Sudden SOB.
2. Chest pain.
3. A change in color of a foot or hand.
4. If taking an anticoagulant and have any of the following symptoms:
a. **Nosebleed that will not stop with pressure.**
b. **Coughing up blood.**

B. Clients should contact the office if they are experiencing any of the following:
1. Fever over 101°F.
2. Increased redness, pain, tenderness to touch, swelling, and/or warmth.
3. New wound or sore on the affected arm or leg.
4. If taking an anticoagulant and have any of the following symptoms:
a. **Vomiting or other illness causes them to miss more than one dose of their medications.**
b. **Vomit that is bright red or dark and looks like coffee grounds or grape jelly.**
c. **Bright red blood in their stools, or black, tarry stools.**

C. Clients should contact the office if they are experiencing any new symptom(s) not present at their last office visit.

CONSULTATION/REFERRAL

A. If you suspect acute limb ischemia, refer client for immediate hospitalization to obtain diagnostic testing to determine the presence of a thrombus and restore circulation to the affected extremity.
B. If chronic limb ischemia has led to ulceration and/or superimposed infection, hospitalization is indicated to initiate wound care consultation and diagnostic testing to determine the degree of arterial occlusion.
C. Referral to a cardiologist is indicated in the presence of persistent PVD symptoms.
D. Refer to pain management if pain is resistant to treatment.
E. Refer to a registered dietitian as indicated by the client's understanding of dietary modification necessary to improve status of risk factors.

INDIVIDUAL CONSIDERATIONS

A. Nonambulatory clients:
1. Using rocking chairs is a possible substitute for persons unable to participate in a walking program.
B. Geriatrics:
1. Be alert to the following geriatric syndrome: depression related to immobility and pain.

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HEART FAILURE

DEFINITION

A. Heart failure (HF) is failure of the heart to pump sufficient blood to meet the metabolic demands of the tissues. The 2017 focused update of the 2013 guidelines from the American College of Cardiology/American Heart Association (ACC/AHA) addresses management of heart failure with reduced ejection fraction (HFrEF) and heart failure with preserved ejection fraction (HFpEF). These guidelines include the following:
1. HFrEF:
a. Ejection fraction (EF) less than 40%, due to weak, inefficient systolic contractions. The left ventricular ejection fraction (LVEF) is a measurement of systolic failure.
b. S3 ventricular gallop rhythm commonly occurs.
c. Systolic failure is often the result of coronary heart disease (CHD) and/or myocardial infarction (MI).
d. It can result from right- or left-sided failure, or both.
2. HFpEF:
a. EF greater than 50%, but with poor compliance of the ventricle, which impedes ventricular diastolic filling (the ventricle is unable to relax).
b. There are two subgroups:
i. HFpEF borderline (or intermediate): clients with an EF of 41% to 49%.
ii. HFpEF with EF greater than 40% who previously had HFrEF (EF <40%), but improvement or recovery was noted in EF.
c. The client may be asymptomatic for years.
d. HF presents with the same symptoms of systolic failure, pulmonary congestion, and peripheral edema.
e. S4 atrial gallop rhythm commonly occurs.

INCIDENCE

A. HFrEF incidence is higher in older females, chronic hypertension (HTN), obesity, left ventricular (LV) hypertrophy, cardiomyopathy, excessive alcohol use, end-stage chronic obstructive pulmonary disease (COPD), valvular disorders, anemia, renal failure, atrial fibrillation (AF), coronary artery disease (CAD), or diabetes. ▶

B. Approximately 5.7 million people in the United States have HF. By the year 2030, the AHA estimates that there will be a 46% increase in HF in clients with chronic diseases.
C. Americans over 40 have a 20% lifetime risk of developing HF.
D. There are 650,000 new clients diagnosed yearly.
E. The 5-year mortality rate is 50% from the time of diagnosis and the 1-year mortality increases to 50% if the client has multiple admissions for HF in the same year.
F. There are one million hospitalizations per year, and according to the Centers for Medicare and Medicaid Services (CMS) the rate of readmissions within 1 month nationally is 23% to 25%.
G. The current cost to treat HF exceeds $30 billion yearly, and it is also estimated that the costs of direct client care (e.g., healthcare services, hospitalizations, medications) for these clients with chronic disease will increase to approximately $53 billion by the year 2030.

PATHOGENESIS

A. Injuries to the myocardium may cause loss of functioning muscle. Compensatory mechanisms, including cardiac hypertrophy and neurohumoral processes, lead to adverse long-term effects. An inotropic insult results in incomplete emptying (systolic failure), and a compliance abnormality results in incomplete filling (diastolic failure). Most HFs have some degree of both abnormalities.

PREDISPOSING FACTORS

A. Atherosclerotic heart disease.
B. MI.
C. Rheumatic heart disease involving mitral and aortic valves.
D. Cardiomyopathies.
E. Hypertensive heart disease.
F. Aortic stenosis or regurgitation.
G. Thyrotoxicosis.
H. Pregnancy-related disorders, such as multiple births with preexisting heart disease.
I. Volume overload.
J. Beta-blockers or other cardiac depressants.
K. Pulmonary embolism (PE).
L. Systemic infection.
M. Arrhythmias.
N. Renal disease.

COMMON COMPLAINTS

A. Clients are assigned the New York Heart Association (NYHA) classifications by their tolerance of physical activity and shortness of breath (SOB). This classification may change according to the progression or regression of their cardiovascular disease (CVD; Table 13.4).
B. Dyspnea on exertion.
C. Hemoptysis.
D. Fatigue.
E. Cough.
F. Orthopnea.
G. Edema/weight gain.
H. Paroxysmal nocturnal dyspnea.
I. Nausea.
J. Right upper abdominal pain or fullness.
K. Chest pain.
L. Palpitations.

TABLE 13.4 FUNCTIONAL CLASSIFICATION FOR HEART FAILURE

Functional Class	Activities	Objective Assessment
Functional capacity class I Objective assessment A	No limitation No undue fatigue, palpitation, dyspnea, or angina with ordinary activity	No objective evidence of CV disease
Functional capacity class II Objective assessment B	Slight limitations Fatigue, palpitation, SOB, and angina with ordinary physical activity	Objective evidence—minimal CV disease
Functional capacity class III Objective assessment C	Marked limitations No discomfort at rest Fatigue, palpitation, SOB, and angina with less than usual activities	Objective evidence—moderately severe CV disease
Functional capacity class IV Objective assessment D	Inability to do physical activity without discomfort Symptoms present at rest and become worse with activity	Objective evidence—severe CV disease

CV, cardiovascular; SOB, shortness of breath.

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OTHER SIGNS AND SYMPTOMS

A. Hemoptysis.
B. Bibasilar crackles.
C. S3 gallop.
D. Murmurs.
E. Exercise intolerance.
F. Weakness.
G. Cough.
H. Orthopnea.
I. Nocturnal dyspnea.
J. Tachycardia.
K. Pallor.
L. Cyanosis.
M. Anorexia.
N. Constipation.
O. Jugular venous distention (JVD).
P. Hepatomegaly.
Q. Hepatojugular reflux (HJR).
R. Murmurs.
S. Exercise intolerance.

SUBJECTIVE DATA

A. Ask the client if they have difficulty breathing.
B. Ask how many pillows they sleep on. Does the client need to sit up in a recliner to sleep?
C. Inquire about how often they wake up at night with SOB.
D. Inquire about how far the client can walk without getting SOB. Have the client describe their routine activities of daily living and how well they tolerate each activity.
E. Discuss the client's history of heart disease, heart attack, HTN, or hyperlipidemia.

F. Ask the client about current medications, as well as prescription, over-the-counter (OTC), and herbal products.
G. Question the client regarding all symptoms found in the "Common Complaints" section.
H. Discuss drug and alcohol history.
I. Has the client ever been treated for cancer/chemotherapy, and how long ago?
J. What is the client's usual weight? Have they experienced more symptoms if they have pedal edema?
K. Does the client have a cough? (Consider angiotensin-converting enzyme inhibitors (ACEIs) as the cause.)

PHYSICAL EXAMINATION

A. Check pulse, respirations, blood pressure (BP), pulse oximetry, height, and weight.
1. Check BP sitting, standing, and lying down.
2. Be alert for abnormal vital signs: hypotension, narrow or wide pulse pressure, tachycardia, bradycardia, and tachypnea.
3. Calculate body mass index (BMI).
4. On all subsequent visits, note weight gain of more than 1 lb per day over 3 consecutive days or 3 lbs in 1 day.
B. Inspect:
1. Inspect overall physical appearance. Is the client in distress?
2. Inspect skin: Note pallor, cyanosis, and temperature.
3. Inspect neck: Check jugular veins for distension.
4. Inspect extremities: Note edema, cyanosis, pallor, and ulcers.
C. Palpate:
1. Palpate abdomen for hepatomegaly and HJR.
2. Palpate extremities for peripheral pulses.
3. Palpate chest wall for displaced point of maximal impulse (PMI), lifts, heaves, and thrills.
D. Auscultate:
1. Auscultate heart for murmurs; tachycardia; S1, S3, or S4 gallops; and other abnormalities.
2. Auscultate lungs: Note moderate-to-severe crackles/rales and other abnormal sounds.
3. Auscultate the neck and carotid arteries.
E. Mental status:
1. Check mental status because confusion may occur, especially in the elderly.

DIAGNOSTIC TESTS

A. Two-dimensional echocardiography with Doppler to evaluate LVEF.
B. Radionuclide ventriculography may be used to measure LVEF and LV volumes.
C. Coronary angiography.
D. Anterior/posterior chest x-ray.
E. EKG for clients with suspected arrhythmia, ischemia, or cardiac disease. Identify acute and old EKG changes to rule out pathologic Q wave, ST-segment elevation, and LV hypertrophy.
F. Natriuretic peptides (brain natriuretic peptide or N-terminal pro-brain natriuretic peptide).

DIFFERENTIAL DIAGNOSES

A. HF:
1. HFrEF.
2. HFpEF:
a. HFpEF borderline clients with an EF of 41% to 49%.
b. HFpEF clients with an EF greater than 40% who previously had HFrEF (EF, 40%) with improvement noted in EF.
B. Renal disease or nephrotic syndrome.
C. Liver disease.
D. Asthma.
E. COPD: To distinguish between progressing HF and a COPD exacerbation when both conditions are present, the presence of weight gain and an S3 gallop indicates HF, not COPD.

PLAN

A. General interventions according to the 2017 focused update of the ACC/AHA guidelines for HF:
1. Determine the etiology of the failure state and treat appropriately.
2. Treat HF stages:
a. Stage A: Treat/manage the client's underlying conditions (HTN, AF, hyperlipidemia, diabetes, tobacco cessation, obesity, substance abuse [alcohol, cocaine, etc.]).
b. Stage B: Begin ACEIs or angiotensin receptor blockers (ARBs), if intolerant to ACEIs, along with beta-blockers for clients with HFrEF. Add statin therapy if client has a history of MI. If the client has signs of water retention or has a history of water retention, diuretics should be started.
c. Stage C: HFrEF: implantable cardioverter defibrillators and cardiac resynchronization therapy:
i. Digoxin may be added if the client is still symptomatic and has been optimized on guideline-directed therapy to help manage symptoms.
ii. Hydralazine and isosorbide dinitrate are indicated for African American clients with HFrEF, clients with kidney dysfunction, or clients who cannot take ACEI or ARB therapy.
iii. Aldosterone receptor antagonist may be considered in the following scenarios:
1) HFrEF less than 30% or 30% to 35% with QRS greater than 130 ms.
2) Post-MI with EF less than 40% + (HF symptoms or diabetes) NYHA II symptoms, and serum creatinine less than 2.5 mg/dL (glomerular filtration rate [GFR] >30 mL/min), and serum potassium less than 5.
iv. Angiotensin receptor-neprilysin inhibitors (ARNIs) help prevent degradation of natriuretic peptides, bradykinin, and other vasoactive peptides. Research shows a reduction in composite endpoints of cardiovascular death or hospital admissions by 20%.
1) Prior to starting, a washout of the previous ACEI is needed. Do not start within 36 hours of last dose of an ACEI.
d. Stage D: advanced treatments such as cardiac transplant, mechanical support, home-based inotropic therapy, and/or hospice care.
3. The goal of therapy is to improve the client's quality of life by reducing symptoms, decreasing morbidity, and prolonging survival.
B. Client teaching:

▶

1. Teach the client to weigh daily at the same time, on the same scale, and in the same clothing. The client is to call if there are gains of more than 2 to 3 lbs in 1 day or more than 5 lbs in 1 week:
   a. Develop a plan of care for the client to increase the diuretic dosing for edema/weight gain to reduce dyspnea and prevent hospitalization. The directions could be to take an extra dose of the diuretic each day for several days until the weight gain, swelling, or symptoms improve.
2. Encourage weight loss if overweight.
3. Recommend regular, moderate exercise as long as dyspnea is not induced. Encourage exercise even though the client may only tolerate a few minutes of walking to increase endurance and strengthen muscles.
4. Encourage smoking cessation.
5. Encourage medication adherence. Instruct the client about all medications and possible side effects. Do not use OTC medicines without consulting the provider. Nonsteroidal anti-inflammatory drugs (NSAIDs) are contraindicated in HF.
6. Utilize continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP) for nighttime sleep and naps for treatment of obstructive sleep apnea (OSA).

C. Dietary management:
1. Read food labels for sodium content.
2. Teach dietary modifications, especially salt restriction of 2,000 to 3,000 mg/d for HFrEF and HFpEF. Less than 2,000 mg/d of sodium is recommended in clients with moderate to severe HF symptoms.
3. Fluid restriction is not recommended unless the client is classified as stage D or is diagnosed with hyponatremia, with sodium levels less than 130 mEq/L. Restrict fluid intake to 2,000 mL/d or less for clients with chronic fluid retention despite use of diuretics and sodium restrictions.
4. Alcohol consumption should be limited to one glass of beer or wine per day. This amount should be counted in the daily fluid restriction.
5. See Appendix B for the Dietary Approaches to Stop Hypertension (DASH) diet (Tables B.1–B.5).
6. Other diet changes include low-fat/low-cholesterol and the use of the following:
   a. Monounsaturated fats, which decrease cholesterol:
      i. Canola oil.
      ii. Olive oil.
   b. Polyunsaturated fats, which decrease cholesterol but not as well as monounsaturated fats:
      i. Vegetable and fish oils.
      ii. Corn, safflower, peanut, and soybean oils.
   c. Avoid saturated fats:
      i. Animal fats and some plant fats.
      ii. Butter and lard.
      iii. Coconut oil and palm oil.

D. Pharmaceutical therapy:
1. Therapy is based on extent of cardiac impairment and severity of symptoms. Medication regimens include a combination of the following classes: diuretics, ACEIs (ARBs if unable to tolerate ACEIs), cardioselective beta-blockers, inotropic agents such as digoxin, vasodilators, nitrates, and anticoagulants if there is an increased risk of thrombus formation. In general, calcium channel blockers (CCBs) are not used in HF management.
2. Drug classifications for antihypertensive and cardiac medication are noted in Table 13.5.
3. Drugs that should be avoided and/or used with caution in HF because they cause exacerbations or are at high risk for adverse reactions include the following:
   a. NSAIDs: increased renal dysfunction, edema, impaired response to ACEI.
   b. Cyclooxygenase (COX-2) inhibitors: increased rate of HF and increased mortality.
   c. Aspirin: interaction between aspirin (ASA) and ACEI and interference with benefits of beta-blockers on LVEF.
   d. Metformin: increased risk of lethal lactic acidosis.
   e. Thiazolidinediones: fluid retention that may precipitate HF.
   f. CCBs: some data on possible effect on systolic dysfunction.
   g. Antidepressants tricyclic antidepressants (TCAs) and selective serotonin reuptake inhibitors (SSRIs): major adverse cardiovascular events including HF, MI, stroke, and cardiovascular death.
   h. Phosphodiesterase inhibitors (PDE3, PDE4, and PDE-5): increased mortality.
   i. Antiarrhythmic: negative inotropic activity and further reduction in LV function can impair the elimination and result in toxicity of the antiarrhythmic.
   j. Chemotherapy: many are cardiotoxic.
   k. Androgens-testosterone patch: edema that may accelerate the rate of HF.
   l. Theophylline serum levels may increase and cause toxicity due to acute decompensation of HF.
   m. Sodium bicarbonate and Fleet Phospho-Soda contain significant quantities of sodium.
   n. Herbal/supplements may also affect HF.
4. Biventricular pacing or an implantable defibrillator may be necessary for advanced HF.
5. Advanced HF therapies can include the following:
   a. Inotropic therapy in hospital and at home.
   b. Implanted mechanical-assist devices as a bridge to transplantation or as a destination therapy.
   c. Heart transplantation.
6. Discuss the client's desire for an evaluation as a heart transplant candidate.
7. Pneumonia vaccination as well as yearly influenza immunization should be encouraged.
8. Discuss the need for compassionate care/hospice when HF does not respond despite maximal therapy.

FOLLOW-UP

A. HF clients should be comanaged with a physician.

B. Close follow-up is essential if the client is to be maintained as an outpatient.

C. Appointments every 1 to 2 weeks may be necessary, with additional appointments depending on the client's symptoms, such as increasing SOB, inability to lie flat to sleep, nocturnal moist cough, and increase in daily weight.

D. Lab monitoring is required for electrolytes, blood urea nitrogen (BUN), creatinine, proteinuria, and digoxin level.

CONSULTATION/REFERRAL

A. Consult a physician when the client requires the next level up in pharmacologic management.

B. Consult a cardiologist for staging and hospital management. ▶

TABLE 13.5 ANTIHYPERTENSIVE AND CARDIAC MEDICATIONS (SORTED ALPHABETICALLY)

Brand Name	Generic Name	Drug Class
Accupril	Quinapril	ACE inhibitor
Aceon	Perindopril erbumine	ACE inhibitor
Adalat CC	Dihydropyridine/nifedipine	CCB
Aldactazide	Spironolactone + HCTZ	Combination: K + sparing + thiazide
Aldactone	Spironolactone	Diuretic: K + sparing
Altace	Ramipril	ACE inhibitor
Amiloride/HCTZ	Amiloride/HCTZ	Combination + diuretic
Atacand	Candesartan cilexetil	ARB
Atacand HCT	Candesartan + HCTZ	Combination: ARB + diuretic
Avalide	Irbesartan + HCTZ	Combination: ARB + diuretic
Avalide Avapro	Irbesartan + HCTZ Irbesartan	Combination: ARB + diuretic ARB
Azor	Amlodipine + olmesartan	CCB + ARB
Benicar	Olmesartan medoxomil	ARB
Benicar HCT	Olmesartan + HCTZ	Combination: ARB + diuretic
Betapace	Sotalol	Beta-blocker/class II and III antiarrhythmic
Betapace AF	Sotalol	Class II and III antiarrhythmics
Betaxolol Bidil	Betaxolol HCL isosorbide dinitrate + hydralazine	Beta-blocker: cardioselective nitrate + diuretic
Bumex	Bumetanide	Loop diuretic
Bystolic	Nebivolol	Beta-blocker: cardioselective
Caduet	Amlodipine + atorvastatin	CCB/amlodipine/atorvastatin
Calan	Verapamil	CCB/antianginal
Calan SR	Verapamil	CCB
Capoten	Captopril	ACE inhibitor/CHF
Capozide	Captopril + HCTZ	Combination: ACE + diuretic
Cardizem LA	Diltiazem	CCB
Cardura	Doxazosin	Alpha-1 receptor antagonist (alpha-blocker)
Catapres	Clonidine	Central alpha agonist
Cleviprex	Dihydropyridine	CCB
Cordarone	Amiodarone	Class II and III antiarrhythmics
Coreg CR	Carvedilol	Beta-blocker: noncardioselective/CHF
Corgard	Nadolol	Beta-blocker: noncardioselective/antianginal
Corlanor	Ivabradine	Hyperpolarization-activated cyclic nucleotide-gated channel blocker
Corzide	Nadolol + HCTZ	Combination: beta-blocker (noncardioselective) + diuretic
Covera HS	Verapamil	CCB/antianginal

(*continued*)

TABLE 13.5 ANTIHYPERTENSIVE AND CARDIAC MEDICATIONS (SORTED ALPHABETICALLY) (*CONTINUED*)

Brand Name	Generic Name	Drug Class
Cozaar	Losartan	ARB
Demadex	Torsemide	Loop diuretic
Digoxin	Digoxin	Cardiac glycoside/antiarrhythmic
Dilacor XR	Diltiazem	CCB/antianginal
Dilatrate SR	Isosorbide dinitrate	Nitrate
Diovan	Valsartan	ARB/CHF class II–IV
Diovan HCT	Valsartan + HCTZ	Combination: ARB + diuretic
Diuril	Chlorothiazide	Thiazide diuretic
Dutoprol Dyazide	Metoprolol extended-release + HCTZ Triamterene + HCTZ	Beta-blocker (cardioselective) + thiazide Combination: K + sparing + thiazide
Dynacirc CR	Isradipine controlled-release	CCBr
Edarbi	Azilsartan	ARB
Edarbyclor	Azilsartan + chlorthalidone	ARB + diuretic
Edecrin	Ethacrynic acid	Loop diuretic
Entresto	Sacubitril + valsartan	Neprilysin inhibitor + ARB
Exforge	Amlodipine + valsartan	CCB + ARB
Fosinopril HCTZ	Various hydrochlorothiazide	ACE inhibitor, thiazide diuretic
Hytrin	Terazosin	Alpha-1 blocker
Hyzaar	Losartan potassium + HCTZ	Combination: ARB + diuretic
Imdur	Isosorbide dinitrate	Nitrate
Inderal	Propranolol	Beta-blocker: noncardioselective/antianginal/antiarrhythmic
Inderide	Propranolol + HCTZ	Combination: beta-blocker (noncardioselective) + diuretic
InnoPran XL	Propranolol HCL extended-release	Beta-blocker: noncardioselective
Inspra	Eplerenone	Aldosterone receptor blocker: mineralocorticoid selective/CHF
Ismo	Isosorbide dinitrate	Nitrate
Isoptin SR	Verapamil	CCB
Kerlone	Betaxolol	Beta-blocker: cardioselective
Lanoxin	Digoxin	Cardiac glycoside/antiarrhythmic
Lasix	Furosemide	Loop diuretic
Levatol	Penbutolol sulfate	Beta-blocker: noncardioselective
Lopressor	Metoprolol tartrate	Beta-blocker: cardioselective/antianginal
Lopressor HCT	Metoprolol + HCTZ	Combination: beta-blocker (cardioselective) + diuretic
Lotensin	Benazepril	ACE inhibitor
Lotensin HCT	Benazepril + HCTZ	Combination: ACE + diuretic
Lotrel	Amlodipine + benazepril	Combination: CCB + ACE inhibitor
Mavik	Trandolapril	ACE inhibitor/CHF

(*continued*)

TABLE 13.5 ANTIHYPERTENSIVE AND CARDIAC MEDICATIONS (SORTED ALPHABETICALLY) (*CONTINUED*)

Brand Name	Generic Name	Drug Class
Maxzide	Triamterene + HCTZ	Combination: K + sparing + thiazide
Mexitil	Mexiletine	Class IB antiarrhythmic
Micardis	Telmisartan	ARB
Micardis HCT	Telmisartan + HCTZ	Combination: ARB + diuretic
Microzide	HCTZ	Diuretic
Minitran	Nitroglycerin	Nitrate
Monoket	Isosorbide dinitrate	Nitrate
Monopril	Fosinopril	ACE inhibitor/CHF
Multaq	Dronedarone	Antiarrhythmic
Nicardipine	Various	CCB
Nitro-BID	Nitroglycerin	Nitrate
Nitro-Dur	Nitroglycerin	Nitrate
Nitrolingual	Nitroglycerin	Nitrate
Nitrostat	Nitroglycerin	Nitrate
Normodyne	Labetalol	Beta-blocker
Norpace	Disopyramide	Antiarrhythmics: class I-ventricular
Norvasc	Amlodipine	CCB/antianginal
Pindolol	Pindolol	Beta-blocker: noncardioselective
Plendil	Felodipine	CCB
Prinivil	Lisinopril	ACE inhibitor/CHF
Prinzide	Lisinopril + HCTZ	Combination: ACE inhibitor + thiazide diuretic
Procanbid	Procainamide	Antiarrhythmics: class Ia-ventricular
Procardia XL	Nifedipine	CCB/antianginal
Quinidine gluconate	Various	Antiarrhythmics: class I atrial and ventricular
Quinidine sulfate	Various	Antiarrhythmics: class I atrial and ventricular
Ranexa	Ranolazine	Antianginal
Rythmol	Propafenone	Antiarrhythmics: class Ic-ventricular
Sectral	Acebutolol	Beta-blocker: cardioselective
Sular	Nisoldipine	CCBr
Tambocor	Flecainide	Antiarrhythmics: class Ic-ventricular
Tarka	Trandolapril + verapamil	Combination: ACE + CCB
Tekamlo	Aliskiren + amlodipine	Direct renin inhibitor + DHP CCBr
Tekturna	Aliskiren	Direct renin inhibitor
Tekturna HCT	Aliskiren + HCTZ	Direct renin inhibitor + thiazide diuretic
Tenex	Guanfacine	Central alpha agonist

(*continued*)

TABLE 13.5 ANTIHYPERTENSIVE AND CARDIAC MEDICATIONS (SORTED ALPHABETICALLY) (*CONTINUED*)

Brand Name	Generic Name	Drug Class
Tenoretic	Atenolol + chlorthalidone	Combination: beta-blocker (cardioselective) + diuretic
Tenormin	Atenolol	Beta-blocker: cardioselective/antianginal
Teveten	Eprosartan	ARB
Teveten HCT	Eprosartan + HCTZ	Combination: ARB + diuretic
Thalitone	Chlorthalidone	Antihypertensive/diuretic
Tiazac	Diltiazem	CCB/antianginal
Tikosyn	Dofetilide	Class III antiarrhythmic
Timolide	Timolol maleate + HCTZ	Combination: beta-blocker (noncardioselective) + diuretic
Toprol XL	Metoprolol	Beta-blocker: cardioselective/antianginal/CHF class II or III
Trandate	Labetalol	Beta-blocker: noncardioselective
Tribenzor	Olmesartan + amlodipine + HCTZ	ARB + CCB + thiazide diuretic
Twynsta	Telmisartan + amlodipine	ARB + CCB
Uniretic	Moexipril + HCTZ	Combination: ACE + diuretic
Univasc	Moexipril	ACE inhibitor
Valturna	Aliskiren + valsartan	Direct renin inhibitor + ARB
Vaseretic	Enalapril + HCTZ	Combination: ACE + diuretic
Vasotec	Enalapril	ACE inhibitor/CHF with digitalis and diuretics
Verelan PM	Verapamil	CCB
Zaroxolyn	Metolazone	Diuretic: quinazoline
Zebeta	Bisoprolol	Beta-blocker: cardioselective
Zestoretic	Lisinopril + HCTZ	Combination: ACE + diuretic
Zestril	Lisinopril	ACE inhibitor/CHF with digitalis and diuretics
Ziac	Bisoprolol + HCTZ	Combination: beta-blocker (cardioselective) + diuretic

ACE, angiotensin-converting enzyme; ARB, angiotensin II receptor blocker; CCB, calcium channel blocker; CHF, congestive heart failure; DHP, dihydropyridine; HCL, hydrochloride; HCTZ, hydrochlorothiazide, HR, heart rate, SA node, sinoatrial node.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. HF is uncommon in healthy females without coexisting heart disease.

2. Pregnancy in a client with heart disease is considered high risk.

3. Refer to a high-risk obstetrician.

B. Adults:

1. The 5-year survival rate is about 50% for both males and females.

2. Predictors of poor outcome include an EF of less than 25%, ischemic etiology, ventricular arrhythmias, serum sodium greater than 130 mEq/L, poor functional class, low cardiac index, and high filling pressures.

C. Geriatrics:

1. Common signs and symptoms of HF are less specific in older adults and atypical symptoms may predominate.

2. Age-associated changes in pharmacokinetics must be taken into account when prescribing drugs for HF.

3. Effective communication among health professionals, clients, and families is necessary.

4. Given the life-limiting nature of HF in frail older adults, it is critical for clinicians to discuss end-of-life issues with clients and their families as soon as possible. The Medicare hospice benefit is granted to clients who have been certified by two physicians to have a life expectancy of 6 months or less if their terminal illness runs its natural course. The criteria for determining that HF is terminal are:

a. NYHA class III (symptomatic with less than ordinary activities) or IV (symptomatic at rest).

b. LVEF less than or equal to 20%.

c. Persistent symptoms despite optimal medical management.

d. Inability to tolerate optional management because of hypotension with or without renal failure.

5. Geriatric red flag:

a. For the older adult with existing cognitive, functional, or mobility impairments, the nurse practitioner (NP) should practice special consideration of the following geriatric syndromes if adding or increasing a diuretic:
   i. Incontinence.
   ii. Falls.
      1) Avoid diuretics after 4 p.m. to reduce the risk of falls from attempting to get to the bathroom during the night. Discuss ways the client can adjust fluid intake and toileting habits. Alert the client that avoidance of incontinent episodes by reducing oral intake could result in dehydration and/or urinary tract infection.

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HYPERTENSION

DEFINITION

A. Discord exists between the American College of Cardiology/American Heart Association (ACC/AHA) (2017) and the Eighth Joint National Committee (JNC 8) guideline (2014) as to the definition and stages of hypertension (HTN). The JNC 8 guideline provided more permissive (higher) blood pressure (BP) goals, especially for those 60 and older. The newer AHA/ACC guideline defines HTN as a systolic blood pressure (SBP) of 130 mmHg or more, a diastolic blood pressure (DBP) of 80 mmHg or more, or describes a condition in which a person is taking antihypertensive medications.

B. Resistant HTN is defined as follows:

1. BP that is not at target despite a three-drug regimen, including an angiotensin-converting enzyme inhibitor (ACEI) or an angiotensin receptor blockers (ARBs) + a calcium channel blocker (CCB) + a diuretic appropriate for the client's glomerular filtration rate (GFR).

2. Controlled BP while taking four or more medications is also considered resistant HTN.

C. Standing and supine BPs should be measured before the initiation of combination antihypertensive therapy. Orthostatic (postural) hypotension is diagnosed when, within 2 to 5 minutes of quiet standing, one or more of the following is present:

1. At least a 20 mmHg fall in systolic pressure.

2. At least a 10 mmHg fall in diastolic pressure.

3. Symptoms of cerebral hypoperfusion, such as dizziness.

D. The average nocturnal BP is approximately 15% lower than daytime values. Failure of BP to fall by at least 10% during sleep is called "nondipping" and is a stronger predictor of adverse cardiovascular outcomes than daytime BP.

E. Isolated systolic hypertension (ISH) is defined when the SBP is greater than or equal to 140, with the DBP normal or below normal (<90 mmHg). ISH usually affects the elderly, increasing their risk of stroke or myocardial infarction (MI).

F. Isolated diastolic hypertension (IDH) is defined as a diastolic pressure greater than or equal to 90 mmHg, with a systolic pressure less than 140 mmHg. IDH is more common in younger males who are overweight/obese and in individuals younger than 40 years.

G. Malignant HTN is marked HTN with retinal hemorrhages, exudates, or papilledema. Malignant HTN is usually associated with diastolic pressures above 120 mmHg.

INCIDENCE

A. Worldwide, HTN affects about one billion people and is the leading preventable risk factor for premature death and disability worldwide.

B. Approximately one in three adults in the United States have HTN.

C. The incidence of resistant HTN is rising. The data indicate a 21.7% incidence.

D. African American females have the highest death related to HTN (37.7 deaths per 100,000 population).

PATHOGENESIS

A. Over 90% of cases have no identifiable cause, thus constituting the category of primary or essential HTN. The remaining 10% of cases have the following secondary causes.

1. Renal causes:
   a. Glomerulonephritis.
   b. Pyelonephritis.
   c. Polycystic kidney disease.

2. Endocrine causes:
   a. Primary hyperaldosteronism.
   b. Pheochromocytoma.
   c. Hyperthyroidism.
   d. Cushing syndrome.

3. Vascular causes:
   a. Coarctation of aorta.
   b. Renal artery stenosis.

4. Obstructive sleep apnea (OSA).

5. Chemical-/medication-induced HTNs:
   a. Oral contraceptives.
   b. Nonsteroidal anti-inflammatory drugs (NSAIDs).
   c. Decongestants.
   d. Antidepressants.
   e. Sympathomimetics.
   f. Corticosteroids.
   g. Lithium.
   h. Ergotamine alkaloids.
   i. Cyclosporine.
   j. Monoamine oxidase inhibitors (MAOIs), in combination with certain drugs or foods.
   k. Appetite suppressants, in combination with certain drugs or foods.
   l. Cocaine.
   m. Amphetamines.

PREDISPOSING FACTORS

A. When making a diagnosis, consider not only the absolute BP reading, but also the presence or absence of other cardiovascular risk factors. Factors include the following:

1. Family history of HTN.

2. Obesity.

3. Alcohol consumption.

4. Stress.

▶

5. Sedentary lifestyle.
6. African American ancestry.
7. Male sex.
8. Age older than 30 years.
9. Excessive salt intake.
10. Medications.
11. Drug use.

COMMON COMPLAINT

A. HTN is asymptomatic in the majority of clients.

OTHER SIGNS AND SYMPTOMS

A. Headaches.
B. Advanced disease: organ-specific complaints with end-organ damage.
C. Retinopathy.

POTENTIAL COMPLICATIONS

A. Cerebrovascular accident (CVA).
B. MI.
C. Renal failure.
D. Heart failure (HF).
E. Peripheral arterial disease (PAD).

SUBJECTIVE DATA

A. Ask the client about any family history of HTN or cardiac or renal disease.
B. Ask if the client has ever been diagnosed with HTN or cardiac or renal disease.
C. Ask if the client ever had any high BP readings.
D. Ask if the client has ever been treated for any of the earlier problems.
E. Ask about other risk factors, such as smoking, drinking, high-fat intake, obesity, and/or diabetes.
F. Inquire about the client's lifestyle, exercise regimen, work environment, and stress level.
G. Ask the client about symptoms that suggest secondary etiology:
1. Palpitations, headache, diaphoresis (pheochromocytoma).
2. Anxiety, weight gain or loss (thyroid abnormality).
3. Muscle weakness, polyuria (primary aldosteronism).

H. Find out if the client is taking drugs that elevate BP (noted under the "Pathogenesis" section).
I. Ask if the client feels nervous when having their BP taken in the office ("white coat HTN").
J. Review current medications, including prescription, over-the-counter (OTC), and herbal products.
K. Review current recreational/illicit drug use.

PHYSICAL EXAMINATION

A. Check pulse, BP, height, weight, waist circumference, and distribution of body fat. Calculate body mass index (BMI).
1. The diagnosis of HTN is made after averaging two or more properly measured readings at each of two or more visits after an initial screen.
2. When client's SBP and DBP fall into two different categories, use the higher category to classify their BP.
3. For accurate measurement, use correct size of cuff for client (adult, large adult, or thigh cuff).

B. Inspect:
1. Observe overall appearance.
2. Conduct funduscopic exam; look for papilledema, exudates, arteriovenous nicking, anterior nicking.
3. Inspect the neck for jugular vein distension.
4. Observe for pedal edema.

C. Palpate:
1. Palpate the neck; check thyroid for enlargement.
2. Palpate the abdomen for masses or organomegaly.
3. Palpate the extremities; assess peripheral pulses and note edema.
4. Assess deep tendon reflexes (DTRs).

D. Auscultate:
1. Auscultate heart, noting the point of maximal impulse (PMI).
2. Auscultate lungs; check for bronchospasm and rales.
3. Auscultate neck; assess carotid arteries for bruits.

DIAGNOSTIC TESTS

A. Hematocrit.
B. Liver function tests (LFTs), lactate dehydrogenase (LDH), uric acid.
C. Chemistry profile.
D. Fasting lipid profile (total and high-density lipoprotein [HDL] cholesterol and triglycerides).
E. Urinalysis for proteinuria.
F. Estimated GFR.
G. EKG.
H. If history, physical exam, or lab tests indicate the need, obtain the following:
1. Intravenous pyelography (IVP).
2. Renal arteriogram.
3. Plasma renin.
4. Catecholamines.
5. Chest radiography.
6. Aortogram.
7. Ultrasonography.
8. Sleep study.

I. Monitor potassium levels if on ACEIs/ARBs or spironolactone.

DIFFERENTIAL DIAGNOSES

A. Primary HTN.
B. Secondary HTN.
C. Drug-induced HTN.
D. White coat syndrome.

PLAN

A. General interventions (Table 13.6):
1. Advise overweight clients to lose weight. Loss of as little as 10 lbs reduces BP in many clients. ▶

TABLE 13.6 WHELTON 2017 HIGH BLOOD PRESSURE CLINICAL PRACTICE GUIDELINE CLASSIFICATIONS

BP Classification	SBP (mmHg)	DBP (mmHg)
Normal	<120	*And* <80
Pre-HTN	120–129	>80
Stage 1 HTN	130–139	*Or* 80–89
Stage 2 HTN	≥140	*Or* ≥90

BP, blood pressure; DBP, diastolic blood pressure; HTN, hypertension; SBP, systolic blood pressure.

2. Advise the client to limit or discontinue alcohol intake.
3. Encourage the client to stop smoking.
4. Encourage increased physical activity. The 2017 ACC/AHA guidelines on lifestyle management outline the newest physical activity recommendation, which advises adults to engage in 40 minutes of aerobic physical activity three to four times a week. The aerobic exercise should involve moderate to vigorous intensity.
5. Encourage some form of relaxation technique.

B. Client teaching:
1. Stress asymptomatic nature of disease.
2. Stress importance of ongoing monitoring and treatment under the direction of a healthcare provider.
3. Review risk factors for cardiac, renal, and cerebrovascular disease and possible preventive measures.
4. The ACC maintains the CardioSmart Patient Education Portal for an online BP and other heart conditions management tool to educate and motivate clients. ACC's CardioSmart is a free resource located at www.cardiosmart.org. CardioSmartTXT *PREVENT* is a 6-month program of health tips and reminders sent via two text messages a week. CardioSmartTXT *QUIT* is a 2-month program to assist clients in smoking cessation. Four text messages are sent a day with information and assistance with smoking cessation.

C. Dietary management:
1. Review specific dietary measures. Give dietary recommendation sheets. See Appendix B for low-fat/low-cholesterol and Dietary Approaches to Stop Hypertension (DASH) diets.
2. Diet alone will only make the smallest incremental change in BP; therefore, it should be combined with lifestyle modification and lower sodium intake; stopping smoking, weight loss, and exercise are essential.
3. It is essential for the client/family to read labels for sodium, fat content, and serving sizes.
4. Other dietary changes include low-fat/low-cholesterol diets and limiting fats:
a. Use monounsaturated fats to decrease cholesterol:
i. Canola oil.
ii. Olive oil.
b. Use in limited quantities: Polyunsaturated fats decrease cholesterol but not as well as monounsaturated fats:
i. Vegetable and fish oils.
ii. Corn, safflower, peanut, and soybean oils.
c. Limit saturated fats:
i. Animal fats and some plant fats.
ii. Butter and lard.
iii. Coconut oil and palm oil.

D. Pharmaceutical therapy:
1. If lifestyle changes alone are not adequate to control HTN, consider drug therapy. Medication doses are dependent on age, ethnicity, and comorbid conditions. Most clients will require two or more medications to control their BP. Consider starting antihypertensives and/or diuretics (see Table 13.5 for drugs and classifications). The JNC 8 recommendations were released in 2014 for initiation and BP goals (Table 13.7). The JNC 8 published an extensive algorithm for treatment of HTN. The algorithm is available at jama.jamanetwork.com/solr/searchresults.aspx?q=jnc8&fd_JournalID=67&f_JournalDisplayName=JAMA&SearchSourceType=3.

TABLE 13.7 MODIFIABLE AND NONMODIFIABLE RISK FOR CONTROL OF HYPERTENSION

Modifiable	Nonmodifiable
Sedentary lifestyle	Age
Smoking	Gender
Diet	Ethnicity
Lipid control	Diabetes
Sodium intake	Postmenopausal
Alcohol intake	Family history
Obesity	

2. The 2017 guidelines released by the AHA, the ACC, and the Centers for Disease Control and Prevention (CDC) recommend medication classifications for treatment of HTN in the presence of medical conditions (Table 13.8).
3. Antihypertensive/diuretics should be started low and increased if there is inadequate response to initial therapy and nonadherence is ruled out. Consider the following:
a. Increasing drug dose.
b. Substituting another drug.
c. Adding a second drug from another class; a diuretic is recommended if one is not already being used.
d. **Beta-blockers are no longer first-line antihypertensive agents. Atenolol may increase central aortic pressure.**
e. **ACEIs and ARBs are critical medications to prescribe and titrate to maximum dose as a first-line medication in people with renal disease, diabetes, and proteinuria.**
4. If response is still inadequate, add a second or third drug or a diuretic if one has not already been tried.
5. Evaluate the client for secondary causes if severe HTN is resistant to therapy.
6. Resistant HTN; rule out all inadequate response to the three-drug therapy (ACEI or ARB or CCB + diuretic).
a. "White-coat" HTN: Have the client begin to take and record their BP at home and report the values.
b. Use of size-appropriate BP cuffs on obese clients.
c. Nonadherence to therapy, including side effects, medication regimen too complex, and/or cost/affordability.
d. Volume overload due to excessive salt intake, progressive renal damage, fluid retention from BP reduction, and inadequate diuretic therapy.
e. Drug problems, such as dose too low, wrong type of diuretic, inappropriate combinations, rapid inactivation, drug actions, and interactions.
f. Associated conditions, such as smoking, obesity, sleep apnea, insulin resistance, ethanol intake greater than 30 mL (1 oz) per day, panic attacks, chronic pain, and organic brain syndrome.
g. Adding spironolactone can decrease SBP by an average of 25 mmHg and DBP by an average of 12 mmHg in resistant hypertensive clients.
7. Treat with decongestants very cautiously. Pseudoephedrine HCl (Sudafed) has the least cardiovascular effect. ▶

TABLE 13.8 WHELTON 2017 HIGH BLOOD PRESSURE CLINICAL PRACTICE GUIDELINE

Ethnicity/Population	Age (Years)	Begin Initiation of Pharmacologic Treatment to Lower Blood Pressure	Blood Pressure Goals for Treatment	Other Comments
General population	>60	Initiate therapy for SBP ≥130 mmHg or DBP ≥80 mmHg.	Treat to goal of SBP <130 mmHg and DBP <80 mmHg.	If pharmacologic treatment results in a lower achieved SBP and treatment is well-tolerated, treatment does not need to be adjusted.
General population	<60	Initiate therapy to lower BP at SBP ≥130 mmHg.	Treat to goal of SBP <130 mmHg.	
General population	<60	Initiate therapy to lower BP at DBP ≥80 mmHg.	Treat to goal of DBP <80 mmHg.	
Clients with chronic kidney disease	>18	Initiate therapy to lower BP at SBP ≥130 mmHg or DBP ≥80 mmHg.	Treat to goal of SBP <130 mmHg and DBP <80 mmHg.	
Clients with chronic kidney disease (regardless of race or diabetes status)	>18			Initial or add-on antihypertensive therapy should include an ACEI or ARB to improve kidney outcomes.
Clients with diabetes	>18	Initiate therapy to lower BP at SBP ≥130 mmHg or DBP ≥80 mmHg.	Treat to goal of SBP <130 mmHg and DBP <80 mmHg.	The American Diabetes Association criteria differ from all else in that their guidelines remain treating diabetics with hypertension to an SBP goal of <140 mmHg and to a DBP of <80 mmHg.
General non-Black population including those with diabetes				Initial antihypertensive therapy should include a thiazide-type diuretic, CCB, ACEI, or ARB.
General Black population including those with diabetes				Initial antihypertensive therapy should include a thiazide-type diuretic or CCB.

ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BP, blood pressure; CCB, calcium channel blocker; DBP, diastolic blood pressure; SBP, systolic blood pressure.

8. Diuretics may worsen gout and diabetes.
9. Beta-blockers are contraindicated in asthma, HF, and heart block.
10. Use diltiazem HCl (Cardizem) and verapamil HCl (Calan) cautiously in HF or block.
11. ACEIs may cause coughing.
12. Abrupt cessation of therapy with a short-acting beta-blocker, such as propranolol, or the short-acting alpha-2-agonist clonidine can lead to a potentially fatal withdrawal syndrome. Gradual discontinuation of these agents over a period of weeks should prevent this syndrome.

FOLLOW-UP

A. If drug therapy is initiated, see the client again in 2 to 4 weeks for follow-up.
B. Once the client is stable, see them every 3 to 6 months.
C. Evaluate the client yearly, including uric acid, creatinine, and potassium.
D. Review and discuss drug therapy compliance, effectiveness, and adverse reactions (including effect on sexual activity) at each visit.
E. Home and ambulatory blood pressure monitoring (ABPM) is an adjunctive tool for management of HTN.
1. BP tracking apps are available on iTunes for the iPhone, iPod touch, and iPad.
2. BP tracking apps for Android are available on Google Play and Amazon Appstore.
3. A printable BP tracker log is located at organizedhome.com/sites/default/files/image/pdf/health_blood_pressure_tracker.pdf.
F. Consider sleep study for diagnosis of OSA (see the "Obstructive Sleep Apnea" section of this chapter).
G. Clients with elevated BP without diabetes, chronic kidney disease (CKD), or cardiovascular disease should be treated by nonpharmacologic therapy (e.g., diet, sodium reduction, weight loss, exercise, smoking cessation) and should be evaluated annually.

EMERGENT ISSUES/INSTRUCTIONS

A. Consult a physician if the client is having an acute hypertensive emergency: DBP greater than 130 mmHg.

CONSULTATION/REFERRAL

A. If the client is pregnant, consult a physician before prescribing medications. Many antihypertensive drugs are harmful to the fetus.

B. Consult/comanage with a physician if the client needs more than three drugs for therapy.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy. Refer to Chapter 16, "Obstetrics Guidelines."

1. HTN may be either chronic or pregnancy-induced.

2. HTN is considered chronic if it is present before pregnancy or diagnosed prior to the 20th week of gestation.

3. Pregnancy-induced hypertension (PIH) is diagnosed if SBP increases 30 mmHg or more, or if DBP increases 15 mmHg or more, compared with BP readings before the 20th week of gestation. When BP readings are not known, a reading of 140/90 or higher is considered abnormal.

4. Maternal as well as fetal mortality and morbidity improve with treatment.

B. Geriatrics:

1. The Whelton 2017 general guideline for clients older than 60 is to treat when BP is greater than 130/80. HTN in the elderly places the client at risk for coronary events, stroke, HF, and PAD.

2. The JNC-8 guideline on managing HTN in adults older than 60 is to treat BP to a goal less than 150/90:

a. Avoid SBP less than 130 mmHg and DBP less than 60 mmHg due to effects on cerebral perfusion.

3. Elderly persons with HTN are more likely to develop orthostatic and postprandial hypotension, which may result in falls or syncope.

a. Evaluate side effects including dizziness and sedation. Beta-blockers may cause depression or confusion in the elderly and are no longer recommended.

b. Check for orthostatic BP changes.

4. The general approach to drug therapy in the geriatric population is to start low and go slow. Specific drug choices should be determined by comorbidities, side effects, and costs:

a. Start at low doses, with slow titration up to goal.

5. Check the Beers list for harmful drugs in the geriatric population. A printable pocket card is available for download at www.americangeriatrics.org/files/documents/beers/PrintableBeersPocketCard.pdf.

6. To avoid hyperkalemia in the elderly, potassium-sparing diuretics should not be given with ACEI or ARBs.

7. Geriatric red flag:

a. If adding or increasing diuretics to manage BP in clients with existing cognitive, functional, or mobility impairment, the nurse practitioner (NP) should practice special consideration of the following geriatric syndromes:

i. Incontinence.

ii. Falls.

1) Avoid diuretics after 4 p.m. to reduce the risk of falls from attempting to get to the bathroom during the night. Discuss ways the client can adjust fluid intake and toileting habits. Alert the client that avoidance of incontinent episodes by reducing oral intake may result in dehydration and/or urinary tract infection.

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LYMPHEDEMA

DEFINITION

A. Peripheral vascular disease (PVD) is a general term that encompasses all occlusive or inflammatory diseases that occur within the peripheral arteries, veins, and lymphatic system. Lymphedema is a chronic condition caused by the accumulation of lymphatic fluid in the interstitial tissue.

INCIDENCE

A. Almost one million Americans have lymphedema, and the incidence worldwide is projected to approach 100 million.

PATHOGENESIS

A. Lymphedema occurs when lymph fluid is unable to flow in a normal manner and accumulates in an extremity. The propensity for lymphedema can be inherited or caused by another condition such as lymphangitis, malignancy, filariasis, removal of lymph nodes, and major trauma or burns.

PREDISPOSING FACTORS

A. Cancer.

B. Radiation therapy.

C. Surgical removal of lymph nodes.

D. Parasitic infection (filariasis).

E. Congenital disorder involving the structure of the lymph system:

1. Milroy disease.

2. Meige disease.

F. Obesity.

G. Surgical implantation of pacemaker or arteriovenous shunt.

H. Systemic diseases (hyperthyroidism, hypertension, renal or cardiac disease).

I. Vascular disease (chronic venous insufficiency [CVI] or postthrombotic syndrome).

J. Sedentary lifestyle.

COMMON COMPLAINTS

A. Severe edema that is consistent with the distal aspect of the extremity.

B. Hard skin over an edematous area.

C. Loss of range of motion.

POTENTIAL COMPLICATIONS

A. Infection, including lymphangitis and cellulitis.
B. Lymphangiosarcoma.

SUBJECTIVE DATA

A. Ask client when the symptom were first noticed.
B. Have client describe duration of symptoms.
C. Review any history of cancer, radiation, and chemotherapy.
D. Review recent history of invasive procedures or surgery.
E. Ask client to list all medications currently being taken, particularly substances not prescribed and illicit drugs such as cocaine.
F. Ask client to describe any pain.
G. Ask the client what makes the symptoms better and what makes them worse.
H. Have client rate discomfort on a scale of 0 to 10, with 0 being comfortable.

PHYSICAL EXAMINATION

A. Check vital signs.
  1. Check blood pressure, resting heart rate, respirations, temperature, height, weight, and body mass index.
B. Inspect:
  1. Assess for signs of erythema, increased temperature, and edema.
C. Palpate:
  1. Palpate lymph nodes distal and proximal to the site.
  2. Palpate pulses distal and proximal in all extremities.
  3. Palpate extremity for tenderness.
D. Auscultate:
  1. Heart: rate, rhythm, heart sounds, murmur, rub, and gallops.
  2. Lungs: lung sounds in all fields.

DIAGNOSTIC TESTS

A. Serum laboratory tests: complete blood count (CBC) with differential and brain natriuretic peptide (BNP).
B. CT scan of the affected extremity.
C. Doppler ultrasound of the affected extremity.
D. MRI.
E. Lymphoscintigraphy.
F. Additional tests to monitor coexisting systemic and/or vascular disease.

DIFFERENTIAL DIAGNOSES

A. Unilateral limb edema:
  1. Lymphedema.
  2. Venous insufficiency.
  3. Deep vein thrombosis or postthrombotic syndrome.
  4. Arthritis.
  5. Baker cyst.
  6. Presence or reoccurrence of cancer.
B. Symmetrical limb edema:
  1. Congestive heart failure (CHF).
  2. CVI.
  3. Renal dysfunction.
  4. Hepatic dysfunction.
  5. Hypothyroidism (myxedema).
  6. Medication-induced edema.
  7. Lipedema.

PLAN

A. Client teaching: *See Client Teaching Guide for this chapter, "Lymphedema."*
  1. Protect your arm or leg while recovering from cancer treatment.
  2. Avoid heavy lifting, if it is an arm.
  3. Avoid strenuous exercise.
  4. Avoid heat on your arm or leg.
  5. Avoid tight clothing.
  6. Inspect the affected limb daily. Note any cracks or cuts that increase the risk of infection and treat as directed.
  7. Apply lotion daily to protect and prevent dry skin.
  8. Avoid burns to the skin.
  9. Take extra care when shaving and use a clean razor on clean skin.
  10. Use insect repellent to prevent insect bites, which could become infected.
  11. Wash skin daily with a mild soap and dry skin carefully paying special attention to any skin folds.
  12. Prevent excessive sun exposure by applying sunscreen or wearing appropriate clothing to cover skin.
B. Nonpharmaceutical therapy: consists of two phases of complex decongestive therapy (CDT).
  1. Intensive reduction phase:
    a. Duration: usually past 2 to 4 weeks for severe lymphedema. If lymphedema is mild to moderate, it is possible to skip to the maintenance phase of the CDT.
    b. Steps of CDT in the intensive reduction phase:
      i. Low-stretch bandages (an adjustable compression garments).
      ii. Manual lymphatic drainage (MLD): MLD consists of gentle, skin-stretching movements performed by a certified therapist.
      iii. Sequential gradient pump: This is a durable medical equipment that provides gentle massage that mimics MLD.
      iv. Exercises: A physical therapy referral for a home exercise plan would be appropriate. A home exercise plan would likely include resistance bands or light weights and walking.
      v. Skincare and risk precautions: See the "Client Teaching" section earlier.
  2. Maintenance phase:
    a. Duration: lifelong, although the frequency of the treatments can be tailored to the symptoms.
    b. Steps of CDT in the maintenance phase:
      i. Compression garments.
      ii. Exercises in the form of a home exercise plan.
      iii. Skincare and risk precautions: See the "Client Teaching" section earlier.
      iv. MLD, if required.
      v. Sequential gradient pump, if required.
C. Surgery:
  1. Lymphaticovenous anastomosis (LVA): surgical attachment of nonfunctional lymph nodes to the venous system, thereby allowing for drainage of access fluid.
  2. Vascularized lymph node transfer (VLNT): transplantation of functional lymph nodes into an extremity to restore lymphatic function.
  3. Suction assisted protein lipectomy (SAPL): a treatment in which suction is used to remove lymphatic solids and fatty deposits. This surgery is most commonly used to treat those who are poor candidates for LVA and/or VLNT.

FOLLOW-UP

A. Follow-up is determined by client's needs, frequency and intensity of symptoms, and presence of other medical conditions. ▶

B. PVD manifesting persistent symptoms should always be followed by a cardiologist.

EMERGENT ISSUES/INSTRUCTIONS

A. Clients should contact the office if they are experiencing any new symptom(s) not present at their last office visit.

B. Clients should contact the office if they have any of the following symptoms:

1. Fever over 101°F.

2. Increased redness, pain, tenderness to touch, and/or warmth in the affected arm or leg.

3. New pain, swelling, or warmth in an arm or leg.

CONSULTATION/REFERRAL

A. If you suspect acute limb ischemia (ALI), refer the client for immediate hospitalization to obtain diagnostic testing to determine the presence of a thrombus and restore circulation to the affected extremity.

B. If chronic limb ischemia has led to ulceration and/or superimposed infection, hospitalization is indicated to initiate wound care consultation and diagnostic testing to determine the degree of arterial occlusion.

C. Referral to a cardiologist is indicated in the presence of persistent PVD symptoms.

D. Referral to a certified lymphedema therapist and physical therapist is indicated to best manage chronic lymphedema.

E. Referral to physical therapy is indicated to teach a home exercise program.

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MURMURS

DEFINITION

A. A murmur is turbulent blood flow through the heart as a result of one or more of the following etiologies:

1. Narrow valve opening, stenosis.

2. Incomplete valve closure, regurgitant or insufficient blood flow.

3. Abnormal opening through chambers, atrial or ventricular septal defect (VSD).

4. Rapid blood flow through normal valve structures; occurs during pregnancy, with increased physiologic demand states, such as thyrotoxicosis.

5. No abnormality; occurs in clients with thin chest walls.

INCIDENCE

A. Innocent heart murmurs are quite common. They affect 40% to 45% of children and about 10% of adults at some point during their lifetime. Innocent heart murmurs are more common in females during pregnancy. Abnormal heart murmurs occur most often in people who have certain heart conditions, such as a defective heart valve (e.g., aortic stenosis, mitral regurgitation).

PATHOGENESIS

A. Pathogenesis depends on specific etiology, but rheumatic heart disease, calcific changes, ischemic insults, congenital abnormalities, and degenerative diseases can all contribute to the development of a murmur.

COMMON COMPLAINTS

A. Often no symptoms are present and murmur is found on routine examination.

B. Complaints with advanced valvular disease:

1. Chest pain.

2. Dyspnea.

3. Palpitations.

4. Shortness of breath (SOB).

5. Exercise intolerance.

6. Postural light-headedness.

SUBJECTIVE DATA

A. Has the client ever been diagnosed with a murmur?

B. Did the client have frequent strep infections as a child?

C. Ask the client about any recent viral infections.

D. Question the client about chest pain, SOB, palpitations, diaphoresis, light-headedness, or syncope, especially with exertion.

E. Ask the client if any family members had sudden cardiac death before age 55.

PHYSICAL EXAMINATION

A. Check temperature, if indicated, pulse, respirations, and blood pressure (BP).

B. Inspect:

1. Inspect the chest for lifts and heaves.

C. Palpate:

1. Palpate the chest for lifts, heaves, and thrills.

E. Auscultate:

1. Auscultate heart for splitting of heart sounds, clicks, rubs, and murmurs; use bell and diaphragm of stethoscope to auscultate client in left lateral, supine, standing, sitting (and leaning forward), and squatting positions and after having them run in place or do jumping jacks for 2 to 3 minutes.

a. A new, systolic, regurgitant murmur in the setting of an acute myocardial infarction (MI) may indicate a ruptured papillary muscle and possible cardiogenic shock.

b. When a new murmur is audible, differentiate the location, timing, quality, intensity, and duration. Note if radiation to neck, axilla, or back is present.

c. Note location of murmur:

i. Aortic: second right intercostal space (ICS) next to the sternum.

ii. Pulmonic: second left ICS next to sternum.

iii. Tricuspid: fifth left ICS next to sternum.

iv. Mitral: fifth left ICS at midclavicular line.

d. If murmur is heard, have the client squat, stand, and/or perform the Valsalva maneuver. Squatting will increase the blood to the heart and increase the left ventricle blood volume and stroke volume, which will increase the sound of the murmur. Standing and the Valsalva maneuver will provide the opposite, in which the venous return will drop and decrease the ventricle size and stroke volume and soften the sound of the murmur.

e. If the sound of the murmur occurs in the opposite action, softer when squatting and louder when standing or during the Valsalva maneuver, consider hypertrophic cardiomyopathy or mitral valve prolapse (MVP) as the diagnosis.

2. Auscultate the neck and axilla for radiation.

DIAGNOSTIC TESTS

A. EKG.

B. Echocardiogram.

C. Chest radiography.

DIFFERENTIAL DIAGNOSES

A. Major differentiation should be in the description of the murmur, as this aids in identification of the murmur.

1. Timing:

a. Identify when the murmur occurs in the cardiac cycle.

b. Systolic murmurs may or may not be normal.

i. Occurs between the "S1" lub and the "S2" dub.

c. Diastolic murmurs are always abnormal.

i. Occurs between the "S2" dub and the "S1" lub.

2. Quality: Is the sound harsh, blowing, musical, rumbling, vibratory, or soft?

3. Intensity: Murmurs are usually graded on a 6-point scale:

a. Grade I: barely audible.

b. Grade II: audible but soft.

c. Grade III: easily audible without thrill.

d. Grade IV: easily audible, thrill usually palpable.

e. Grade V: audible with only the rim of the stethoscope on the chest wall.

f. Grade VI: audible with the stethoscope barely off the chest wall; thrill present.

4. Duration: Identify the location and timing in the specific phase of the cardiac cycle:

a. Holosystolic: throughout systole.

b. Holodiastolic: throughout diastole.

c. Midsystolic: midway between S1 and S3.

d. Middiastolic: midway between S2 and S1.

e. Decrescendo: starts loud at the beginning, then tapers off.

f. Crescendo: starts soft at the beginning, then gets louder.

5. Radiation: Murmur can be heard in another place, such as the neck, back, left axilla, or across precordium. Sound usually radiates in the direction of blood flow.

6. Location: Identify location on chest wall where murmur is heard the best. Identify site: apex, pulmonary area, tricuspid, and aortic areas. Radiation murmur may also include axilla, left fourth ICS, or base of heart.

7. Configuration: intensity of the murmur over time: Does it plateau, crescendo, decrescendo, or crescendo–decrescendo?

8. Systolic murmurs: Systolic murmurs are benign or pathologic:

a. Early systolic murmurs:

i. Mitral regurgitation: holosystolic, blowing may be loud; located at the fifth ICS and radiates to left axilla/back; heard best in the left lateral position and sudden squatting; intensity decreases with the Valsalva maneuver and standing.

ii. Tricuspid regurgitation: holosystolic, heard in left lower sternal border or apex when right ventricle is enlarged; intensity increases with inspiration and decreases with expiration; straight leg raises may increase intensity; may also see hepatojugular reflux (HJR).

iii. Physiologic: early to midsystolic, low-pitch normal S1 to S2, located at left lower sternal edge at third to fourth ICS; heard best with bell and in supine and disappears when sitting up or holding breath; commonly seen in pregnancy and infection.

b. Midsystolic to late systolic murmurs:

i. Aortic stenosis: loud, hard crescendo–decrescendo at second right ICS and radiates to neck; heard best leaning forward, increases with leg raise and lying flat; decreases with Valsalva and handgrip standing.

ii. Pulmonic stenosis: prolonged, loud S2 or crescendo–decrescendo, usually greater than 3/6 at second ICS and radiates to neck; increases with inspiration.

iii. Hypertrophic cardiomyopathy (aortic outflow obstruction): peaks at midsystole; loud, harsh tone at left, lower sternal border that may radiate to the neck; increases with Valsalva maneuver and standing, decreases with sudden squatting; note carotid upstroke brisk.

c. Late systolic murmurs:

i. MVP: midsystolic click heard before late systolic murmur, heard best at fifth left ICS; heard best with diaphragm; sitting or squatting may increase intensity.

ii. Tricuspid valve prolapse: heard over the left lower sternal border, delayed onset of murmur with inspiration secondary to an increase in the right ventricular volume.

9. Diastolic murmurs: Murmurs are always pathologic.

a. Early diastolic murmur:

i. Aortic regurgitation: high-pitch faint, decrescendo may start with S2 at third left ICS and radiates down sternal edge; heard best leaning forward, holding breath; increases with sudden squatting or handgrip; may hear displaced point maximal intensity, S3, bounding pulse.

ii. Pulmonary regurgitation: valvular, dilation of valve annulus, congenital defect (tetralogy of Fallot, VSD), and pulmonic stenosis; best heard over left second/third ICS; may sound high-pitched with "blowing" sound in clients with hypertension (HTN); may be pansystolic, having decrescendo configuration.

b. Middiastolic murmur:

i. Mitral stenosis: rumbling extends beyond middiastole at fifth ICS, heard best using the bell of the stethoscope; increases with left lateral position; may hear snap after S2.

ii. Tricuspid stenosis: increased flow across the tricuspid valve, heard best at the left sternal border; identified by its increase in intensity of the murmur with inspiration (Carvallo sign); commonly seen with mitral stenosis.

PLAN

A. General interventions:

1. Major therapeutic goals are to preserve quality of life, increase life expectancy and exercise capacity, and reduce risk of complications. ▶

2. Activity restriction is not necessary in clients with asymptomatic valvular disease.

B. Client teaching:

1. Reassure the client regarding specific diagnosis.

2. Counsel the client regarding their specific condition.

3. Teach the client signs and symptoms to report to the health provider, including chest pain, SOB, difficulty breathing, and so forth.

C. Medical and surgical management:

1. Clients who need progressive increases in medications to control symptoms may be candidates for valve replacement surgery.

D. Pharmaceutical therapy:

1. The American College of Cardiology/American Heart Association (ACC/AHA) 2014 guidelines do not recommend routine endocarditis antimicrobial prophylaxis treatment for common valvular lesions, including a bicuspid aortic valve, acquired aortic or mitral valve disease (including MVP with regurgitation), and hypertrophic cardiomyopathy with latent or resting obstruction.

2. Endocarditis prophylaxis treatment: cardiac conditions:

a. Prophylactic treatment is recommended for high-risk cardiac condition abnormalities. Specific cardiac conditions include the following:

i. Prosthetic cardiac valve or prosthetic material used for cardiac valve repair.

ii. Previous infective endocarditis.

iii. Certain congenital heart diseases, such as cyanotic congenital heart disease that has not been repaired; a congenital heart disease that has been repaired with an artificial material or device for 6 months after repair; and repaired congenital heart defects with continued problems such as leaks or insufficient flow at the prosthetic device or adjacent to the repair.

iv. Postcardiac transplant valvulopathy.

b. Procedures for high-risk clients previously mentioned who require prophylaxis treatment:

i. All dental procedures with manipulation of gingival tissue or periapical region of teeth or perforation of the oral mucosa.

ii. Incision or biopsy of respiratory mucosa or any invasive procedure of the respiratory tract system.

iii. Procedures that include infected skin or musculoskeletal tissue.

iv. Preventive treatment with antibiotics is not recommended for procedures that include the reproductive tract, urinary tract, or gastrointestinal (GI) tract.

c. Antibiotic prophylactic regimens include single dose 30 to 60 minutes prior to the procedure:

i. Amoxicillin 2 g PO, intramuscular (IM), or intravenous (IV) for adults.

ii. Ampicillin 2 g IM or IV or 50 mg/kg IM or IV.

iii. Allergy to penicillin: cephalexin 2 g PO for adults.

iv. Azithromycin or clarithromycin 500 g for adults.

v. If allergic to these, consider cefazolin or ceftriaxone 1 g IM or IV or clindamycin 600 mg IM or IV for adults.

d. Other pharmaceutical treatments depend on the specific valvular abnormality.

i. Mitral stenosis: The mitral valve has a narrowing that does not allow adequate blood to the left ventricle during diastole, usually due to rheumatic heart disease. Mitral heart disease is the most commonly seen valve effect with rheumatic heart disease.

ii. Diuretics such as furosemide (Lasix) or hydrochlorothiazide (HydroDiuril) are used to control edema.

iii. Digoxin (Lanoxin) or beta-blockers are used to control atrial fibrillation (AF) and irregular heart rate.

iv. Warfarin (Coumadin) and the antiplatelet agent aspirin (Bayer) are used to prevent clotting.

v. MVP: Echocardiogram is the recommended test for diagnosis of MVP. Usually no medications are recommended except when symptomatic and required.

1) Beta-blockers (e.g., atenolol) may be used for palpitations.

2) Diuretics should be avoided in clients who are volume-reserved.

3) Oral contraceptives should be avoided in females who exhibit neurologic symptoms.

vi. Mitral regurgitation: diuretics, digitalis, and afterload-reducing agents for congestive heart failure (CHF):

1) Aortic stenosis.

2) Diuretics are used for CHF.

3) Avoid vasodilators; they may result in profound, irreversible hypotension.

4) Echocardiograms should be performed every 6 to 12 months to follow progression of narrowing of the left ventricle across the aortic valve.

vii. Aortic regurgitation: Afterload-reducing agents, digitalis, and diuretics are recommended.

FOLLOW-UP

A. Most clients with valvular disease should be evaluated at least once a year.

B. Clients on oral anticoagulation drugs need monthly follow-up or as needed prothrombin time (PT)/international normalized ratio (INR).

CONSULTATION/REFERRAL

A. Consult a physician if the client is diagnosed with a new murmur or exercise-induced symptoms during a sports physical.

B. Refer clients with newly diagnosed murmurs to a cardiologist after obtaining echocardiogram results.

C. Drug therapy should be initiated according to diagnosis and symptoms.

D. Onset of AF with rapid ventricular response is an indication for immediate hospitalization.

E. Refer clients with systemic embolization to a physician for emergent anticoagulation therapy and chronic oral anticoagulant therapy. Discuss the possibility of valve replacement with a cardiologist.

F. If a new murmur is diagnosed in a pregnant them with a history of cardiac disease, refer them to a physician immediately.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
  1. The development of a new, "high-flow" murmur in a healthy client is not uncommon due to physiologic changes occurring during pregnancy.

B. Geriatrics:
  1. Aortic stenosis, which is a systolic murmur, occurs in about 2% of people older than the age of 65. It occurs more often in males than in females.
  2. Symptoms of aortic stenosis in the elderly may include the following:
    a. Chest discomfort: The chest pain may get worse with activity and reach into the arm, neck, or jaw. The chest may also feel tight or squeezed.
    b. Cough, possibly bloody.
    c. Breathing problems when exercising.
    d. Becoming easily tired.
    e. Feeling the heart beat (palpitations).
    f. Fainting, weakness, or dizziness with activity.
  3. A systolic murmur, heard best in the aortic area, may indicate aortic sclerosis due to aging of the aortic valve rather than true aortic stenosis.

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PALPITATIONS

DEFINITION

A. Palpitations are a feeling or an unpleasant awareness of the heartbeat in the chest. It may be described as feeling a sensation of the heart "flip-flopping" or feeling a "rapid flutter" of the heart.

INCIDENCE

A. The incidence of palpitations may range from 1% to 8% of clients in a general practice setting.

PATHOGENESIS

A. Palpitations may be caused by the following:
  1. Increase in stroke volume or contractility.
  2. Sudden change in heart rate or rhythm.
  3. Unusual cardiac movement within thorax.
  4. Hyperkinetic states, which cause constant pounding.
  5. Valvular heart disease that produces large stroke volumes.
  6. Catecholamine release during anxiety or panic attacks.

PREDISPOSING FACTORS

A. Cardiac defects.
B. Severe anemia.
C. Hyperthyroidism.
D. Pregnancy.
E. Fever.
F. Anxiety.
G. Stimulants, such as caffeine and certain drugs.
H. Emotions, such as fear.
I. Exertion.
J. Diabetes mellitus and insulin reaction.

COMMON COMPLAINTS

A. Palpitations are often described as a turning over or flopping sensation in the chest, but symptoms vary enormously.
B. Most clients are free of palpitations at the time of the exam.

OTHER SIGNS AND SYMPTOMS

A. Fluttering in the chest.
B. Shortness of breath (SOB).
C. Pounding in the chest and neck.
D. Diaphoresis.
E. Light-headedness.
F. Anxiety or fear.

SUBJECTIVE DATA

A. Ask the client when symptoms first presented, including age, and how they have changed.
B. Have the client describe the characteristics of the palpitations, such as rapid, regular, irregular, or slow.
C. Ask the client what precipitates the palpitations. Does anything terminate them, or do they go away on their own?
D. Inquire whether symptoms occur or change with position (standing, bending over, lying down, left lateral decubitus position) and/or exercise.
E. Ask the client about other symptoms associated with the palpitations, such as dizziness or syncope.
F. Ask how often the episodes occur and how long each lasts.
G. Discuss any previous treatments for this condition and the results.
H. Ask the client about risk factors for coronary heart disease (CHD) and cardiac history.
I. Obtain a complete list of medications the client is currently taking, including over-the-counter (OTC) and herbal products:
  1. Specifically question the client's use of OTC decongestants and diet pills.
  2. Are there any new medications or changes in routine medications?

PHYSICAL EXAMINATION

A. Check pulse (count the pulse for 1 full minute), respirations, and blood pressure (BP).
B. Inspect:
  1. Inspect overall appearance.
  2. Inspect the skin for diaphoresis and pallor.
  3. Inspect the neck for thyromegaly or jugular vein distension.
  4. Inspect the legs for edema.
C. Palpate:
  1. Palpate the skin for temperature and dryness.
  2. Palpate the lower extremities for edema and calf tenderness.
  3. Palpate the neck for thyroid enlargement.
D. Auscultate:
  1. Auscultate the heart for abnormal rhythms. Auscultate heart sitting, standing, and left lateral decubitus position. Ask client to walk quickly down the hallway and back and then auscultate heart in all positions again.
  2. Auscultate the lungs.
  3. Auscultate the neck and carotid arteries for bruits.
E. Mental status:

1. Does the client appear light-headed, anxious, or fearful?

DIAGNOSTIC TESTS

A. Diagnostic testing is highly recommended for clients with an arrhythmia or at risk for an arrhythmia, and clients who are anxious and want to explore the causes of their symptoms. The following testing is recommended:

1. Hemoglobin to rule out anemia, if suggestive on exam.

2. Thyroid-stimulating hormone (TSH) to rule out hyperthyroidism, if suggestive on exam.

3. EKG during episode, if possible.

4. Ambulatory monitoring if symptoms continue, either 24-hour Holter monitor or client-activated transtelephonic monitoring.

5. Treadmill test if palpitations are provoked by exercise.

DIFFERENTIAL DIAGNOSES

A. Palpitations are secondary to an underlying problem, such as anxiety, medications, or cardiac or pulmonary origin.

PLAN

A. General interventions:

1. Provide reassurance if the palpitations result from a neurotic concern.

B. Client teaching:

1. Caution the client to avoid any factors that trigger episodes. Factors may include stress, exercise, foods, and medications.

2. Teach the client the vagal maneuver, which is effective in halting palpitations.

C. Medical and surgical management:

1. Correct any underlying problem (e.g., cardiac or pulmonary).

2. Treat medical conditions accordingly.

3. Management of arrhythmias should be monitored by a cardiologist.

D. Pharmaceutical therapy:

1. Discontinue all nonessential medications that could cause palpitations.

FOLLOW-UP

A. Depending on the etiology of palpitations and the existence of comorbid conditions, the prognosis in clients with no underlying cardiac disease is generally favorable.

CONSULTATION/REFERRAL

A. Consult a physician if the client has a history of palpitations leading to syncope or near syncope, angina-like chest pain, or dyspnea. These clients are candidates for referral to a cardiologist and/or inpatient evaluation. Refer any client with an arrhythmia to a cardiologist.

B. Hemodynamically compromised clients need prompt hospital admission.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Elderly individuals are at increased risk for adverse effects from antiarrhythmic medications.

2. If drug treatment is necessary, lower doses should be initiated and titrated slowly to desired effect.

3. Polypharmacy (variously defined as the use of more than five to nine medications) may be the cause of cardiac-related symptoms.

4. Use caution when prescribing aspirin therapy in older adults.

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PERIPHERAL ARTERIAL DISEASE

DEFINITION

A. Peripheral arterial disease (PAD) is a circulatory disorder characterized by the buildup of plaque on the interior surface of arteries. These plaques harden and narrow the diameter of the arteries, which reduces the volume of blood circulating to internal organs and extremities. The arteries affected by PAD include all arteries in the body, with the exception of the cerebral and coronary arteries. The decreased circulation seen in PAD can also be caused by nonatherosclerotic conditions. Some of these conditions are arteritis, trauma, radiation damage, and fibromuscular dysplasia. Symptoms of PAD can occur in the upper or lower extremities.

B. Classification of PAD:

1. Asymptomatic PAD:

a. No symptoms but the presence of risk factors or a new diagnosis of a common coexisting disease (coronary artery disease [CAD] or cerebrovascular disease) should prompt further evaluation.

2. Intermittent claudication (IC):

a. Discomfort (e.g., cramping or pain) with physical exertion that remits within 10 minutes of rest.

3. Chronic limb ischemia (CLI):

a. Pain at rest with a nonhealing wound, skin ulceration, or gangrene.

b. Onset is defined as chronic (i.e., ≥2 weeks).

4. Acute limb ischemia (ALI):

a. Pain at rest in a pale, pulseless extremity that is usually cold (poikilothermia) and has paresthesias and/or paralysis.

b. Onset is defined as acute (i.e., <2 weeks).

C. Other conditions contained within PAD:

1. Buerger disease (thromboangiitis obliterans): a disease manifested by inflammation, peripheral edema, and microthrombi, leading to gangrene of the hands and feet; usually caused by tobacco abuse and clients are thought to have a genetic predisposition to develop this condition.

2. Raynaud syndrome: a vasospastic disorder manifested by a response in the extremities to cold temperatures or stress where pallor, cyanosis, numbness, and/or pain are experienced.

3. Leriche syndrome: the triad of claudication, absent or diminished femoral pulses, and erectile dysfunction.

INCIDENCE

A. Approximately 8 million adults in the United States have PAD. This correlates to 20% of Americans older than 80 years of age.

B. PAD is more common in males than in females.

C. PAD is more common in clients of African and Hispanic descent when compared with the non-Hispanic White population.

▶

PATHOGENESIS

A. PAD is most commonly precipitated by atherosclerosis. An atherosclerotic plaque develops in response to turbulent blood flow on the endothelial cells of the vessel wall. The plaque contains inflammatory cells and a thrombogenic lipid core that is covered by a fibrous cap. When the fibrous cap is disturbed, the lipid core can precipitate to development of a thrombus and lead to occlusion of the vessel.

PREDISPOSING FACTORS

A. Smoking.
B. Diabetes.
C. Dyslipidemia.
D. Hypertension (HTN).
E. Obesity.
F. Atherosclerotic disease in another vascular bed (e.g., coronary, carotid, subclavian, renal, mesenteric artery stenosis, or abdominal aortic aneurysm).
G. Age.
  1. Age ≥65 years, increased occurrence.
  2. Age 50 to 64 years, with family history of PAD or risk factors for atherosclerosis (e.g., diabetes, smoking history, hyperlipidemia, and/or HTN).
  3. Age <50 years, with diabetes and one additional risk factor for atherosclerosis (e.g., coronary, carotid, subclavian, renal, mesenteric artery stenosis, and abdominal aortic aneurysm).

COMMON COMPLAINTS

A. Pain with activity is commonly characterized as cramping and/or aching:
  1. Upper extremity pain in the forearm, hand, and digits.
  2. Lower extremity pain in the foot, calf, hip, thigh, and/or buttocks:
    a. Foot pain is most common in tibial or peroneal artery stenosis.
    b. Calf pain is most common with superficial femoral or popliteal artery stenosis.
    c. Thigh pain is most common in aortoiliac and common femoral artery stenosis.
    d. Hip and buttock pain are most common with aortoiliac arterial stenosis.
B. Pain at rest.
C. Calf weakness or fatigue.
D. Numbness or tingling.
E. Dizziness with upper extremity exertion.
F. Syncope with upper extremity exertion.
G. Extremity ulceration.

OTHER SIGNS AND SYMPTOMS

A. Decreased peripheral pulses.
B. Blanching of the affected limb with elevation.
C. Ulcerations or infection on distal aspects of extremities.
D. Erectile dysfunction.

POTENTIAL COMPLICATIONS

A. Nonhealing lower extremity ulcerations.
B. Infection.
C. Amputation.
D. Common coexisting diseases:
  1. CAD; also known as coronary heart disease (CHD).
  2. Cerebrovascular disease.
  3. Subclavian artery stenosis.

SUBJECTIVE DATA

A. Ask client what activity brought about or preceded the episode or whether it occurs at rest. If ambulation was the precipitating factor, how far was the client able to walk?
B. Have client describe duration of pain and what time of day the symptoms began.
C. Ask client what alleviates their pain.
D. Ask client whether any previous episodes have occurred.
E. Ask the client to list all medications, including over-the-counter (OTC) and herbal products currently being taken or recently stopped.
F. Ask client to quantify their smoking history.
G. Ask client if they have medical history of myocardial infarction (MI) or cerebrovascular accident (CVA).
H. Ask client if they have any history of impotence or erectile dysfunction.

PHYSICAL EXAMINATION

A. Clients presenting with ALI should be quickly assessed for the need to call emergency services/911 for immediate transport to the hospital.
  1. Symptoms of ALI as evidenced by the six Ps: pain, pallor, paresthesia, paralysis, pulseness, and poikilothermia (the inability to maintain a constant core temperature).
B. Check vital signs.
  1. Check blood pressure (BP) in both upper extremities.
    a. A difference in systolic blood pressure (SBP) of 10 mmHg or greater in upper extremities is associated with upper extremity PAD and cerebrovascular disease.
    b. A difference in SBP of 15 mmHg or greater in upper extremities is associated with lower extremity PAD.
  2. Check BP in both lower extremities.
  3. Document resting heart rate, respirations, height, and weight.
C. Inspect:
  1. Perform a funduscopic exam: Check for retinal vascular changes that indicate a retinal vascular occlusion, such as macular edema and neovascularization.
    a. Macular edema is the swelling of the central part of the retina.
    b. Neovascularization is the growth of abnormal vessels secondary to decreased perfusion of the retina.
  2. Inspect abdomen for a pulsating abdominal mass.
  3. Inspect extremities. Note edema, pallor, and cyanosis. Note color of extremities in dependent and elevated positions (e.g., elevation pallor and dependent rubor).
  4. Inspect distal skin, hair, and nails. Note any temperature discrepancies or trophic changes that are indicative of ischemia.
  5. Assess lower extremities for any ulcerations or diffuse erythema.
  6. Assess for Homans sign (i.e., calf pain with forced dorsiflexion).
  7. Assess whether pain occurs when affected limb is elevated.
D. Palpate:
  1. Palpate pulses, noting symmetry:
    a. Bilateral upper extremities (brachial and radial).
    b. Abdominal (aorta).
    c. Bilateral groin (femoral).
    d. Bilateral lower extremity pulses (popliteal, dorsalis pedis, and posterior tibialis).
  2. Palpate capillary refill.

▶

3. Perform an Allen test: Occlude the radial and ulnar arteries with the fist closed. Open the hand and then release one of the occluded arteries. Repeat but release the other artery. Each time, prompt capillary refill should occur.

4. Palpate neck for carotid bruits.

5. Palpate the abdominal aorta, noting any lateral pulsation, which is indicative of an aortic aneurysm.

E. Auscultate:

1. Auscultate heart: Assess rate, rhythm, heart sounds, murmur, and gallops.

2. Auscultate the carotids, abdomen, and bilateral groin for bruits.

3. Auscultate lungs: Assess lung sounds, noting any sign of heart failure (HF).

DIAGNOSTIC TESTS

A. Doppler ankle–brachial index (ABI).

1. Interpretation of ABI ratios:

a. 1.00 to 1.29: normal.

b. 0.91 to 0.99: borderline PAD.

c. 0.41 to 0.90: mild to moderate PAD.

d. 0.00 to 0.40: severe PAD.

B. Basic metabolic panel (BMP; including blood urea nitrogen [BUN], creatinine, sodium, and potassium).

C. Lipid profile.

D. C-reactive protein (CRP), homocysteine, and D-dimer.

E. EKG (12-lead).

F. Doppler ultrasound.

G. Abdominal ultrasound.

H. Treadmill testing.

I. CT angiography (CTA).

J. Magnetic resonance angiogram (MRA).

K. Arteriography, ordered and performed by a surgeon.

DIFFERENTIAL DIAGNOSES

A. PAD.

B. Venous stasis.

C. Venous obstruction/claudication.

D. Spinal stenosis.

E. Nerve root compression.

F. Arthritis of the hip.

G. Peripheral neuropathy.

H. Arteritis.

PLAN

A. General interventions.

1. The goal of therapy is to improve the client's quality of life by reducing morbidity and prolonging survival.

B. Client teaching: *See Client Teaching Guide for this chapter, "Peripheral Arterial Disease."*

1. Encourage smoking cessation, weight loss, and exercise, if applicable.

2. Encourage strategies to better manage other chronic medical conditions that directly affect the progression of PAD, that is, diabetes, dyslipidemia, obesity, and HTN.

3. Proper foot care:

a. Instruct client to wear proper-fitting shoes that protect the feet.

b. Inspect inside of shoes before donning.

c. Encourage client to inspect feet daily for signs of trauma or infection.

d. Instruct client to dry their feet well, including between toes, after bathing.

4. Prevention:

a. Control other chronic medical conditions, that is, diabetes, dyslipidemia, HTN, and obesity.

C. Dietary management:

1. To manage dyslipidemia and HTN, counsel the client on nutrition and low-fat, low-cholesterol, and low-sodium diet.

2. To manage diabetes, counsel the client on diabetic diet and carbohydrate counting.

3. To manage infection related to PAD, counsel the client on high-calorie, high-protein diet. Consider the addition of vitamins and minerals to promote wound healing, specifically zinc and vitamins C and A.

4. Give diet handouts and/or refer to a registered dietitian.

D. Pharmaceutical therapy:

1. Goal of therapy: prevention of thromboembolism.

a. Pentoxifylline (Trental):

i. 400 mg tablet.

1) Dosage indications based on creatinine clearance (CrCl):

a) CrCl less than 10 mL/min: 400 mg, taken once a day.

b) CrCl of 10 to 50 mL/min: 400 mg, taken BID.

c) CrCl greater than 50 mg/min: 400 mg, taken TID.

b. Cilostazol (Pletal):

i. 50 mg and 100 mg tablets.

1) **Warning: Metabolites of Pletal are inhibitors of phosphodiesterase III and are contraindicated in clients with congestive heart failure (CHF) of any severity.**

2) Dosage indications:

a) 50 mg, taken BID if taken in coadministration with ketoconazole, itraconazole, erythromycin, and diltiazem.

b) 100 mg, taken BID at least half an hour before or 2 hours after breakfast and dinner.

ii. 81 and 325 mg tablets, taken once daily.

c. Clopidogrel bisulfate (Plavix):

i. 75 mg, taken once daily.

d. Aspirin (acetylsalicylic acid; Ecotrin).

i. Beers criteria caution:

1) Use aspirin with caution in older adults older than 80 years with no history of cardiovascular disease (CVD): There is no clear evidence that aspirin provides primary prevention for cardiac events, but there is a risk for gastrointestinal (GI) bleeds.

2) In adults older than 65 years, avoid use of greater than 325 mg due to risk of GI bleed, unless other alternatives are not effective and the client can take a gastroprotective agent.

3) In adults older than 75 years, or adults older than 65 taking corticosteroids, anticoagulants, or antiplatelet agents, there is an increased risk of upper GI bleeds. The use of proton pump inhibitors (PPIs) reduces but does not eliminate risk. The risk of GI bleed increases with duration of use of nonsteroidal anti-inflammatory drugs (NSAIDs), including high-dose aspirin (ASA). At 1 year, the risk is 2% to 4% and this increases with each subsequent year.

2. Risk factor reduction:
   a. Manage dyslipidemia.
      i. Low-density lipoprotein cholesterol (LDL-C) goal: less than 100 mg/dL and less than 70 mg/dL for clients at high risk for CAD.
   b. Manage HTN.
      i. BP goal in clients without diabetes: less than 140/90 mmHg.
      ii. BP goal in clients with diabetes or chronic kidney disease: less than 130/80 mmHg.
   c. Manage diabetes.
      i. Hemoglobin A1C goal: less than 7.0%.

E. Surgical therapies:
1. Considered in clients with pain at rest, tissue loss, or significant physical limitations that prevent exercise.
   a. Bypass.
   b. Stenting.
   c. Angioplasty/percutaneous transluminal angioplasty.

F. Nonsurgical therapies:
1. Smoking cessation program.
2. Daily walking program.
   a. Instruct the client to walk to the point of pain, then stop and resume walking when pain remits.
   b. Medical clearance may need to be obtained for the client to exercise.

FOLLOW-UP

A. PAD manifesting persistent symptoms should always be followed by a cardiologist.

B. Follow-up is determined by the client's needs, frequency and intensity of symptoms, and the presence of other medical conditions.

EMERGENT ISSUES/INSTRUCTIONS

A. Clients should contact the office if they have any of the following symptoms:
1. **Worsening pain in arm or leg.**
2. **Fever over 101°F.**
3. **Any change in feeling of a hand or foot.**
4. **Any change in the color of a hand or foot.**
5. **Difficulty walking.**
6. **Vomiting or other illness that causes them to miss more than one dose of their medications.**
7. **If taking an antiplatelet therapy and have any of the following symptoms:**
   a. **Vomit that is bright red or dark and looks like coffee grounds or grape jelly.**
   b. **Blood in their bowel movements that looks black or tarry in color.**

B. Clients should contact the office if they are experiencing any new symptom(s) not present at their last office visit.

CONSULTATION/REFERRAL

A. If you suspect ALI, refer client for immediate hospitalization to obtain diagnostic testing to determine the presence of a thrombus and restore circulation to the affected extremity.

B. If CLI has led to ulceration and/or superimposed infection, hospitalization is indicated to initiate wound care consultation and diagnostic testing to determine the degree of arterial occlusion.

C. Refer to a cardiologist in the presence of persistent PAD symptoms.

D. Refer to a vascular surgeon for further evaluation of angioplasty, stenting, or bypass surgery.

E. Refer to a podiatrist to trim toenails and assess client for proper-fitting shoes.

F. Refer to pain management if pain is resistant to treatment.

G. Refer to a registered dietitian as indicated by the client's understanding of dietary modification necessary to improve status of risk factors.

INDIVIDUAL CONSIDERATIONS

A. Nonambulatory clients:
1. Using rocking chairs is a possible substitute for persons unable to participate in a walking program.

B. Geriatrics:
1. Refrain from aggressive therapies such as percutaneous or surgical revascularization in stenosis unless claudication or critical limb ischemia is present.
2. Review comorbid conditions, such as renal function, GI bleed risk, and polypharmacy prior to prescribing pharmaceutical therapy. The risk/benefit ratio is varied in older and frail adults and nonpharmacologic therapy may be the best choice depending on individual considerations (see the "Deprescribing" section of Chapter 3).
3. Be alert to signs and symptoms of depression related to immobility and pain.

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SUPERFICIAL THROMBOPHLEBITIS

DEFINITION

A. Superficial thrombophlebitis occurs when venous turbulence, static, or coagulability allows microthrombi to form in the presence of vessel wall (endothelial) injury, which triggers an inflammatory response in a superficial vein.
1. Most superficial thrombophlebitis occurs in the lower extremity but may also occur in the breast and penis (Mondor disease).
2. Superficial thrombophlebitis may also occur in the upper extremities and neck secondary to invasive ▶

intravenous (IV) catheters used in medical procedures and medication administration.

3. Generally, superficial thrombophlebitis is usually benign and self-limiting, but may persist for a period of time (3–4 weeks or longer) before resolution.

B. Superficial thrombophlebitis with an infection is referred to as a "septic thrombophlebitis."

INCIDENCE

A. With pregnancy, there is approximately a four- to fivefold increase of phlebitis over a nonpregnant female. Eighty percent of thromboembolic events in pregnancy are venous (0.5–2.0 per 1,000).

B. The prevalence of superficial thrombophlebitis ranges from 4% to 7% in clients with an indwelling catheter.

C. Superficial phlebitis is common after a vein radiofrequency or laser ablation.

PATHOGENESIS

A. Superficial thrombosis is caused by infection, abuse of IV drugs, chemical irritation from overuse of IV route for diagnostic tests and drugs, and/or trauma. Several episodes of superficial thrombosis can signal an underlying problem, such as carcinoma of the pancreas and hemoglobinopathies.

B. Thrombi in the upper extremities commonly have iatrogenic causes, such as IV catheters.

C. Superficial thrombophlebitis during pregnancy through the first 6 weeks postpartum is linked to a reduced fibrinolytic state.

PREDISPOSING FACTORS

A. Previous thrombophlebitis is the highest risk factor for recurrence.

B. Hypercoagulability, such as pregnancy (50% of events) through 6 weeks postpartum (50% of events).

C. History of polyarteritis nodosa, thromboangiitis obliterans (Buerger disease), or Behçet disease.

D. Hemoglobinopathies:

1. Factor V Leiden mutation.
2. Protein C deficiency.
3. Protein S deficiency.
4. Prothrombin gene mutation.
5. Antithrombin III deficiency.
6. Factor XII deficiency.

E. Estrogen therapy, especially high-dose hormone replacement therapy.

F. Malignancy (especially in the tail of the pancreas).

G. Systemic lupus erythematosus with a positive anticardiolipin antibody.

H. Sepsis.

I. Recent surgery.

J. Long bone trauma.

K. Recent IV catheter access.

L. Prolonged immobilization.

M. Obesity, specifically with a body mass index exceeding 25 kg/m^2.

N. Varicose veins or venous stasis.

O. Age older than 60.

P. Stroke.

Q. Myocardial infarction (MI).

R. Family history of deep vein thrombosis (DVT).

S. Smoking.

T. Hypertension (HTN).

U. Infection.

V. IV drug abuse.

COMMON COMPLAINTS

A. Warm, tender, inflamed vessel with palpable cord.

B. Discoloration: redness along the course of the superficial vein in the acute phase with progression to a brown pigmentation in the chronic phase.

C. Tenderness or pain localized to the affected vein.

OTHER SIGNS AND SYMPTOMS

A. Fever/no fever.

B. Localized edema.

POTENTIAL COMPLICATIONS

A. Superficial thrombophlebitis extending into the deep venous system.

B. DVT.

C. Conversion to suppurative thrombophlebitis:

1. Metastatic abscess formation.
2. Septicemia.
3. Septic emboli.

SUBJECTIVE DATA

A. Query the client regarding onset, duration, and intensity of symptoms.

B. Ask the client about fever or other related symptoms.

C. Obtain a thorough medical history and account of recent physical activity.

D. Ask the client about any recent experience of any type of injury.

E. Inquire whether the client has ever had similar symptoms or previous thrombophlebitis. If so, discuss previous treatment and therapy used and the results.

F. Review current medications: prescription, over-the-counter (OTC), and herbal products.

1. **Ask specifically about oral contraceptives and hormone therapy.**

G. Review the client's occupation for sedentary lifestyle.

H. Review any recent plane travel.

I. Review history for recent invasive procedures.

PHYSICAL EXAMINATION

A. Check temperature (if indicated with inflammation), pulse, respirations, and blood pressure (BP).

B. Inspect:

1. Assess overall appearance. Evaluate for the presence of respiratory distress.
2. Inspect extremities, noting erythema and edema.
3. Assess for increased warmth over the affected vein.

C. Auscultate:

1. Auscultate heart, noting rate, rhythm, heart sounds, murmurs, and gallops.
2. Auscultate lungs for lung sounds in all fields.

D. Palpate:

1. Palpate extremities; check all pulses, including femoral, posttibial, pedal, and radial.
2. Palpate extremities for tenderness and palpable cord.
3. Palpate lymph nodes distal and proximal to the site.
4. Test for Homans sign in lower extremities bilaterally if DVT is suspected.

DIAGNOSTIC TESTS

A. Duplex ultrasound identifies the presence, location, and extent of the venous thrombosis.

B. Doppler ultrasound. ▶

C. Magnetice resonance venography (MRV), which is noninvasive and sensitive for diagnosis but may not be readily available.

D. Laboratory tests are ordered dependent on the clinical situation.

1. Complete blood count (CBC) with differential.

2. Screening for hypercoagulability should not be considered for one episode of superficial thrombophlebitis.

3. Screening for hypercoagulability should be considered for recurrent superficial thrombophlebitis.

4. Blood cultures.

DIFFERENTIAL DIAGNOSES

A. Thrombophlebitis.
B. Varicose veins.
C. Cellulitis.
D. Strained muscle.
E. Insect bites.
F. Erythema nodosum.
G. Cutaneous polyarteritis nodosa.
H. Kaposi sarcoma.
I. Hyperalgesic pseudothrombophlebitis.

PLAN

A. General interventions:

1. The goal is to offer comfort, time for healing, and prevent an evolution into the deep venous system.

a. Prescribe supportive hose/compression stockings.
b. Have the client apply heat and elevate the extremity for varicose veins or superficial thrombophlebitis.
c. Prescribe bedrest for superficial thrombophlebitis.
d. DVT: Hospitalization is required.
e. Instruct clients with thrombophlebitis to discontinue oral contraceptives and hormone replacement.
f. Alternative forms of birth control recommended by the American Congress of Obstetricians and Gynecologists (ACOG) include the following:

i. Intrauterine device (IUD), including IUDs that contain progestin.
ii. Progestin-only oral contraceptives.
iii. Progestin-only implants.
iv. Barrier methods.
v. Surgical procedures: vasectomy and tubal ligation.

B. Client teaching: *See Client Teaching Guides for this chapter, "Superficial Thrombophlebitis" and "Varicose Veins."*

1. Advise all clients to stop smoking.

2. Instruct the client to avoid prolonged sitting or standing and not to cross or massage their legs.

3. Advise the client to avoid constrictive clothing, such as knee-high hosiery.

C. Pharmaceutical therapy:

1. Nonsteroidal anti-inflammatory drugs (NSAIDs) are used for treatment of pain. No NSAID has been identified as superior for treatment.

2. The use of anticoagulation therapy for treatment of lower extremity superficial thrombophlebitis is controversial. Unfractionated heparin and low-molecular-weight heparin (LMWH) are both used for treatment to reduce risk of DVT and/or recurrent phlebitis.

3. The American College of Chest Physicians recommends anticoagulation for clients with lower extremity superficial thrombophlebitis at increased risk of thromboembolism. This is defined as an affected venous segment greater than or equal to 5 cm in proximity (≥5 cm) to the deep venous system and positive medical risk factors. The American College of Chest Physicians' full evidence-based clinical practice guidelines on antithrombotic therapy are available at journal.chestnet.org/article/S0012-3692(15)00335-9/pdf.

4. Antibiotics, if infection is suspected.

D. Surgery:

1. Biopsy.

2. Vein ablation, only if symptoms are significant and persistent.

3. Vein ligation, only if symptoms are significant and persistent.

FOLLOW-UP

A. Schedule an appointment for clients with superficial thrombophlebitis to return in 7 to 10 days or earlier as needed. Repeat physical examination as needed to evaluate resolution or progression of the thrombophlebitis.

B. Periodic follow-up is needed to monitor clients on anticoagulation therapy.

C. After acute problem is resolved, consider laboratory evaluation for hypercoagulation syndrome (e.g., protein C, protein S, and antithrombin III).

D. Monitor bone loss with a dual-energy x-ray absorptiometry (DEXA) scan with prolonged use of heparin.

E. Screening all females for thrombophilias before starting oral contraceptives is not recommended by the ACOG.

F. Females with a history of thrombosis who have not had a complete evaluation should be tested for both antiphospholipid antibodies and inherited thrombophilias.

EMERGENT ISSUES/INSTRUCTIONS

A. Clients should contact the office if they have any of the following symptoms:

1. Worsening pain in an arm or leg.
2. Fever over 101°F.
3. Any temperature change in a hand or foot.
4. Any change in feeling of a hand or foot.
5. Any change in the color of a hand or foot.
6. Difficulty walking.
7. Vomiting or other illness that causes them to miss more than one dose of their medications.

B. Clients should contact the office if they are experiencing any new symptom(s) not present at their last office visit.

CONSULTATION/REFERRAL

A. If septic thrombophlebitis or DVT is diagnosed, refer the client to a physician.

B. Hospitalization is required to initiate heparin therapy.

C. Comanage pregnancy with an obstetrician.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Routine anticoagulation therapy for all pregnant individuals is not recommended. Therapeutic anticoagulation is recommended for clients with acute thromboembolism during the current pregnancy or those at high risk of thrombosis, such as individuals with mechanical heart valves.

2. Warfarin and NSAIDs are contraindicated.

3. Heparin is the preferred anticoagulant in pregnancy. Neither unfractionated heparin nor LMWH crosses the placenta.

4. Warfarin, LMWH, and unfractionated heparin do not accumulate in breast milk and do not induce an ▶

anticoagulant effect in the infant and therefore are considered compatible with breastfeeding.

B. Geriatrics:

1. Chronic NSAID use is contraindicated in the geriatric population. Short-term use requires caution in certain populations due to the risk for cardiovascular disease (CVD) and kidney injury.

2. The overall prognosis is poor for geriatric clients with septic thrombophlebitis.

3. Static or seated exercise therapy or even the use of a self-propelled wheelchair (using the feet to walk with wheelchair forward) or a rocking chair may provide an activity substitute for persons unable to participate in a walking program.

4. Be alert to the geriatric syndrome of depression related to immobility and pain.

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SYNCOPE

DEFINITION

A. Syncope is a brief, sudden loss of consciousness and muscle tone secondary to cerebral ischemia, or inadequate oxygen or glucose delivery to brain tissue. Recovery is spontaneous.

INCIDENCE

A. Syncope is a common problem in all age groups. Between 12% and 48% of healthy young adults have lost consciousness (one-third following trauma), but most do not seek medical attention. Adults older than age 75 in long-term care facilities have a 7% annual incidence of syncope and 30% have had previous episodes. Syncopal episodes account for approximately 1% to 6% of hospital admissions and 3% of ED visits.

PATHOGENESIS

A. The most common cause of syncope is inadequate cerebral perfusion caused by one of the following:

1. Vasomotor instability associated with a decrease in systemic vascular resistance and/or venous return. The following may cause syncope:

a. Vasovagal episodes.

b. Situational syncope, from coughing, micturition, and defecation.

c. Medications:

i. Vasodilators.

ii. Antiarrhythmics.

iii. Diuretics.

iv. Neurologic agents.

v. Glucose-regulating drugs.

vi. Impotence therapy.

2. Decrease in cardiac output caused by blood flow obstruction within the heart or pulmonary circulation or by arrhythmias. This may be caused by the following:

a. Aortic, pulmonic, and mitral stenosis.

b. Idiopathic hypertrophic subaortic stenosis (IHSS) or hypertrophic obstructive cardiomyopathy (HOCM).

c. Pump failure.

d. Subclavian steal syndrome.

e. Seizures.

3. Focal or generalized decrease in cerebral perfusion leading to transient ischemia due to cerebrovascular disease:

a. Carotid arterial disease.

4. Metabolic abnormalities:

a. Hypoglycemia.

b. Hypocarbia and hypoxia usually do not result in syncope unless they are profound, although consciousness may be altered.

5. Psychiatric illnesses associated with syncope include the following:

a. Generalized anxiety.

b. Panic attacks.

c. Major depressive disorders.

6. Unexplained cause.

PREDISPOSING FACTORS

A. Advanced age, caused by altered regulation of cerebral blood flow and/or systemic arterial pressure due to aging process and increased medication use.

B. Other factors, depending on etiology.

C. Medication use (previously noted).

COMMON COMPLAINTS

A. Dizziness.

B. Light-headedness.

C. Fainting with no memory of events.

OTHER SIGNS AND SYMPTOMS

A. Neuroautonomic regulations:

1. Event triggered by changing position, turning head, or wearing tight collars.

2. Nausea, warmth, diaphoresis, or weakness 1 hour after eating.

B. Cardiac causes:

1. Exercise-induced palpitations, chest pain, and shortness of breath (SOB), with no warning prior to episode.

C. Neurologic causes:

1. Vertigo.

2. Diplopia.

3. Facial paresthesias.

4. Ataxia.

5. Auditory, visual, or vestibular disturbances.

D. Metabolic or endocrine causes:

1. Restlessness.

2. Anxiety.

3. Confusion.

4. No recent food intake, low glucose level.

E. Psychiatric:

1. Graceful fainting in presence of an audience.

SUBJECTIVE DATA

A. Inquire if the client ever experienced similar symptoms or episodes before. If so, when and at what age did it begin?

B. Ask the client or witness of the episode to give a detailed description of loss of consciousness. Was loss of consciousness complete, and if so for how long? What was the posture of the client before, during, and after the event? Did it ▶

occur abruptly, or were there symptoms leading up to the event?

C. Question the client regarding events leading up to the episode, noting prodromal symptoms such as headache, aura, nausea/vomiting, light-headedness, diaphoresis, feeling, or warmth.

D. Obtain a detailed account of symptoms during and after the episode, noting mental status. Did the client recover on their own, or did the client require assistance? Were there any associated symptoms that occurred during the event? SOB, chest pain, loss of bowel, or bladder control?

E. If syncope has occurred in the past, are there any events that precipitate an episode? Exertion, exercise, coughing, or standing quickly?

F. Obtain a detailed medication history, addressing prescribed and over-the-counter (OTC) drugs, alcohol, and illicit preparations.

G. Review the client's medical history.

PHYSICAL EXAMINATION

A. Check temperature, if indicated, pulse, respirations, and blood pressure (BP).

1. Measure BP and pulse in both arms and legs. Note BP differences between the arms.

2. Measure BP several times during a 2-minute period with the client standing.

3. Check for orthostatic hypotension, which is defined as a drop of 20 mmHg or more in systolic blood pressure (SBP) on standing.

a. First, measure BP after the client lies supine for 5 to 10 minutes.

b. Then have the client stand and measure BP several times during a 2-minute period.

4. Inspect the range of motion in the neck.

B. Palpate:

1. Palpate the abdomen, noting pulsatile expansion.

C. Auscultate:

1. Auscultate the heart with position changes. Note murmurs or extra heart sounds to rule out structural disease. Identify dysrhythmias such as bradycardia, supraventricular tachycardia, tachycardia, atrioventricular (AV) blocks, atrial fibrillation (AF), bundle branch blocks, and sinus pauses or arrests.

2. Auscultate the carotid arteries.

3. Auscultate the abdomen for bruits.

D. Perform neurologic exam:

1. Perform a complete exam, if indicated, including assessing cranial nerves II to XII, Babinski reflex, and gait.

E. Mental status:

1. Assess mental health, if indicated.

DIAGNOSTIC TESTS

A. The 2017 American College of Cardiology/American Heart Association (ACC/AHA) guideline for syncope recommends the following diagnostic tests based on history and physical findings:

1. If carotid sinus syndrome is suspected, carotid sinus massage can be attempted. Avoid in clients with history of transient ischemic attack (TIA) or stroke in the past 3 months and in clients with carotid bruits. Recommend physician or cardiology specialist assistance when performing carotid massage along with continuous cardiac monitoring. Use caution when performing carotid sinus massage. Please consider contraindications, complications, and guidelines for performing procedure.

2. Echocardiogram for clients with a history of heart disease, structural heart disease, or syncope secondary to cardiovascular cause (known heart disease), family history of unexplained sudden death in the family, syncope with exertion or supine, abnormal EKG, sudden onset of palpitation prior to syncope, or arrhythmia on EKG.

3. EKG for clients with suspected arrhythmia or cardiac disease. Identify acute and old EKG changes to rule out pathologic Q wave, ST-segment elevation, and left ventricular (LV) hypertrophy.

4. Orthostatic challenge test if syncope is related to position change or suspect reflex mechanism.

5. Neurologic/serum laboratory testing for other concerns of nonsyncopal loss of consciousness. Laboratory testing includes chemistry profile, thyroid-stimulating hormone (TSH), and free T4. Consider a glucose tolerance if diabetes is suspected. Brain natriuretic peptide may be useful to evaluate cardiac versus noncardiac causes of syncope.

6. Chest radiography, for essential baseline data. Wide mediastinum signals aortic dissection.

7. In-hospital monitoring is recommended for unstable clients in life-threatening conditions.

8. Holter monitor for 24 to 48 hours.

9. External event monitor.

10. Exercise testing is recommended for clients with syncope that occurs during, or quickly after, cessation of exercise. Echocardiogram is recommended prior to this testing.

11. Cardiac catheterization.

12. Lung scan.

13. Treadmill test.

14. Electrophysiologic studies are recommended for clients with unexplained syncope.

DIFFERENTIAL DIAGNOSES

A. Irregular neuroautonomic regulations:

1. Neurocardiogenic causes.

2. Situational causes, such as coughing, defecation, diving, micturition, sneezing, swallowing, trumpet playing, vagal stimulation, weightlifting, postprandial state.

3. Orthostatic causes:

a. Hyperadrenergic state.

b. Hypoadrenergic state, primary or secondary autonomic insufficiency.

c. Carotid sinus syncope.

d. Cardioinhibitory state.

e. Vasodepressor stimulation.

f. Mixed.

B. Cardiac causes:

1. Mechanical causes, such as aortic dissection, aortic stenosis, atrial myxoma, cardiac tamponade, global myocardial ischemia, hypertrophic cardiomyopathy, mitral stenosis, myocardial infarction (MI), prosthetic valve dysfunction, pulmonary embolism (PE), pulmonary hypertension (HTN), pulmonary stenosis, and Takayasu arteritis.

2. Electrical causes, such as AV block, long QT syndrome, pacemaker, sick sinus syndrome, supraventricular tachyarrhythmias, and ventricular tachyarrhythmias.

C. Neurologic causes:

1. Neuralgias: glossopharyngeal, trigeminal. ▶

2. Normal pressure hydrocephalus.
3. Subclavian steal.
4. Vertebrobasilar artery disease: compression, migraine, and TIA.

D. Metabolic or endocrine causes: hypoadrenalism, hypoglycemia, hyponatremia, hypothyroidism, and hypoxia.

E. Psychiatric causes: anxiety, hysteria, major depression, panic disorder, somatization, and hyperventilation syndrome.

PLAN

A. General interventions:
1. Management is directed at primary cause of the episode.

B. Client teaching:
1. If the client has orthostatic hypotension, suggest that they wear elastic stockings, change positions slowly, sleep with the head of the bed elevated, and exercise their legs before standing.
2. If syncope is induced by situations, warn the client to avoid or alter their approach to such precipitating events.
3. If the client has prodromal symptoms, such as nausea, light-headedness, pallor, sweating, or palpitations, advise them to lie down when they occur.
4. If the client has hypersensitive carotid sinus reflex, recommend that they loosen their collar.
5. Instruct client to avoid prolonged standing. If they cannot avoid it, they should contract their calf muscles to increase venous blood flow.
6. Some driving restrictions exist for clients at risk of recurrent syncope. Driving restrictions are enforced by state law. Review restrictions with the client and the family as indicated by diagnosis.

C. Dietary management:
1. If not contraindicated, instruct clients with orthostatic hypotension to use salt liberally.

D. Pharmaceutical therapy for neurocardiogenic syncope includes the following:
1. Nonpharmacologic methods suggested:
a. Avoid volume depletion.
b. Maintain adequate sodium levels by increasing salt intake in their diet.
c. Wear thigh-high elastic support hose with 30 to 40 mmHg pressure.
d. Orthostatic training is also recommended two times a day.
2. Drugs of choice: beta-blockers (Inderal 80–160 mg/d, metoprolol 50–100 mg/d) for vasovagal syncope.
3. Fludrocortisone acetate (Florinef Acetate), a corticosteroid, may be used alone or with beta-blockers. Initial dosage is 0.1 to 0.4 mg/d; this may be increased gradually to 1.0 to 2.0 mg/d.
4. Other drugs include anticholinergic agents (disopyramide 100–200 mg BID sustained-release), and selective serotonin reuptake inhibitors (SSRIs; Zoloft 50 mg/d, Prozac 20 mg/d, Paxil 20 mg/d).

FOLLOW-UP

A. Scheduling of return visits depends on the etiology and severity of syncope and on whether the client has been placed on medications.

CONSULTATION/REFERRAL

A. Consult with or refer the client to a physician when cardiac or neurologic involvement is suspected.

B. Consult with or refer the client to a physician if medication therapy is required.

INDIVIDUAL CONSIDERATIONS

A. Adults:
1. In young adult athletes, be aware of symptoms of Marfan syndrome.
2. In older adults, coronary atherosclerosis may present along with syncope.

B. Geriatrics:
1. The most common cause of syncope in the elderly is postural hypotension, which can be due to a combination of factors, both intrinsic and extrinsic. This is often the impetus for falls.
2. Intrinsic factors can include noncompliant arteries, reduced skeletal muscle pumping action of venous return due to physical inactivity, and aging of the sinoatrial node and conduction system due to progressive atherosclerosis and/or structural heart disease.
3. The most common extrinsic factor involves medications that lower BP and cause vasodilation of blood vessels. Nurse practitioners (NPs) should routinely evaluate their geriatric client's cardiac medications and reduce doses or deprescribe when appropriate.
4. In the elderly, syncope often has more than one cause. For example, the combination of taking several heart and BP drugs and standing for a long period of time may lead to syncope even though no single factor might cause it.

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CLIENT TEACHING GUIDE

ATHEROSCLEROSIS AND HYPERLIPIDEMIA

PROBLEM Hyperlipidemia (excess lipids in the blood) is often referred to as "hardening of the arteries." The excess lipids increase your risk of developing heart disease and heart attacks.

CAUSE Elevated blood cholesterol levels lead to plaque formation in the walls of the major arteries in the body. The higher the level of low-density lipoprotein (LDL), or "bad" cholesterol, the greater the chance of developing heart disease. On the other hand, the higher the level of high-density lipoprotein (HDL), or "good" cholesterol, the lower the risk of heart disease.

PREVENTION/TREATMENT PLAN Lowering your risk of heart disease involves the following:

A. Diet changes to lower your bad cholesterol (LDL) and raise your good cholesterol (HDL).
B. Lose weight. Start with losing 5 to 10 lbs.
C. Start or increase your physical activity. Walking is a good exercise to start getting active.
D. Other ways to modify your risk factors:
  1. Stop smoking. **It is never too late to stop smoking.**
  2. Control your blood pressure.
  3. If you are a diabetic, control your blood sugar level.

Activity: Regular exercise, such as walking vigorously for 30 minutes three times a week, increases your good cholesterol levels, lowers blood sugar, and promotes weight loss.

Diet: Follow the Dietary Approaches to Stop Hypertension (DASH) and low-fat/low-cholesterol diet.

A. Reduce total fat calories and cholesterol.
B. Reduce total saturated fats and replace with monounsaturated fats, such as canola oil, olive oil, and margarine.
C. Increase fiber with oatmeal, bran, or fiber supplements.
D. Increase daily intake of fruits and vegetables.
E. Try garlic, soy protein, and vitamin C to help lower LDL cholesterol.

Medications: You may be prescribed a medicine to lower your cholesterol. You will need to come to the doctor's office and have your blood drawn to monitor your liver and cholesterol levels.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Chest pain.
B. Shortness of breath or trouble breathing while exercising.
C. Abdominal pain.
D. Muscle pain or weakness.
E. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

ATRIAL FIBRILLATION

PROBLEM Atrial fibrillation (AF) is the condition that causes the upper chambers of the heart (the atria) to beat faster and irregularly (also called fibrillation). The upper chambers of the heart do not beat at the same time as the lower chambers (the ventricles). When AF occurs, blood clots can form in the heart and then travel to the brain, causing a stroke.

CAUSES AF is caused by a malfunction of the heart's pacemaker. Many things can cause the heart's pacemaker to malfunction, including excessive alcohol intake, emotional stress, physical stress, recent heart surgery, medication side effects, and a long list of medical conditions. These medical conditions include coronary artery disease, leaky heart valves, high blood pressure (BP), heart failure, heart attack, thyroid disease, infections, inflammation around the heart, sleep apnea, obesity, and lung diseases such as chronic obstructive pulmonary disease, bronchitis, asthma, and emphysema.

PREVENTION

A. Stop smoking. Discuss smoking cessation with your healthcare provider. **It is never too late to stop smoking.**
B. Reduce or eliminate intake of alcohol and caffeine.
C. Lose weight. Discuss losing weight with your healthcare provider.
D. Make a list of your current medical conditions and current medications. Keep an updated copy in your wallet.
E. When traveling:
  1. Always travel with enough of your medication to last through your vacation plus an additional 3 days.
F. Take all of your medications as prescribed. If you become ill and are unable to take your medications, call your healthcare provider's office.

TREATMENT PLAN

A. Take your medications as ordered by your healthcare provider.
B. Effectively manage all other medical conditions, paying special attention to cholesterol, BP, thyroid disease, sleep apnea, and any lung diseases.
C. Follow up with your primary healthcare provider and/or cardiologist on a regularly scheduled basis.
D. If you are on a blood thinner that requires regular blood work, make sure you get your blood checked on the schedule set by your healthcare provider.

Activity: Get regular exercise, after discussing the type and frequency of exercise that is safe for you with your healthcare provider.

Diet: Eat a balanced, low-fat, and low-salt diet in addition to dietary guidelines suggested by your healthcare provider.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office in Case of the Following:

A. Seek care in the ED if you have any of the following symptoms:
  1. Palpitations or a fluttering in the chest.
  2. Chest pain.
  3. Weakness or extreme tiredness.
  4. Shortness of breath at rest or with activity.
  5. Dizziness, disorientation, confusion, or headache.
  6. Passing out or losing consciousness.
  7. Frequent urination or a compelling urge to urinate.
  8. Anxiety or panic symptoms.
  9. Sudden weakness in an arm or leg.
  10. Sudden change in vision.
  11. Trouble speaking or understanding others.

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CLIENT TEACHING GUIDE

ATRIAL FIBRILLATION (*CONTINUED*)

12. If taking a blood thinner and have any of the following symptoms:
   a. Nosebleeding that will not stop or coughing up blood.

B. Contact the office if you are taking a blood thinner and have any of the following symptoms:
   1. Vomiting or other illness that causes you to miss more than one dose of your medications.
   2. Vomit that is bright red or is dark with the appearance of coffee grounds or grape jelly.
   3. Bleeding concerns, such as blood in stool, stools that appear black or tarry in color, and/or heavy menstrual periods.

C. Contact the office if you are experiencing any new symptom(s) not present at your last office visit.

D. Other: _____

Phone: _____

RESOURCES American Heart Association: https://www.heart.org/en/health-topics/atrial-fibrillation

National Heart, Lung, and Blood Institute: https://www.nhlbi.nih.gov/health-topics/atrial-fibrillation

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CLIENT TEACHING GUIDE

CHRONIC VENOUS INSUFFICIENCY

PROBLEM Chronic venous insufficiency (CVI) is a condition in which blood has difficulty flowing back to the heart from the arms or legs. This usually occurs when the valves along the inside of the veins are damaged and allow blood to flow backward. The pooling of blood leads to swelling, pain, a heavy feeling, darkening of the skin, and infections. Without treatment, CVI can lead to blood clots and serious infections that could lead to amputation.

CAUSES Many things may increase the chance of having CVI. Some, like gender, age, or how tall you are, cannot be changed. Others can be changed; these include prolonged standing or sitting and excessive weight.

PREVENTION

A. Avoid standing or sitting for long periods of time.
B. If overweight or obese, lose weight. Discuss what you can do to lose weight with your healthcare provider.

TREATMENT PLAN

A. Take your medications as ordered by your healthcare provider.
B. Wear compression stockings. Put them on before getting out of bed in the morning. Take them off just before going to bed at night.
C. Raise the affected arm or leg whenever lying down to improve pain and swelling.
D. Avoid standing or sitting for long periods of time.
E. Follow up with your primary healthcare provider on a regularly scheduled basis.

Activity:

A. Get regular exercise. Discuss what type and frequency of exercise is safe for you with your healthcare provider.
B. Exercise leg muscles by pumping your ankles when sitting. Rocking in a rocking chair is another option.

Diet: Discuss the type of diet that best suits your needs with your healthcare provider: diabetic diet, low-fat diet, low-cholesterol diet, and/or low-sodium diet.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have the Following:

A. Seek care in the ED if you have a change in color of a foot or hand.
B. Contact the office if you are experiencing any of the following:
1. Fever over 101°F.
2. Increased redness, pain, tenderness to touch, swelling, and/or warmth.
3. New wound or sore on the affected arm or leg.
4. Sudden shortness of breath.
5. Chest pain.
6. If taking aspirin or a blood thinner and have any of the following symptoms:
   a. Vomiting or other illness that causes you to miss more than one dose of your medications.
   b. Vomit that is bright red or dark and looks like coffee grounds or grape jelly.
   c. Blood in your bowel movements that looks black or tarry in color.
   d. Severe headache.
   e. Sudden weakness in an arm or leg.
   f. Memory loss or confusion.
   g. Sudden change in vision.
   h. Trouble speaking or understanding others.

C. Contact the office if you are experiencing any new symptom(s) not present at your last office visit.
D. Other: _____

Phone: _____

RESOURCE Vascular Web: https://vascular.org/patient-resources/vascular-conditions/chronic-venous-insufficiency

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CLIENT TEACHING GUIDE

DEEP VEIN THROMBOSIS

PROBLEM You have inflammation or a blood clot in one of the veins in your body. Phlebitis is inflammation, and thrombosis means a blood clot. Symptoms include the following:

A. Pain.
B. Fever.
C. Swelling.
D. Tenderness in the affected leg or arm.
E. The vein may feel somewhat "hard" to touch.

CAUSE Blood clots form because of bedrest, surgery, a heart attack, a severe illness, and birth control pills. A blood clot can also form after breaking a hip or leg, pregnancy, with cancer, and with some medications.

TREATMENT PLAN/CARE

A. If you have phlebitis, you may be given an anti-inflammatory medicine.
B. Some clients need to go to the hospital and get intravenous (IV) medicine to break up the clot.
C. You may be given blood thinners either as a pill or by self-injection.
D. Be sure to take all of the medicines given as directed to help with the blood clot.
E. Do not smoke: This worsens your condition. **It is never too late to stop smoking.**
F. If you currently take any hormones, such as birth control pills, your healthcare provider may talk to you about stopping them.
G. Manage all other medical conditions, especially high blood pressure, diabetes, and high cholesterol, and try to lose any extra weight.
H. Follow up with your primary healthcare provider and/or cardiologist on a regularly scheduled basis.

Activity:

A. Get out of bed as soon as possible after surgery.
B. While in bed, perform range-of-motion exercises with your legs.
C. Exercise leg muscles by pumping your ankles when sitting.
D. Do not sit with your legs crossed.
E. Avoid standing or sitting for long periods of time.
F. Do not wear tight clothing, such as knee-high hosiery.
G. Wear special supportive hosiery called compression stockings. Put them on before getting out of bed in the morning and take them off before going to bed at night.
H. When traveling:
    1. Try to take rest breaks on a regular basis.
    2. Continue to do the ankle pumping exercise when in the car or on the plane.
    3. Wear loose-fitting clothes that are comfortable.
    4. Avoid drinking alcohol.
    5. Drink plenty of fluids unless you are instructed not to do so by your healthcare provider.
    6. Ask your healthcare provider if you should wear compression stockings.
I. Wear a MedicAlert bracelet if you are put on blood thinners; you will notice that you bruise more easily while on your blood thinner.
J. You may need to come back to the office to have your labs drawn.

Diet:

A. Discuss the type of diet that best suits your needs: diabetic, low-fat, low-cholesterol, and/or low-sodium.
B. If you are taking the blood thinner Coumadin, you will be given a list of foods that should not be eaten. These foods interfere with how the blood thinner works.
C. Avoid alcohol.

(*continued*)

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CLIENT TEACHING GUIDE

DEEP VEIN THROMBOSIS (*CONTINUED*)

Medications: If you currently take birth control pills, ask your healthcare provider if you should stop taking them. You may be prescribed a blood thinner by injection or pills. Continue to take this medicine even if you feel better.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have the Following:

A. Seek care in the ED if you have any of the following symptoms:
   1. Sudden shortness of breath.
   2. Chest pain.
   3. A change in color of a foot or hand.
   4. If taking a blood thinner and have any of the following symptoms:
      a. Nosebleed that will not stop with pressure.
      b. Coughing up blood.

B. Contact the office if you are experiencing any of the following:
   1. Fever over 101°F.
   2. Increased redness, pain, tenderness to touch, swelling, and/or warmth.
   3. New wound or sore on the affected arm or leg.
   4. If you are taking a blood thinner and have any of the following symptoms:
      a. Nosebleed that will not stop with pressure.
      b. Coughing up blood.
      c. Vomiting that is bright red or dark that looks like coffee grounds or grape jelly.
      d. Blood in your bowel movements that looks black or tarry in color.
      e. Severe headache.
      f. Sudden weakness in an arm or leg.
      g. Sudden change in vision.
      h. Trouble speaking or understanding others.
      i. Memory loss or confusion.

C. Contact the office if you are experiencing any new symptom(s) not present at your last office visit.

D. Other: _____

Phone: _____

You can build your own identification bracelets or neck chains from American Medical ID: www.americanmedical-id.com/category/medical-ID-bracelets-62.

RESOURCE Vascular Cures—What Is Deep Vein Thrombosis: https://vascularcures.org/deep-vein-thrombosis-dvt

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CLIENT TEACHING GUIDE

LYMPHEDEMA

PROBLEM Lymphedema is the backup of fluid in the lymphatic system into an arm or leg. The fluid causes severe swelling, which restricts movement and can lead to infection and, in rare cases, a form of cancer called lymphangiosarcoma.

CAUSES Many factors can cause lymphedema. These include a diagnosis of cancer treated with radiation, surgical removal of lymph nodes, infection, and disorders from birth that affect the structure of the lymph system.

PREVENTION

A. Protect your arm or leg while recovering from cancer treatment.
B. Avoid heavy lifting, if it involves an arm.
C. Avoid strenuous exercise.
D. Avoid heat on your arm or leg.
E. Avoid tight clothing.
F. Inspect the affected limb daily. Note any cracks or cuts that increase the risk of infection, and treat as directed.
G. Apply lotion daily to protect and prevent dry skin.
H. Avoid burns to the skin.
I. Take extra care when shaving and use a clean razor on clean skin.
J. Use insect repellent to prevent insect bites, which could become infected.
K. Wash skin daily with a mild soap and dry skin carefully paying special attention to any skin folds.
L. Prevent excessive sun exposure by applying sunscreen or wearing appropriate clothing to cover skin.

TREATMENT PLAN

A. Raise the affected arm or leg when lying down to reduce pain and swelling.
B. Wear compression stockings. Put them on before getting out of bed in the morning. Take them off just before going to bed at night.
C. Apply lotion every day to the affected area.
D. Check the affected area every day and report any skin changes to your healthcare provider, especially any cracks or cuts.
E. Follow up with your primary healthcare provider and other specialty providers on a regularly scheduled basis.

Activity:

A. Get regular exercise. Discuss with your healthcare provider or physical therapist what type and frequency of exercise is safe for you.
B. Exercise leg muscles by pumping your ankles when sitting. Rocking in a rocking chair is another option.

Diet:

A. Discuss with your healthcare provider the type of diet that best suits your needs: diabetic diet, low-fat diet, low-cholesterol diet, and/or low-sodium diet.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Fever greater than 101°F.
B. Increased redness, pain, tenderness to touch, and/or warmth in the affected arm or leg.
C. New pain, swelling, or warmth in an arm or leg.
D. Other: _____

Phone: _____

RESOURCES National Cancer Institute: cancer.gov/cancertopics/pdq/supportivecare/lymphedema/healthprofessional/page2

National Lymphedema Network: lymphnet.org

Vascular Cures Lymphedema Flyer - https://vascularcures.org/wp-content/uploads/2021/08/VC_Lymphedema_Final-HR.pdf

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CLIENT TEACHING GUIDE

PERIPHERAL ARTERIAL DISEASE

PROBLEM Peripheral arterial disease is a condition in which fatty deposits build up on the inside of the vessels that carry blood to the hands and feet. This makes it difficult for blood to travel to hands and feet, causing pain with activity and when resting. If these blockages increase in size, the pain will also worsen; infections can also occur, which could lead to amputation.

CAUSES Many things cause these fatty deposits to build up inside arteries. Some factors such as age, gender, and genetics cannot be changed. Others can be changed; these include smoking, diabetes, high cholesterol, high blood pressure (BP), and excessive weight.

PREVENTION

A. Stop smoking. **It is never too late to stop smoking.**
B. To prevent injury that could progress into an infection, do the following:
  1. Wear well-fitting shoes that protect your feet.
  2. Look and feel inside your shoes before putting them on.
  3. Look at your feet daily for any signs of injury or infection.
  4. Dry feet well after bathing, including between toes.
  5. Do not trim your own toenails or shave off calluses. A healthcare provider should do this for you to prevent infection.
  6. Do not use a heating pad or hot water on your hands or feet to keep warm. Wear gloves or socks instead.

TREATMENT PLAN

A. Take your medications as ordered by your healthcare provider.
B. Effectively manage all other medical conditions, paying special attention to cholesterol, BP, diabetes, and obesity.
C. Follow up with your primary healthcare provider and/or cardiologist on a regularly scheduled basis.

Activity:

A. Get regular exercise, after discussing with your healthcare provider the type and frequency of exercise that is safe for you.

Diet:

A. Discuss the type of diet that best suits your needs: diabetic diet, low-fat diet, low-cholesterol diet, and/or low-sodium diet.
B. Follow the dietary guidelines suggested by your healthcare provider.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have the following:

A. Contact the office if you have any of these symptoms:
  1. Worsening pain in arm or leg.
  2. Fever over 101°F.
  3. Any temperature change in a hand or foot.
  4. Any change in feeling of a hand or foot.
  5. Any change in the color of a hand or foot.
  6. Difficulty walking.
  7. Vomiting or other illness that causes you to miss more than one dose of your medications.
  8. If taking aspirin and have any of the following symptoms:
    a. Vomit that is bright red or dark and looks like coffee grounds or grape jelly.
    b. Blood in your bowel movements (BMs) that looks black or tarry in color.
B. Contact the office if you are experiencing any new symptom(s) not present at your last office visit.
C. Other: _____

Phone: _____

RESOURCE Vascular Cures–PAD: http://vascularcures.org/peripheral-artery-disease-pad

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CLIENT TEACHING GUIDE

SUPERFICIAL THROMBOPHLEBITIS

PROBLEM Superficial thrombophlebitis occurs when a vein is irritated or injured. The portion of the vein that is affected can develop a blood clot or become infected, causing redness, swelling, and pain. If untreated, the infection and/or blood clot can progress into a life-threatening condition such as sepsis, deep vein thrombosis, or pulmonary embolism.

CAUSES Many things can cause irritation and injury to veins. These include a recent intravenous line placed during a hospital stay, infection, some medications, and pregnancy. Some risk factors cannot be changed, such as genetics, whereas others can be changed, including inactivity and excessive weight.

PREVENTION

A. Avoid standing or sitting for long periods of time.
B. If you smoke, stop or reduce the amount you smoke. **It is never too late to stop smoking.**
C. If overweight or obese, lose weight. Discuss what you can do to lose weight with your healthcare provider.

TREATMENT PLAN

A. Take your medications as ordered by your healthcare provider.
B. Raise the affected arm or leg when lying down to improve pain and swelling.
C. Warm compress to the affected area may improve pain.
D. Wear compression stockings. Put them on before getting out of bed in the morning. Take them off just before going to bed at night.
E. Avoid standing or sitting for long periods of time.
F. Follow up with your primary healthcare provider on a regularly scheduled basis.

Activity:

A. Get regular exercise. Discuss with your healthcare provider what type and frequency of exercise is safe for you.
B. Exercise leg muscles by pumping your ankles when sitting. Rocking in a rocking chair is another option.

Diet:

A. Discuss with your healthcare provider the type of diet that best suits your needs: diabetic diet, low-fat diet, low-cholesterol diet, and/or low-sodium diet.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need To Notify the Office If You Have Any of the Following:

A. Worsening pain in an arm or leg.
B. Fever over 101°F.
C. Sudden shortness of breath.
D. Chest pain.
E. New pain, swelling, change in color, or temperature in an arm or leg.
F. Difficulty walking.
G. Vomiting or other illness that causes you to miss more than one dose of your medications.
H. If you are taking a blood-thinning medication and have any of the following symptoms:
  1. Vomit that is bright red or dark and looks like coffee grounds.
  2. Bright-red blood in your stools, or black, tarry stools.
  3. Severe headache.
  4. Sudden weakness in an arm or leg.
  5. Memory loss or confusion.
  6. Sudden change in vision.
  7. Trouble speaking or understanding others.
I. Other: _____

Phone: _____

RESOURCE Mayo Clinic—Superficial Thrombophlebitis: mayoclinic.com/health/thrombophlebitis/DS00223

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CLIENT TEACHING GUIDE

VARICOSE VEINS

PROBLEM Varicose veins are caused when the valves inside the veins are damaged and allow blood to flow backward instead of toward the heart. This backflow of blood increases the pressure in the vein, leading to pain and swelling; this makes them more visible. Varicose veins can worsen and increase the risk of blood clots, infection, bleeding, and changes to the skin.

CAUSES There are many factors that increase the chance of developing varicose veins. Some, such as age and genetics, cannot be changed. Some can be changed; these include prolonged standing, restrictive clothing, excessive weight, and smoking.

PREVENTION

A. Avoid prolonged standing. If prolonged standing is required, shift weight from one leg to the other.
B. When possible, prop your feet up instead of keeping both feet on the floor or letting them hang down.
C. Stop smoking. Discuss smoking cessation with your healthcare provider.

TREATMENT PLAN

A. Raise the affected arm or leg when lying down to improve pain and swelling.
B. Wear compression stockings. Put them on before getting out of bed in the morning. Take them off just before going to bed at night.
C. Avoid standing or sitting for long periods of time.
D. Follow up with your primary healthcare provider on a regularly scheduled basis.

Activity:

A. Get regular exercise. Discuss with your healthcare provider what type and frequency of exercise is safe for you.
B. Exercise leg muscles by pumping your ankles when sitting. Rocking in a rocking chair is another option.

Diet:

A. Discuss with your healthcare provider the type of diet that best suits your needs: diabetic diet, low-fat diet, low-cholesterol diet, and/or low-sodium diet.
B. If you are taking the blood thinner Coumadin, ask your healthcare provider about which foods are high in vitamin K and whether you should limit those foods in your diet.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Other: _____

Phone: _____

EMERGENT ISSUES/INSTRUCTIONS

A. Seek care in the ED if you have any of the following symptoms:
1. **A bleeding varicosity with eroded surrounding skin.**
2. **A varicosity that has bled and is at risk of bleeding again.**
3. **An ulceration that is worsening and/or painful despite treatment**.

RESOURCE Vascular Cures–Varicose Veins: http://vascularcures.org/varicose-veins

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CHAPTER 14

GASTROINTESTINAL GUIDELINES

L. Douglas Smith Jr., Ann McQueen Blair, and Justin Calabrace

ABDOMINAL PAIN

DEFINITION

A. Abdominal pain is a common and nonspecific complaint. The evaluation of abdominal pain is challenging for clinicians as it may represent a benign complaint, or it may herald significant abdominal pathology. The responsibility of clinicians is to determine if clients can be observed and treated symptomatically or if they require further investigation and referral to a specialist. Pain in the abdomen is secondary to problems relating to abdominal organs, and it is categorized as follows:

1. Acute pain: pain of less than a few days that has worsened progressively until presentation.
2. Chronic pain: pain lasting greater than 12 weeks and remaining unchanged over a period of months to years. Recurrent abdominal pain is also included in this category.
3. Subacute pain: pain lasting from a few days to several weeks.
4. Emergent pain: pain that lasts 3 hours or longer, accompanied by a fever or vomiting.

B. Pain may be categorized by the following descriptions:

1. Visceral pain is usually dull and aching in character.
2. Parietal pain is sharp and well-localized.
3. Referred pain is aching and perceived to be near the surface of the body.

INCIDENCE

A. Abdominal pain is a common complaint. While there is significant variance among the literature, as many as 50% of adults will complain of abdominal pain at some point. Gastroenteritis and irritable bowel syndrome (IBS) are the most common causes of acute pain, and chronic stool retention is the most common cause of chronic pain. Other causes of abdominal pain include the following:

1. Acute appendicitis: occurs 233:100,000.
2. Acute cholecystitis: varies according to age, sex, and ethnic origin.
3. Intestinal obstruction, usually small intestines: accounts for 20% of acute abdominal conditions.
4. Pregnancy-associated abdominal pain: ectopic pregnancy, miscarriage, and abruptio placentae.

PATHOGENESIS

A. Pathogenesis depends on the origin of pain. Pain may result from inflammation, ischemia, distension, altered motility, obstruction, or ulceration.

PREDISPOSING FACTORS

A. Abdominal trauma.
B. Motor vehicle accidents with trauma such as ruptured spleen.
C. Dietary intolerances.
D. Pregnancy.
E. Torsion.
F. Psychogenic pain.
G. Sickle cell disease.
H. Infection.
I. Diabetes.
J. Vascular disease.
K. Congenital or acquired abdominal abnormalities.

COMMON COMPLAINTS

A. Clinical presentation of abdominal pain is determined in part by the site of the involvement:

1. Acute or chronic onset of pain.
2. Vomiting.
3. Diarrhea.

OTHER SIGNS AND SYMPTOMS

A. Referred shoulder pain.
B. Fever.
C. Nausea and/or projectile vomiting.
D. Rigid abdomen.
E. Abdominal distension.
F. Constipation or diarrhea.
G. Guarding.
H. Rebound tenderness.
I. Biliary pain and right subcostal tenderness.
J. Anorexia.
K. Periumbilical discomfort; consider appendicitis if, within 2 to 12 hours, pain localizes in right lower quadrant (RLQ) at McBurney point.
L. Dysuria.
M. Abdominal mass; do not overlook the possibility of pregnancy as the cause of a mass.
N. Melena (most common in peptic ulcer disease [PUD]).
O. Tachycardia.

SUBJECTIVE DATA

Evaluate for a "surgical abdomen," defined as a rapidly worsening pathology in the absence of surgical intervention that will result in a poor prognosis. Clients should not eat or drink while a diagnosis of a surgical abdomen remains under consideration. Once a surgical abdomen has been excluded, the remainder of the evaluation will be guided by the chronicity of symptoms, along with the location of pain.

The contributions of Cheryl A. Glass and Kathy R. Reese to this chapter in prior editions are acknowledged here.

A. Review onset, duration, course, and quality of pain:
1. When did the pain start?
2. What were you doing when the pain started?
3. Has this ever occurred before?
4. What was the primary diagnosis?
5. What was the previous treatment and was it effective?
6. Is there anyone else having the same symptoms in your home?
7. Review the progression of pain.
B. Determine pain rating on a 10-point scale, with 0 being no pain and 10 being equivalent to the worst pain the client has ever felt.
C. Qualify the duration of pain in minutes, hours, days, weeks, or months. Does the pain interfere with sleep?
D. Review the pattern of pain:
1. Review aggravating factors.
2. Review alleviating factors.
3. Does the pain radiate?
4. Does the pain have any relationship to food intake?
E. Questions specific to females:
1. Determine the client's last menstrual period (LMP).
2. Has the client had a hysterectomy or tubal ligation?
3. Does the client have a recent history of dyspareunia or dysmenorrhea that suggests pelvic pathology?
4. Is there any history of physical abuse?
5. What type of contraception is used? Specifically evaluate for an intrauterine device (IUD).
F. Review the client's current medications and drug history, especially antibiotic, laxative, acetaminophen, aspirin, and nonsteroidal anti-inflammatory drug (NSAID) use. Clients taking corticosteroids may have a significant masking of pain.
G. Rule out abdominal trauma from domestic violence, motor vehicle accidents, falls, or assaults.
H. Review bowel habits and note changes: constipation, diarrhea, anorexia, food intolerance, nausea, vomiting, or bloating.
I. Review the client's history for sickle cell disease. Any individual of African American or Mediterranean descent presenting with leg or abdominal pain should be questioned regarding sickle cell disease or trait.
J. Review urinary function. Is there any urinary frequency, urgency, dysuria, flank pain, or back pain? If the client is male, do they have any hesitancy, difficulty starting the urine stream, nocturia, low urinary volume, or any lower abdominal distension indicating urinary retention?
K. Review alcohol intake/history.
L. Has the client had any unexplained weight loss?
M. Evaluate sexual activity to rule out potential sexually transmitted infection (STI):
1. Evaluate if clients have new partners.
2. Are their partners experiencing any symptoms?

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure (BP); include orthostatic BP.

Tachycardia or hypotension may be signs of a ruptured aortic aneurysm, septic shock, gastrointestinal (GI) hemorrhage, ectopic pregnancy, or volume depletion. Absence of a fever in the older adults or immunosuppressed does not exclude a serious illness or infection.

B. Inspect:
1. Observe general appearance: facial expressions, walk, skin turgor; refusal to move/writhing; note grimacing during exam.
2. Perform eye and oral exam to rule out iritis and aphthous ulcers of the mouth (extraintestinal manifestations of inflammatory bowel disease [IBD]).
3. Examine the abdomen for the presence of a hernia at the umbilicus, groin, or near the site of prior surgical incisions.
4. Examine the abdomen for overt masses or pulsations.
5. Examine the eyes and skin for jaundice.
6. Observe for any bruising or other signs of domestic violence in the "bathing suit" areas: breasts, abdomen, back that would be easily covered with clothes.
C. Auscultate:
1. Auscultate for bowel sounds in all four quadrants of the abdomen.
2. Evaluate heart and lungs.
3. Listen for bruits of aorta, iliac, and renal bruits.
D. Percuss:
1. Percuss abdomen for tympanic and dullness sounds.
E. Palpate:
1. Palpate abdomen for masses, rebound tenderness, and peritoneal signs:
a. Before palpating the abdomen, ask the client to bend the knees and place hands at sides to help with relaxation of the wall musculature.
b. Older adults may lack classical peritoneal signs of rebound and guarding.
2. Check the abdomen for tender pulsatile mass at midline; it may indicate abdominal aortic aneurysm (AAA).
3. Palpate back; check for cerebrovascular accident (CVA) tenderness.
4. Perform a bimanual examination in females regardless of whether the client has had a hysterectomy or is postmenopausal:
a. Evaluate the size and symmetry of the uterus.
b. Evaluate the adnexal areas for presence of appropriately sized, mobile ovaries. A fixed, painful adnexal mass is suggestive of an endometrioma or tubo-ovarian abscess.
c. Endometriosis is suggested by localized tenderness in the cul-de-sac or uterosacral ligaments, palpable tender nodules, pain with uterine movement, or tender fixation of adnexal or uterus in a retroverted position.
5. Check for obturator sign, or abdominal pain in response to passive internal rotation of the right hip from 90° angle hip–knee flexion position. A positive sign indicated pain secondary to irritation of obturator muscle with inflamed appendix.
6. Assess psoas sign or increased abdominal pain occurring when the client attempts to raise their right thigh against the pressure of your hand placed over their right knee. Pain is caused by inflammation of the psoas muscle in acute appendicitis.
F. Rectal exam:
1. Perform a rectal exam, including testing of stool for occult blood. **Failure to perform a rectal examination in clients with undifferentiated abdominal pain can lead to misdiagnosis.**

DIAGNOSTIC TESTS

A. In all individuals of childbearing age, assume the client is pregnant until proven otherwise. Vaginal bleeding with or without abdominal pain should prompt a transvaginal ultrasound and a serum human chorionic gonadotropin (HCG).
B. Complete blood count (CBC) with differential.
C. Comprehensive metabolic profile (CMP). ▶

D. Amylase.
E. Lipase.
F. Urinalysis; consider culture.
G. Stool guaiac or fecal immunoassay test for occult blood.
H. Plain x-ray abdomen; x-ray with oral contrast for certain indications.
I. CT abdomen/pelvis.
J. Abdominal ultrasonography; may be limited in scope depending on indication (e.g., right upper quadrant [RUQ] if hepatic or gall bladder pathology suspected).
K. *Helicobacter pylori* fecal antigen.
L. Coproporphyrin, if lead poisoning is suspected.
M. Chest x-ray.
N. GI series radiography.
O. Endoscopy.
P. Sigmoidoscopy.
Q. Barium enema (BE): Avoid with suspected obstruction.
R. Consider endoscopic retrograde cholangiopancreatography (ERCP) to visualize the distal common bile duct.
S. EKG to evaluate for cardiac etiology.
T. Blood cultures for clients with abdominal pain associated with either fever or hypothermia or when sepsis is suspected.

DIFFERENTIAL DIAGNOSES

A. Location and duration of abdominal pain can often help significantly in narrowing the differential diagnosis.

1. RUQ pain:
   a. Acute cholecystitis and biliary colic:
      i. Biliary tract: increased serum amylase.
      ii. Ascending cholangitis: fever and jaundice in a client with RUQ pain.
      iii. Acute cholecystitis: maximal pain in the RUQ or epigastrium, radiating to the scapular region, accompanied by nausea, vomiting, and fever without jaundice. "Murphy's sign," or tenderness to palpation during an inspiratory pause, may be seen with acute cholecystitis. RUQ tenderness to percussion or pressure of the gallbladder is also a suggestive finding.
      iv. Ketoacidosis: presents with severe abdominal pain in 8% of instances and may be accompanied by emesis and an elevated white cell count. Acute intra-abdominal events such as cholecystitis may be the precipitant of ketoacidosis, as well as metabolic abnormalities including diabetic, starvation, and alcoholic ketoacidoses.
   b. Acute hepatitis.
   c. Hepatic abscess.
   d. Hepatomegaly due to congestive heart failure (CHF).
   e. Perforated duodenal ulcer (DU).
   f. Acute pancreatitis; bilateral pain.

Pancreatitis is accompanied by an increased serum lipase.

   g. Herpes zoster.
   h. Myocardial ischemia.
   i. Pleural or pulmonary pathology (e.g., pneumonia, pulmonary embolism [PE], or empyema).

2. RLQ pain:
   a. Appendicitis: often begins with symptoms of dull, steady, periumbilical pain and anorexia before localizing to the RLQ at McBurney point.
   b. Regional enteritis.
   c. Leaking aneurysm.
   d. Ruptured ectopic pregnancy.
   e. Twisted ovarian cyst.
   f. Pelvic inflammatory disease (PID).
   g. Ureteral calculi.
   h. Incarcerated, strangulated inguinal hernia.
   i. Endometriosis.
   j. Meckel diverticulitis.
   k. Abdominal wall hematoma.

3. Left upper quadrant (LUQ) pain:
   a. Gastritis.
   b. Acute pancreatitis: epigastric pain that is relatively sudden, boring to the back, and associated with nausea, vomiting, and anorexia.
   c. Splenic enlargement, rupture, infarction, aneurysm.
   d. Myocardial ischemia.
   e. Left lower lobe pneumonia.
   f. Renal colic: radiates to the groin.

4. Left lower quadrant (LLQ) pain:
   a. Sigmoid diverticulitis.
   b. Regional enteritis.
   c. Leaking aneurysm.

AAA may present with a tender pulsatile mass at the abdominal midline. Vascular disorders such as acute arterial insufficiency, due to atherosclerosis or embolus, may present with severe abdominal pain in acute pathology or exacerbation, though may more commonly present with mild, constant pain for several days in a subacute or chronic presentation. Dissection or rupture of an AAA produces severe acute abdominal pain and often radiates to the back or genitalia.

   d. Ruptured ectopic pregnancy.

Rupture of the fallopian tube generally causes sudden, acute, and localized abdominal pain. Internal hemorrhage causes syncope and referred shoulder pain, caused by phrenic nerve irritation. Diagnosis before tubal rupture may be difficult because symptoms and physical findings mimic other conditions such as appendicitis.

   e. Incarcerated ovarian cyst.
   f. PID.
   g. Ureteral calculi.
   h. Incarcerated, strangulated inguinal hernia.

5. Generalized abdominal pain:
   a. Trauma:
      i. **Any person with a possible blow to the abdomen should have orthostatic BP taken, careful palpation of the abdomen, serial abdominal circumferences measured, and be considered for abdominal imaging. Serial hemoglobin (Hgb) and hematocrit (Hct) measurements should be obtained, if necessary.**
      ii. In abdominal trauma, the spleen is the most commonly injured organ, especially in blunt abdominal trauma; the onset can be immediate or delayed.
      iii. Nontraumatic splenic rupture is often associated with acute infectious mononucleosis.
   b. Intestinal obstruction: obstruction that develops slowly over weeks to months may be relatively subtle in presentation.

Acute obstruction presents with severe "colicky" pain or pain that is wavelike in nature; it makes the pain relentless.

   c. Peritoneal irritation: severe pain due to the rich innervation of the parietal peritoneum. Focal injury results in well-localized discomfort that is described as a sharp aching or burning. ▶

d. Metabolic disturbances may mimic intra-abdominal etiologies.

Porphyria and lead poisoning sometimes simulate bowel obstruction because they can cause cramping, abdominal pain, and hyperperistalsis.

e. Nonspecific dysfunctional abdominal pain and psychogenic abdominal pain are diagnoses of exclusion.

PLAN

A. General interventions:

1. If necessary, prepare the client for emergency transport and hospitalization.

2. Management and follow-up of other causes of abdominal pain are variable and depend on diagnosis.

B. Client teaching: *See Client Teaching Guide for this chapter, "Abdominal Pain":*

1. Counsel the client with chronic or recurrent abdominal pain to keep a pain diary that includes activity, foods, and other pain triggers; duration of pain; and what provides relief of symptoms.

C. Pharmaceutical therapy:

1. Treatment depends on the findings from the history, physical, and testing, as well as the clinical diagnosis.

FOLLOW-UP

A. Variable, depending on diagnosis.

B. Review pain diary.

CONSULTATION/REFERRAL

A. Consult the physician or surgeon for the client with acute abdominal pain and pain related to abdominal trauma.

B. For the obstetric client, consult a physician for any bleeding or abdominal pain.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. There is no evidence that acute intra-abdominal surgical emergencies are more common during pregnancy if ectopic pregnancy is excluded:

2. The presence of peritoneal signs, rebound tenderness, and abdominal guarding is never normal in pregnancy.

3. Bleeding complications include the following:

a. First trimester: miscarriage and ectopic pregnancy.

b. Second and third trimester: abruptio placentae.

4. Physiologic changes of pregnancy may affect the presentation and evaluation of abdominal pain. The enlargement of the uterus can impede physical examination, affect the normal location of pelvic and abdominal organs, and mask or delay peritoneal signs. (Refer to section "Appendicitis" in this chapter.)

5. Severe preeclampsia: The clinical manifestations of liver involvement include RUQ or midepigastric pain, elevated transaminases, and, in severe cases, subcapsular hemorrhage or hepatic rupture.

B. Adults:

1. Obesity distorts the abdominal exam, making organ palpation or pelvic examination difficult.

2. With males older than age 40 and females older than age 50, suspect cardiac origin when presenting with epigastric pain. Consider obtaining EKG for this age group.

Although myocardial infarction (MI) "classically" presents with anterior chest pressure or pain, the client may also have a gastritis or heartburn sensation, coupled with nausea and diaphoresis.

C. Geriatrics:

1. Delirium is a common early sign of acute illness or infection in older adults. It is more common in clients with existing cognitive impairment. Intra-abdominal pathology should be considered for clients presenting with altered mental status. Additional information regarding the evaluation of altered mental status and delirium are available in Chapter 23, "Neurologic Guidelines".

2. Older adults may have a vague or atypical presentation of pain, including fatigue and chest pain, varying in location, severity, and presence of a fever or nonspecific findings on examination:

a. Classic findings of acute peritonitis, rebound tenderness, and local rigidity occur less in older adults.

b. For dementia or noncommunicative clients, the American Medical Directors Association has endorsed the Pain Assessment in Advanced Dementia (PAINAD) Scale, which scores the following items:

i. Breathing: Examples include normal, labored, hyperventilation.

ii. Negative vocalization: Examples include moaning, negative language, crying.

iii. Facial expression: Examples include smiling, frowning, grimacing.

iv. Body language: Examples include relaxed, tense, rigid, or striking out.

v. Consolability: Examples include extent to which a caregiver is able to console, distract, or reassure.

3. In an older client, a similar presentation to IBD with abdominal pain and a change in bowel habits can be the first sign of colon cancer.

4. AAA is observed almost exclusively in older adults. Risk increases with each decade after 65:

a. Approximately 5% to 10% of males 65 years and older have an AAA.

b. Maintain a high index of suspension in clients who present with a clinical picture suggestive of renal colic or musculoskeletal back pain.

5. Older adults with urinary tract infection (UTI) are less likely to have dysuria, frequency, or urgency.

6. Clients older than 65 years have a 30% to 50% risk of gallstones and may not present with significant pain; less than half have fever, vomiting, or leukocytosis.

7. Fever and an elevated white blood cell (WBC) count occur in less than half of older adults with diverticulitis. Only about 25% with diverticulitis present with a guaiac positive stool.

8. The incidence of PUD is more common in older adults due to the availability and use of NSAIDs. The most common presenting symptom with PUD in older adults is melena.

9. Although the older adults may state they are having loose liquid stool, evaluate for fecal impaction. Diarrhea rather than constipation is sometimes the only complaint due to the collection of liquid stool distending the proximal colon and passing around the obstructing stool.

10. MI and pneumonia also commonly present as vague abdominal complaints in the older adults.

11. Weight loss/appetite suppression: Weight loss is a predictor of mortality. Clinically significant weight loss is considered to be 5% loss of usual body weight in 3 months or 10% in 6 months. Dehydration is more common in the older adults. Signs of dehydration include confusion, ▶

muscle weakness, fever, dizziness, poor skin turgor, hypotension, and tachycardia:

a. Monitor weight patterns/body mass index (BMI). (Caution: BMI is not used to identify loss of muscle mass.)

b. Monitor both serum albumin and anemia: Hypoalbuminemia and anemia are not necessarily associated with low bodyweight. They can be caused by decreases in liver protein synthesis:

i. Serum albumin concentration is commonly recommended.

ii. Hgb, prealbumin, and transferrin.

iii. Cholesterol.

12. Depression: Anxiety and depression are common in IBD and are factors that lead to decreased health-related quality of life.

13. Due to polypharmacy, a full review of medications, herbals, and over-the-counter (OTC) drugs should be undertaken to identify medications that should be avoided in the geriatric population, duplicate medications, and medications that are able to be decreased or deprescribed.

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APPENDICITIS

DEFINITION

A. Appendicitis is acute inflammation of the appendix caused by obstruction of the appendiceal lumen. There is no single sign, symptom, or diagnostic test that accurately confirms the diagnosis of inflammation. Perforation is rare in the first 12 hours, but the rate of perforation increases after 72 hours. Prompt, early diagnosis and operative intervention is the goal of treatment. The differential diagnoses for appendicitis include all abdominal sources of pain.

INCIDENCE

A. Acute appendicitis occurs at a rate of 29:100,000. It is most common between the ages of 10 and 30, with a slight male predominance. It is the most common condition in children (1%–8%) and during pregnancy (0.06%–0.1%) that requires emergency abdominal surgery. One in every 2,000 adults older than age 65 will develop appendicitis.

PATHOGENESIS

A. Foreign bodies, fecal material, tissue hypertrophy, strictures, or a bend or twist on the organ may cause obstruction of the appendix. The obstruction causes colicky pain. Bacterial invasion causes inflammation and leads to gangrene and perforation. The most common bacteria are *Escherichia coli*, *Pseudomonas*, *Bacteroides fragilis*, and *Peptostreptococcus* species.

PREDISPOSING FACTORS

A. Pregnancy.
B. Torsion.
C. Abdominal trauma.
D. Male, age 10 to 30.
E. Recent gastrointestinal (GI) infection.
F. Low-fiber diet.
G. Positive family history.

COMMON COMPLAINTS

The classic history of anorexia and periumbilical pain followed by nausea, right lower quadrant (RLQ) pain, and vomiting occurs in only 50% of cases.

A. Adults:

1. Generalized or localized abdominal pain in the epigastric or periumbilical areas. Within 2 to 12 hours, pain localizes in RLQ at McBurney point, and intensity increases.

2. Pain typically develops before vomiting.

3. Nausea and/or vomiting (may be projectile).

4. Anorexia signals organic cause of abdominal pain.

5. Nonspecific symptoms including indigestion, flatulence, change in bowel habits, and malaise.

6. The location of the appendix is altered in pregnancy.

7. The location of pain can vary according to the position of the appendix and the region that is inflamed.

B. Older adults are less likely to present with anorexia, RLQ pain, fever, or leukocytosis.

OTHER SIGNS AND SYMPTOMS

A. Rigid abdomen.
B. Changes in pulse (tachycardia), breathing (tachypnea), or skin temperature concerning for developing sepsis.
C. Involuntary guarding.
D. Rebound tenderness.

SUBJECTIVE DATA

A. Evaluate for a "surgical abdomen," defined as a rapidly worsening prognosis in the absence of surgical intervention. Clients should not eat or drink while a diagnosis of a surgical abdomen remains under consideration. Once a surgical abdomen has been excluded, the remainder of the evaluation will be guided by the chronicity of symptoms along with the location of pain:

B. Review onset, duration, course, and quality of pain. Has pain ever occurred before? If so, what was the primary diagnosis? What was the previous treatment and was it effective?

C. Qualify the duration of pain in minutes, hours, days, weeks, or months. Does it interfere with sleep? Is there a pattern to the pain?

D. Have the client rate the pain on a 10-point pain scale, with 0 being no pain and 10 being the worst pain the client has ever felt.

E. Review the pattern of pain:

1. Review aggravating factors.

2. Review alleviating factors.

3. Does the pain radiate?

4. Does the pain have any relationship to food?

F. Questions specific to females:

1. Determine the client's last menstrual period (LMP) to rule out pregnancy. ▶

2. What type of contraception is used? Specifically evaluate for an intrauterine device (IUD).

3. Has the client had a hysterectomy or tubal ligation?

4. Does the client have a recent history of dyspareunia or dysmenorrhea that suggests pelvic pathology?

G. Review current medications and drug history, especially antibiotic and laxative use. Clients taking corticosteroids may have a significant masking of pain.

H. Rule out abdominal trauma, motor vehicle accidents, falls, and assault.

I. Discuss bowel habits, including any changes, such as constipation or diarrhea, anorexia, food intolerance, nausea and vomiting, bloating.

J. Ask the client about urinary frequency, urgency, dysuria, flank pain, and back pain. In males, ask about hesitancy, difficulty starting the urine stream, nocturia, low urinary volume, or lower abdominal distension (urinary retention).

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure (BP), including orthostatic BP. Often, temperature is greater than 101°F.

B. Inspect:

1. Observe general appearance: facial expressions (grimace during exam), walk, skin color and turgor, level of consciousness, and acuity level of pain.

2. Inspect abdomen for surgical scars.

3. Observe for any bruising or other signs of domestic violence in the "bathing suit" area: breasts, abdomen, or back that would be easily covered by clothes.

C. Auscultate:

1. Auscultate the abdomen for presence of bowel sounds and bruits.

2. Auscultate heart and lungs.

D. Palpate: Note guarding with exam:

1. Palpate the abdomen:

a. Ask the client to bend their knees to help relax the abdominal wall musculature. Place arms at sides.

b. Note any rebound tenderness which indicates peritonitis. ***Perform at the end of the exam*** **because positive response produces pain and muscle spasm that can interfere with subsequent exam.**

2. Palpate back; note cerebrovascular accident (CVA) tenderness.

3. Check for obturator sign, or abdominal pain in response to passive internal rotation of right hip from 90° angle hip–knee flexion position. A positive sign indicates pain secondary to irritation of obturator muscle with inflamed appendix.

4. Assess psoas sign or increased abdominal pain occurring when the client attempts to raise their right thigh against the pressure of your hand placed over their right knee. Pain is caused by inflammation of the psoas muscle in acute appendicitis.

5. Check for the Apley rule; the farther from the navel the pain, the more likely it is organic in origin.

6. Check for Rovsing sign, or referred pain to the RLQ with palpation of the left lower quadrant (LLQ).

7. The location of the appendix and pain is altered during pregnancy (Figure 14.1).

E. Percuss:

1. Percuss abdomen.

F. Rectal exam:

1. Perform rectal exam. Although not demonstrated to show clear clinical benefit in cases of appendicitis, clients with a retrocecal appendicitis might experience more pain during a rectal examination.

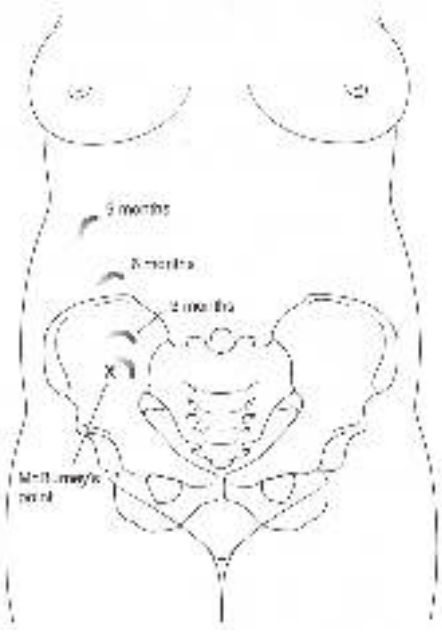


FIGURE 14.1 The position of the appendix alters during pregnancy, and so must the site of incision to gain access.

DIAGNOSTIC TESTS

A. Serum human chorionic gonadotropin (HCG): Pregnancy should be excluded in all individuals of childbearing age. Assume the client is pregnant until proven otherwise.

B. Complete blood count (CBC) with differential—80% of appendicitis cases present with leukocytosis.

C. C-reactive protein (CRP).

D. Urinalysis, to rule out urinary disorders.

E. Abdominal ultrasonography; any person with trauma to abdomen should have abdominal ultrasound. A finding of appendiceal diameter greater than 6 mm is the most suggestive ultrasound finding.

F. CT scan with contrast; significant findings include appendiceal diameter of greater than 6 mm, appendiceal wall enhancement or thickening of greater than 2 mm, periappendiceal fat stranding.

G. MRI may be used as an alternative diagnostic test in pregnancy to avoid exposure to ionizing radiation. Appendiceal diameter of greater than 7 mm is considered significant.

H. Guaiac stool for occult blood.

DIFFERENTIAL DIAGNOSES

A. The appendix has no fixed position. Duration of pain can help significantly in narrowing differential diagnosis. Nonspecific dysfunctional abdominal pain and psychogenic abdominal pain are diagnoses of exclusion:

1. GI:

a. Appendicitis.

b. Ruptured appendix: findings of white blood cell (WBC) greater than 15,000 and fever 103°F or greater. Twenty percent of perforations occur in less than 24 hours from onset and 65% occur in greater than 48 hours.

c. Cecal diverticulitis: presents similar to appendicitis. Right-sided diverticulitis occurs in 1.5% of individuals from Western countries but is more frequent in the Asian population.

d. Meckel diverticulitis: occurs in younger clients but presentation is similar to acute appendicitis. ▶

e. Regional enteritis or ileitis: associated with infection; presents with diarrhea.
f. Crohn's disease (CD)/inflammatory bowel disease (IBD): presents with more chronic symptoms along with fatigue.
g. Incarcerated groin hernia.
h. Abdominal wall hematoma.
i. Bowel obstruction.
j. Intestinal malrotation.
k. Intussusception.
l. Parasitic infection.
m. Constipation.
n. Leaking abdominal aneurysm.

2. Gynecologic:
a. Pelvic abscess: secondary to pelvic inflammatory disease (PID) and usually occurs with vaginal discharge.
b. PID: worse with intercourse and usually occurs with vaginal discharge.
c. Ruptured ovarian cyst: usually worse with exertion and can cause bleeding.
d. Mittelschmerz: timing related to ovulation.
e. Ovarian torsion.
f. Endometriosis.
g. Ectopic pregnancy.
h. Endometritis: associated with fever, vaginal discharge, and usually occurs after a procedure or delivery.
i. IUD.

3. Urologic:
a. Ureteral calculi/renal colic.
b. Testicular torsion.
c. Epididymitis.

PLAN

A. Client teaching: *See Client Teaching Guide for this chapter, "Abdominal Pain."*

B. Medical and surgical management:

1. Appendectomy is the current standard of care for management of appendicitis, and especially perforated appendicitis.

C. Pharmaceutical therapy:

1. Multiple studies have examined whether surgery can be delayed for nonperforated appendicitis. Several meta-analysis and systemic reviews have concluded that for a very small minority of clients with previous surgical complication or severe phobia, delayed appendectomy may be appropriate. Clients should be warned that as many as 50% of clients with delayed appendectomy may develop recurrent symptoms and require emergent surgery at a later time.

2. The use of antibiotics prior to surgery is warranted to decrease the rate of wound infection.

3. Most episodes of appendicitis fall into the category of mild-to-moderate community-acquired intra-abdominal infections without factors for resistance or treatment failure. Antibiotics chosen should cover streptococci, nonresistant Enterobacteriaceae, and anaerobes.

4. Do not give antipyretics to mask fever.

5. Do not administer cathartics because they may cause rupture.

FOLLOW-UP

A. Postoperative follow-up is with the surgeon.

CONSULTATION/REFERRAL

A. Physician consultation and possible emergency transport and hospitalization are often required.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Any abdominal pain or bleeding in the first 8 weeks after a missed menstrual period must be considered a symptom of possible *ectopic pregnancy*.

2. The identification of intra-abdominal masses may be compromised by the enlarged uterus, but this problem may be partially obviated by examining the client in the lateral position.

3. The intestinal tract is progressively displaced upward, outward, and backward during pregnancy; bowel sounds are best heard lateral or superior to the uterus.

B. Adults:

1. Obesity distorts abdominal exam, making organ palpation or pelvic exam difficult.

2. Immunocompromised clients are susceptive to infection. They may not exhibit the typical signs and symptoms of appendicitis; only mild tenderness on exam.

3. The CT exam is useful in the immunocompromised client for diagnosis, although inflammatory changes may be less prominent.

C. Geriatrics:

1. Older adults adults tend to have a diminished inflammatory response, resulting in a less remarkable history and physical examination. For this reason, they more frequently present with perforation. Be aware of vague symptoms, such as milder pain, less pronounced fever, and leukocytosis with shift to left on differential.

2. A redundant sigmoid colon may also cause right-sided pain from sigmoid disease.

3. Older clients have an increased risk of colonic malignancy as the cause for their RLQ pain. Consider CT **and** colonoscopy in clients older than 40 presenting with symptoms of appendicitis, especially if CT scan is negative for obvious etiology.

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BOWEL OBSTRUCTION

DEFINITION

A. Bowel obstruction occurs when the flow of intestinal contents is disrupted along the course of the large or small bowel. Although a more common occurrence in the small bowel, 25% of obstructions occur in the large bowel. The obstructions can occur in two ways:

1. Mechanical: can be partial or complete; occurring insidiously or acutely:
a. Mass/stricture.
b. Adhesions.
c. Volvulus.

d. Intussusception.
e. Congenital anomalies.
f. Fecal impaction.
g. Diverticular disease.

2. Functional: due to altered physiology of the intestines.

B. Intestinal pseudo-obstruction related to nerve and muscle disorders:

1. Parkinson disease.
2. Multiple sclerosis.
3. Hirschsprung disease.
4. Diabetes.
5. Medications.

C. Small bowel obstruction can be characterized as follows:

1. Partial or complete:
a. Simple.
b. Strangulated.

D. The absence of bowel activity, stool and flatus, is a predictor of complications, such as ischemia, necrosis, or perforation.

E. Metabolic disturbances may mimic intra-abdominal etiologies.

Porphyria and lead poisoning sometimes simulate bowel obstruction because they can cause cramping, abdominal pain, and hyperperistalsis.

INCIDENCE

A. In clients with a history of abdominal surgery, the postsurgical incidence is around 9%.

B. In clients without a history of abdominal surgery, the incidence is 3% to 9%.

C. The small bowel is implicated in 75% to 80% of all intestinal obstructions.

D. Average age 65 to 70 in both males and females.

1. Volvulus occurs in the sigmoid colon or cecum in greater than 95% of cases. Typically presents during ages 60 to 80 with other chronic medical and neuropsychologic conditions.

PATHOGENESIS

A. Obstruction can be caused by extrinsic (outside the bowel wall), intrinsic (in the bowel wall), or intraluminal factors. Blockages can be complete or incomplete.

B. With obstruction, dilation of the bowel wall occurs proximally, leading to edema, fluid accumulation, and loss of absorptive function. Emesis can create metabolic alkalosis due to loss of electrolytes. With intensifying distention, the vessels within the wall of the intestines become compromised, leading to reduced perfusion, resulting in ischemia or the tissue and possible necrosis and perforation.

C. Peritoneal irritation: Severe pain due to the rich innervation of the parietal peritoneum. Focal injury results in well-localized discomfort that is described as a sharp aching or burning.

PREDISPOSING FACTORS

A. Cancer:

1. Colorectal cancer (CRC; 30%).
2. Pancreatic.
3. Ovarian.
4. Prostatic neoplasm.

B. Previous radiation therapy.

C. Prior abdominal surgery.

D. Prior colorectal resection.

E. Inflammatory bowel disease (IBD; Crohn's or ulcerative colitis).

F. Abdominal wall hernias.

G. Structural abnormality:

1. Volvulus.
2. Fecal impaction.

H. Rectal stenosis.

I. Endometriosis.

J. Tuberculosis.

K. Lymphogranuloma venereum.

L. Medications:

1. Opioids.
2. Tricyclic antidepressants.

COMMON COMPLAINTS

Acute obstruction presents with severe paroxysmal "colicky" pain or pain that is wavelike in nature; it makes the pain relentless. Systemic signs—fever, tachycardia, hypotension, altered mental state—may be associated with strangulation.

CARDINAL SYMPTOMS

A. Abdominal pain.

B. Distention.

C. Severe and obstinate constipation.

OTHER SIGNS AND SYMPTOMS

A. Nausea and vomiting (may be bile-colored emesis in small bowel obstruction).

B. Focal abdominal pain can indicate peritoneal irritation caused by ischemia or necrosis.

C. Pain occurring in the lower pelvis can be due to rectal tenesmus, indicating rectal obstruction.

D. Pencil thin bowel movements (BMs).

E. Obstructions may develop slowly over weeks to months; may be relatively subtle in presentation.

SUBJECTIVE DATA

A. The history should focus on the identification of risk factors:

1. Review the location, onset, location, onset, characteristics, associated factors, timing, exacerbating factors, and severity of symptoms.
2. Is the client able to pass stool and flatus?
3. Is the client able to keep liquids down?
a. When was the last fluid intake?
b. Is the client urinating?
4. Does the client have any of the following as noted in "Predisposing Factors"?
a. Cancer(s), previous radiation therapy.
b. Does the client have a diagnosis of IBD?
c. Has the client had recent surgeries or a colon resection?
d. Has the client had gastroenteritis? Is any other family member ill?
e. Has the client ever had a colonoscopy and been diagnosed by diverticular disease?
5. Review all medications, over-the-counter (OTC), and herbal products. Does the client have a history of chronic opioid use?

PHYSICAL EXAMINATION

A. Check temperature, blood pressure (BP) sitting and standing, pulse, respirations, height, and weight to calculate a body ▶

mass index (BMI). (Dehydration is manifested by tachycardia and orthostatic hypotension.)

B. Inspect:

1. Clients with complications from obstruction appear acutely ill.

2. Check mucous membranes for signs of severe dehydration.

3. Evaluate presence of abdominal distention.

4. Surgical scars indicating past abdominal surgeries.

5. Hernias: incisional or groin.

C. Auscultate:

1. Abdomen all four quadrants: High-pitched bowel sounds indicate acute obstruction followed by muffling of the bowel sounds as distention progresses; finally, the bowel sounds become hypoactive. Evaluate for ascites.

2. Heart.

3. Lungs.

D. Palpate:

1. Abdomen: Identify mass (abscess, volvulus, tumor) or hernia.

2. Check for hepatosplenomegaly.

3. Evaluate rebound tenderness.

E. Percussion:

1. Abdomen:

a. Tympany of the abdomen due to distention; tympany over the liver suggests free intra-abdominal air.

b. Dullness of the abdomen over fluid-filled dilated loops.

c. Tenderness to percussion can indicated peritonitis.

F. Rectal examination:

1. Rectal examination identifies rectal mass; check for occult blood.

G. Neurologic exam.

DIAGNOSTIC TESTS

A. Laboratory studies:

1. Complete blood count (CBC) with differential.

2. Metabolic profile.

3. Blood urea nitrogen (BUN)/creatinine levels (may be elevated in dehydration due to vomiting).

4. Urinalysis.

5. Guaiac any stool from the rectal examination.

B. Imaging:

1. Abdominal x-ray. (Relevant findings include dilated loops of bowel or a size difference between the proximal and distal bowel with the proximal being dilated and the distal appearing collapsed.)

2. Chest radiograph: used to assess for aspiration in clients who have been vomiting.

3. Ultrasound.

4. Endoscopy.

5. CT:

a. Significant findings include increased thickening of bowel wall greater than 3 mm, mesenteric edema, abdominal ascites, and submucosal edema or hemorrhage.

b. Target sign, whirl sign, and venous cutoff sign are all radiologic findings suggestive of specific etiology.

6. Magnetic resonance (MR) enterography is useful with low-grade bowel obstruction is suspected.

DIFFERENTIAL DIAGNOSES

A. Bowel obstruction:

1. Small bowel obstruction: The most common etiologies are adhesions, hernias, and malignancy.

2. Large bowel obstruction: The most common etiologies are tumor, adhesions, and volvulus.

B. Nonobstructive motility disorder:

1. Paralytic ileus.

2. Pseudo-obstruction.

C. Constipation.

D. Fecal impaction.

E. Porphyria.

F. Lead poisoning.

PLAN

A. General interventions:

1. Treatment depends on the location and severity of the obstruction.

2. Clients with suspected bowel obstruction should be kept NPO to avoid gastric distention. Hospitalized clients may benefit from nasogastric (NG) tube placement with low-intermittent wall suction.

3. The crampy abdominal pain of bowel obstruction does not generally respond to pharmacologic therapy. Use of opioids is discouraged except among clients with cancer.

4. There is a subset of clients with partial bowel obstruction who may be managed with medical observation for 12 to 24 hours by a specialist or surgeon.

B. Pharmaceutical therapy:

1. Treatment depends on the findings from the history, physical, and testing, as well as the clinical diagnosis.

FOLLOW-UP

A. Variable, depending on diagnosis.

EMERGENT ISSUES/INSTRUCTIONS

A. All clients identified as having a complete bowel obstruction should be admitted to the hospital for consideration of surgical exploration.

B. Without treatment, abdominal pain can increase as a result of bowel perforation and ischemia.

CONSULTATION/REFERRAL

A. Surgical consult.

B. Depending on the urgency, consult with a gastroenterologist.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Signs of dehydration include confusion, muscle weakness, fever, dizziness, poor skin turgor, hypotension, and tachycardia.

2. Delirium is a common early sign of acute illness or infection in the older adults. It is more common in clients with existing cognitive impairment. At a minimum, clients and family should be asked if the client has experienced episodes of altered mental status:

3. Older adults have a diminished sensorium, allowing pathology to advance to a dangerous point prior to symptom development:

a. The level of pain is much less severe at presentation and continues to be at a lower level of pain.

B. Although older adults may state they are having loose liquid stool, evaluate for fecal impaction. Diarrhea rather than constipation is sometimes the only complaint due to the collection of liquid stool distending the proximal colon and passing around the obstructing bolus.

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CELIAC DISEASE

DEFINITION

A. Celiac disease, previously known as celiac sprue, is an immune-mediated disorder triggered by ingestion of gluten, a storage protein contained in three grains: wheat, barley, and rye. Clients with celiac disease are unable to tolerate gliadin, the alcohol soluble fraction of gluten. The disease primarily affects the small intestine in those genetically susceptible. Onset of symptoms depends on the amount of gluten in the diet. Dietary nonadherence is the chief cause of persistent or recurrent symptoms. (See Appendix B, "Gluten-Free Diet.")

B. Celiac disease is a common cause of chronic malabsorption due to injury to the small intestine resulting in loss of absorptive surface area and decrease in digestive enzymes, leading to impaired absorption of fat-soluble vitamins, iron, and potentially vitamin B_{12} and folic acid.

C. A substantial number of clients are misdiagnosed as having irritable bowel syndrome (IBS) for years prior to the diagnosis of celiac disease.

D. The complications from celiac disease include osteopenia, osteoporosis, infertility, short stature, delayed puberty, anemia, and gastrointestinal (GI) malignancies and non-Hodgkin lymphoma (NHL).

INCIDENCE

A. Celiac disease can occur at any stage of life. Its prevalence is approximately 1% of the general population in North America. The highest incidence (5%) is noted in the sub-Saharan African population. The true incidence is undetermined due to asymptomatic disease and underdiagnosis.

B. Newly diagnosed clients with celiac disease should inform their first-degree family members of their increased risk of celiac disease and the American College of Gastroenterology (ACG) recommendation for screening.

C. Celiac disease is strongly associated with autoimmune conditions, including type 1 diabetes, Addison disease, and thyroiditis, as well as genetic syndromes, including Down syndrome, Williams syndrome, and Turner syndrome.

D. Liver disease and Crohn's disease (CD) share widespread risk factors. Liver disorders such as autoimmune hepatitis, elevation of liver enzyme levels, primary biliary cirrhosis, nonspecific hepatitis, primary sclerosing cholangitis (PSC), and nonalcoholic fatty liver disease (NAFLD) have been reported in clients with CD.

PATHOGENESIS

A. Both immune and genetic factors play a role in the intestinal reaction to gluten ingestion. After the gluten fragment is ingested and absorbed, broken into fragments by tissue transglutaminase, an immune complex is formed in genetically prone individuals. The formation of the complex then stimulates both cellular and humoral immunity, leading to production of autoantibodies, which attack enterocytes causing atrophy, and inflammatory cytokines, which cause mucosal inflammation and villous atrophy. Celiac disease primarily affects the mucosal layer of the small intestine. The classic celiac lesion is noted in the proximal small intestine.

B. Hallmark histologic findings include presence of intraepithelial lymphocytes in the jejunum, duodenum, or both; crypt hyperplasia; villous atrophy.

C. The Marsh Classification is used to describe the progressive histologic stages of celiac disease. Note: Marsh 1 and Marsh 2 may be seen in sow's and cow's milk allergies.

1. Marsh 0: preinfiltrative stage (normal).
2. Marsh 1: infiltrative lesion (increased intraepithelial lymphocytes).
3. Marsh 2: hyperplastic lesion (type 1 plus hyperplastic crypts).
4. Marsh 3: destructive lesion (type 2 plus villous atrophy of progressively more severe degrees [termed 3a, 3b, and 3c]).
5. Marsh 4 (atrophic–hypoplastic): total villous atrophy, crypt hypoplasia.

PREDISPOSING FACTORS

A. Female.

B. Infectious diarrheal episode or other intestinal disease (e.g., rotavirus).

C. Genetic disorders:

1. Down syndrome (8%–12%).
2. Type 1 diabetes (10%).
3. Turner syndrome (2%–10%).
4. Williams syndrome (8.2%).

D. Strong hereditary component (10% in first-degree relatives; 2% in second-degree relatives).

E. The introduction of gluten before 4 months of age is associated with an increased development of disease.

F. Autoimmune thyroid disorders (Hashimoto thyroiditis and Graves disease).

G. Selective immunoglobulin A (IgA) deficiency.

COMMON COMPLAINTS

A. Asymptomatic.
B. Chronic diarrhea or explosive watery diarrhea.
C. Foul-smelling voluminous stools.
D. Anorexia.
E. Abdominal distention/bloating/flatulence.
F. Abdominal pain.
G. Poor weight gain or weight loss.
H. Vomiting.
I. Steatorrhea (malabsorption of ingested fat).

OTHER SYMPTOMS

A. Behavioral changes, including irritability.
B. Dehydration.
C. Lethargy.
D. Constipation.
E. Failure to thrive (FTT).
F. Short stature and delayed puberty.
G. Dermatitis herpetiformis/skin disorders.
H. Arthritis.
I. Seizures.
J. Weakness and fatigue.
K. Dental enamel hypoplasia of permanent teeth.
L. Iron deficiency anemia unresponsive to treatment.
M. Bruising/bleeding tendency.
N. Osteopenia/osteoporosis.
O. Hair loss.
P. Lactose intolerance.
Q. Aphthous stomatitis.

SUBJECTIVE DATA

A. Review the onset, duration, course, type of symptoms, and aggravating or alleviating factors.
B. Review the client's weight history.
C. Evaluate family history for celiac disease or members with similar histories.
D. Review current medications and drug history, especially antibiotic, laxative, and herbal products.
E. Review bowel habits and note changes: constipation, diarrhea, anorexia, and/or food intolerance.
F. Review the client's tolerance to lactose products.

PHYSICAL EXAMINATION

A. Check vital signs, including height and weight. Follow serial changes in body mass index (BMI).
B. Inspect:
  1. Observe general overall appearance.
  2. Oral examination to evaluate glossitis, dry mucosal membranes (dehydration), and the presence of oral aphthae.
  3. Examine the skin for the presence of dermatitis herpetiformis, a blistering rash involving the scalp, neck, elbows, knees, and buttocks.
  4. Evaluate the abdomen for the presence of bloating and protuberant "potbelly."
  5. Evaluate the client's weight loss, including muscle wasting.
C. Auscultate:
  1. Auscultate for bowel sounds.
D. Percuss:
  1. Percuss abdomen.
E. Palpate:
  1. Palpate the abdomen for masses, rebound tenderness, and peritoneal signs:
    a. Before palpating the abdomen, ask the client to bend their knees and place hands at sides to help with relaxation of the wall musculature.
F. Rectal examination:
  1. Perform a rectal examination, including testing stool for occult blood.

DIAGNOSTIC TESTS

A. Screening of the general population for celiac disease in asymptomatic persons is not recommended by the U.S. Preventive Services Task Force. However, screening of high-risk populations such as those individuals with a positive family history is recommended.
B. The confirmation of a diagnosis of celiac disease is based on the combination of findings from the medical history, physical examination, serology, and upper endoscopy, with histologic analysis of multiple biopsies of the duodenum. **All testing should be performed while clients are following a gluten-rich diet.**
C. Endoscopy for duodenal biopsies is the standard and a critical component for diagnosing celiac disease.
D. Screening and monitoring test for celiac disease. See Table 14.1 for celiac disease tests and possible results:
  1. Tissue transglutaminase antibody (tTG), IgA class: 95% sensitive, 95% specific:
    a. Primary test ordered to screen for celiac disease.
    b. Used for monitoring and evaluating the effectiveness of treatment.
    c. Antibody levels should fall when gluten is removed from the diet.
  2. Total IgA: IgA deficiency is more common in clients with celiac disease.
  3. Anti-tTG antibodies, immunoglobulin G (IgG).
  4. Deamidated gliadin peptide antibodies (anti-DGP, IgA).
  5. Antigliadin antibodies (AGA) IgA.
  6. IgA endomysial antibody (EMA): less frequently ordered, measures same as the anti-tTG.
  7. Antireticulin antibody (ARA): rarely ordered.
  8. Anti-F-actin: ordered if the disease has been diagnosed; evaluates the severity of intestinal damage. May be used for monitoring.
E. Complete blood count (CBC) and electrolytes.
F. Aspartate transaminase (AST) and alanine transaminase (ALT): Liver enzymes normalize on a gluten-free diet.
G. Prothrombin time (PT) may be prolonged with malabsorption of vitamin K.
H. C-reactive protein (CRP).
I. Erythrocyte sedimentation rate (ESR).
J. Total protein.
K. Albumin.
L. Calcium.
M. Iron, transferrin, and ferritin.
N. Vitamin B_{12}, folate levels, and vitamin D. ▶

TABLE 14.1 WHAT DOES THE TEST RESULT MEAN? SOME CELIAC DISEASE TESTS AND RESULTS

Anti-tTG Antibodies, IgA	Total IgA	Anti-tTG Antibodies, IgG	Anti-DGP, IgA	AGAs-IgG	Diagnosis
Positive	Normal				Presumptive celiac disease
Negative	Normal	Negative	Negative	Negative	Symptoms not likely due to celiac disease
Negative	Low	Positive	Negative	Positive	Possible celiac disease (false-negative anti-tTG, IgA, and anti-DGP are due to a total IgA deficiency)
Negative	Normal	Negative	Positive	Positive/negative	Possible celiac disease (may be seen in children younger than 3 years old)

AGAs, antigliadin antibodies; DGP, deamidated gliadin peptide; IgA, immunoglobulin A; IgG, immunoglobulin G; tTG, tissue transglutaminase.

Source: Reprinted with permission from American Association for Clinical Chemistry. (2017, December 4). Celiac disease antibody tests. *Lab Tests Online*. https://labtestsonline.org/tests/celiac-disease-antibody-tests.

O. Stool culture, ova, and parasites.
P. Fecal fat.
Q. Bone density (bone mineral density improves on gluten-free diet).
R. Human leukocyte antigen (HLA) haplotypes.
S. Colonoscopy if bloody stools or symptoms of colitis.
T. Sweat test to exclude cystic fibrosis (CF).
U. Radiograph, including barium swallow study with a small bowel follow-through, is usually nonspecific and is not indicated.

DIFFERENTIAL DIAGNOSES

A. Celiac disease.
B. Microscopic colitis.
C. Food allergies.
D. Inflammatory bowel disease (IBD):
  1. CD.
  2. Ulcerative colitis (UC).
E. Immunodeficiency disorders.
F. Gastroenteritis (viral or bacterial).
G. Parasites.
H. Fungal infection.
I. IBS.
J. Malabsorption.
K. Lymphomas of the small intestine.
L. Pancreatic exocrine dysfunction.

PLAN

A. General interventions:
  1. Lifelong adherence to a gluten-free diet is the only accepted treatment for celiac disease:
    a. Gluten-free dietary instructions should be given and reinforced.
    b. Reinforce the need to read all food labels.
    c. Stress that wheat-free is not the same as gluten-free.
  2. The ACG recommends a referral to a registered dietitian to receive a thorough nutritional assessment and education on a gluten-free diet. A gluten-free diet should be maintained for life. Numerous apps are available for download to smartphones to aid the client in dietary selections.
  3. Monitor for nutritional deficiencies including iron, B vitamins, and vitamin D. Substitute flours are not fortified with B vitamins. Supplement with iron, folate, and vitamin B_{12} as needed.
  4. Screen for osteoporosis.
  5. Keep a food/symptom diary to eliminate trigger foods. See Appendix B, "Gluten-Free Diet."
  6. Gluten has been identified in dietary supplements, over-the-counter (OTC) medications, nonfood items such as lipstick and envelope adhesive, and food items with gluten additives such as condiments. Gluten is also found in the fillers of pill capsules.
  7. Gluten rechallenge is not generally recommended unless the diagnosis remains uncertain.
  8. HLA-DQ2/DQ8 genotyping for genetic risk factors should not be used for routing screening or testing.
B. Pharmacology:
  1. Vitamin and calcium supplements.
  2. Bisphosphonates for osteoporosis.
  3. Corticosteroids may be prescribed for rapid control of symptoms.

FOLLOW-UP

A. After the diagnosis of celiac disease and implementation of a strict gluten-free diet, follow up in 4 to 8 weeks or earlier if the client has other comorbidities.
B. The Celiac Disease Guideline Committee recommends repeating the measurement of tTG after 6 months of a gluten-free diet.
C. Newly diagnosed clients with celiac disease should undergo testing and treatment for micronutrient deficiencies. Deficiency testing should include, but not be limited to, iron, folic acid, vitamin D, and vitamin B_{12}.
D. Refer for dietary consultation with a nutritionist with experience in gluten-free diets.
E. Consider allergy testing.

CONSULTATION/REFERRAL

A. If the gluten-free diet fails or the client experiences new symptoms, a systematic evaluation is required to elucidate other sources of villous inflammation.
B. Endocrine consultation for clients with Hashimoto thyroiditis and celiac disease.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
  1. Clients with untreated celiac disease are at risk for preterm birth, low birthweight babies, recurrent loss, and reduced fertility.
B. Geriatrics:
  1. Older adults may present with nonspecific GI symptoms, abdominal discomfort, bloating, constipation, or dyspepsia.
  2. Diarrhea may be mild or intermittent in older adults.
  3. Celiac disease is the most common cause of steatorrhea in people older than 50 years of age and the second most common cause in people older than 65 years.
  4. Iron-deficiency anemia may also be a presenting symptom. Anemia is present in 60% to 80% of older adults with celiac disease.
  5. Autoimmune thyroid disorders are common with older adults with celiac. The majority present with hypothyroidism.
  6. The risk for NHL is increased in people with celiac disease age 50 years and older.
  7. Due to polypharmacy a full review of medications, herbals, and OTC drugs should be undertaken to identify medications that should be avoided in the geriatric population, duplicate medications, and medications that are able to be decreased or deprescribed. Refer to section on deprescribing in Chapter 3, "Adult-Geriatric Assessments".
  8. For older adults with existing celiac disease, nurse practitioner (NPs) should practice special consideration for the following geriatric syndromes:
    a. Falls:
      i. Ataxia and neuropathy are two main neurologic complications of celiac disease, placing older adults at a higher risk for falls.
      ii. Older adults may rush to get to their toilet and pull their garments down with episodes of diarrhea, thereby placing themselves at increased risk for falls.
      iii. Up to 70% of adults and older adults with celiac disease have osteopenia and therefore are at increased risk for fractures.
    b. Weight loss/appetite suppression:
      i. Because of GI distress, abdominal discomfort, and diarrhea, older adults are at increased risk of avoiding eating.
      ii. Monitor weight patterns/BMI.

RESOURCES

Celiac Disease Foundation: www.celiac.org
Gluten-free Recipes: www.celiac.com
Gluten-free Drugs: www.glutenfreedrugs.com

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CHOLECYSTITIS

DEFINITION

A. Cholecystitis is the acute or chronic inflammation of the gallbladder. Acute cholecystitis is associated with stone formation (cholelithiasis) in 90% of all cases, causing obstruction and inflammation. Biliary sludge is a feature of chronic cholecystitis.

B. Following a cholecystectomy, stones may recur in the bile duct.

INCIDENCE

A. Gallbladder disease afflicts more than 20 million people (10%–20% of adults) in the United States. Most clients have asymptomatic gallbladder disease; however, 20% progress to experiencing acute biliary symptoms. Once acute symptoms develop, there is a 30% chance of persistent recurrences, and therefore acute cholecystectomy is the recommended treatment for symptomatic clients. Mortality related to acute cholecystitis is 5% to 10%, with the highest risk for clients older than age 60. The most common complication of acute cholecystitis is the development of gallbladder gangrene (20%), with the potential for subsequent perforation (2%). Gangrenous cholecystitis is most common in older clients, diabetics, or clients who delay treatment.

PATHOGENESIS

A. Cholecystitis occurs subsequent to bile stasis, bacterial infection, ischemia, or cystic obstruction by a gallstone. Acute cholecystitis is related to the impaction of a calculus in the neck of the gallbladder in approximately 90% of cases. Spontaneous resolution may occur after the reestablishment of cystic duct patency.

B. *Escherichia coli* is the primary microorganism in 80% of cholecystitis infections. Other pathogens include *Enterobacter, Enterococcus*, and *Klebsiella*. Positive culture rates range from 22% to 46%.

C. Estrogen-induced alteration in bile salts may favor stone formation. Stones occur when cholesterol supersaturates the bile in the gallbladder and precipitates out of the bile. Cholesterol stones are the most common type of gallstones in the United States.

D. Pigment stones occur when free bilirubin combines with calcium. Pigment stones are found in clients with cirrhosis, hemolysis, and infections in the biliary tree.

E. Acalculous cholecystitis is associated with infection and local inflammation. Formation of gallstones is not necessary for the obstruction of the bile duct.

PREDISPOSING FACTORS

A. Female.

B. Sudden starvation/prolonged fasting.

C. Medications:

1. Cholesterol-lowering drugs: Stone formation increases in users of cholesterol-lowering drugs, which are known to increase biliary cholesterol saturation.

2. Thiazide diuretics.

3. Ceftriaxone.

4. Cyclosporine.

5. Opiate narcotic analgesics.

6. Estrogen usage (oral contraceptives [OCPs] and hormone therapy [HT]): Stone formation increases in users of contraceptives and estrogens, which are known to increase biliary cholesterol saturation.

7. Octreotide.

D. Bile acid malabsorption.

E. Genetic predisposition: Native Americans and those of Chinese or Japanese descent have a high incidence.

F. Total parenteral nutrition (TPN).

G. Obesity.

H. Status postbariatric surgery. (Clients who have had either the Roux-en-Y or laparoscopic banding surgeries are at increased risk for gallstones.)

I. Pregnancy secondary to elevated progesterone.

J. Increasing age.

K. Hemolytic anemia.

L. Diabetes.

M. Hepatic cirrhosis.

N. Biliary tree infections.

COMMON COMPLAINTS

A. Abrupt, severe abdominal pain lasting greater than 6 hours.

B. Constant aching pain or gnawing pain in the right upper quadrant (RUQ), right subcostal region, with radiation to the back and right shoulder.

C. Nausea and vomiting.

OTHER SIGNS AND SYMPTOMS

A. Anorexia.

B. Heartburn.

C. Upper abdominal fullness.

D. Biliary colic: sudden onset of severe pain in the epigastrium or right hypochondrium that subsides relatively slowly. Tenderness may remain for days.

E. Fat intolerance.

F. Fever (low-grade).

G. Mild jaundice (20%).

H. Complicated disease such as an abscess or perforation; symptoms includes more severe localized persistent pain, tenderness, fever, chills, and leukocytosis.

I. Chronic diarrhea (4–10 bowel movements [BMs] every day for at least 3 months).

SUBJECTIVE DATA

A. Review the onset, location, duration, course, and quality of pain.

B. Use a pain rating scale, such as a 10-point pain scale, with 0 being no pain and 10 being equivalent to the worst pain the client has ever felt. Determine progression of the pain as well.

C. Review any alleviating factors, such as antacids, and any worsening factors, such as deep inspiration.

D. Review any pain radiating to the jaw, neck, shoulder, or arm.

E. Review onset of pain in relation to last meal and foods ingested.

F. Ask the client about recurrent history of epigastric pain. ▶

G. Obtain history and demographic data that may indicate the risk factors for biliary disease.
H. Review the date of the client's last menstrual period (LMP), and if they are pregnant establish gestational age.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure (BP). Tachycardia, increased fever, and elevated white blood cell (WBC) are markers of inflammatory disease.
B. Inspect:
1. Observe general appearance, walking, and facial expressions such as grimacing during the examination. Overall appearance is generally unremarkable between attacks; ill appearance occurs during acute attack.
2. Observe skin turgor and color (15% have jaundice).
3. Clients generally remain still during exam due to peritoneal inflammation, which aggravates pain with movement.
C. Auscultate:
1. Heart.
2. Lung fields.
3. Abdomen for bowel sounds.
D. Percuss:
1. Percuss abdomen.
E. Palpate:
1. Palpate the abdomen; check for tenderness in the RUQ, especially with inspiration; assess for guarding and rebound tenderness.
2. Check Murphy sign. Positive Murphy sign is inspiratory arrest secondary to extreme tenderness when subhepatic area is palpated during deep inspiration.
F. Rectal exam, if indicated.

DIAGNOSTIC TESTS

A. Laboratory tests:
1. Complete blood count (CBC) with differential.
2. Amylase.
3. Alkaline phosphate.
4. Bilirubin.
5. Aspartate transaminase (AST).
6. Alanine transaminase (ALT).
7. Urinalysis to rule out pyelonephritis and renal calculi.
8. Pregnancy test if childbearing age.
9. Stool for occult blood to rule out bleeding.
B. Radiography:
1. Ultrasonography: study of choice and can often establish the diagnosis. Assess for presence of gallstones, gallbladder wall thickening (>4–6 mm), and sonographic Murphy sign.
2. Cholescintigraphy (hepatobiliary iminodiacetic acid [HIDA]) scan is indicated if the diagnosis is uncertain after an ultrasound.
3. Magnetic resonance (MR) cholangiography: sensitive for stone in cystic duct.
4. CT scan.
5. Endoscopic retrograde cholangiopancreatography (ERCP) and percutaneous transhepatic cholangiography (PTC).
6. Chest radiography to rule out pneumonia.
C. EKG to rule out myocardial infarction (MI).

DIFFERENTIAL DIAGNOSES

A. Acute cholecystitis.
B. Biliary colic: caused by contraction of the gallbladder after a fatty meal pressing a stone toward the orifice of the cystic duct. Distinguishing features of biliary colic include pain that peaks after several hours and then totally resolves (<4–6 hours). No associated fever, nausea, vomiting, or leukocytosis. Usually clients with acute cholecystitis have experienced episodes of biliary colic in the past, which can complicate diagnosis.
C. Acute pancreatitis.
D. Appendicitis.
E. Peptic ulcer disease (PUD)/perforation.
F. Acute hepatitis.
G. Pneumonia or pleurisy.
H. MI.
I. Renal calculi or other pathology associated with the right kidney.
J. Gastroesophageal reflux disease (GERD).
K. Pregnancy: urolithiasis, pyelonephritis.
L. Functional bowel disorders including nonulcerative dyspepsia, irritable bowel syndrome (IBS): pain predominant, functional gallbladder disorder.
M. Sphincter of Oddi dysfunction.
N. Intra-abdominal abscess.
O. Fitz-Hugh–Curtis syndrome: perihepatitis caused by gonococcal infection.

PLAN

A. General interventions:
1. Clients with a single episode of biliary colic (pain <4–6 hours with full resolution) are reasonable candidates for expectant management as long as they continue to be free of recurrent pain.
B. Client teaching:
1. No activity restriction is required.
2. Treatment depends on acuteness, severity, and frequency of attack(s). Heat may be used as needed for pain. If pain continues to worsen, have the client contact their healthcare provider. Hospitalization and/or surgery may be required depending on the severity of the attack.
C. Dietary management:
1. Counsel the client to avoid fatty foods.
2. Encourage the client to avoid fasting and starvation diets, which make the bile even more lithogenic.
D. Surgical management:
1. Cholecystectomy is recommended for symptomatic clients with positive findings on imaging. **The standard of care is the laparoscopic cholecystectomy.** Conversion from a laparoscopic procedure to an open surgical procedure is approximately 5%.
2. Clients are admitted to the hospital for intravenous (IV) fluid replacement, pain control, and antibiotics. Interventional therapy depends on client's risk factors for surgery and severity of symptoms.
3. Conservative treatment includes IV antibiotics, bowel rest, and an interventional cholecystectomy 6 to 8 weeks later.
4. A WBC count above 15,000 cells/μL indicates a nonresolving inflammation and is a predictor of treatment failure after 24 to 48 hours of conservative observation. Other predictive factors of failed conservative treatment include the following:
a. Age greater than 70 years.
b. Diabetes.
c. Distended gallbladder.
5. Percutaneous drainage (cholecystostomy) is reserved for clients who fail conservative treatment, or as a salvage procedure for high-risk clients.
6. Extracorporeal shockwave lithotripsy. ▶

E. Pharmaceutical therapy:

1. Nonsteroidal anti-inflammatory drugs (NSAIDs) are recommended for pain control.

2. Anticholinergics are not helpful.

3. Oral bile acid therapy decreases the amount of cholesterol produced by the liver and absorbed by the intestines. This therapy is reserved for poor surgical candidates who are at high risk for developing recurrent gallstones. Bile acid is not prescribed for calcified radio-opaque or radiolucent bile pigment stones or those with urgent need for surgery:

a. Actigall (Ursodiol):

i. Dissolution of stones: 8 to 10 mg/kg/d in two to three divided doses.

ii. Prevention therapy 300 mg BID.

b. Urso (Ursodiol) 250 mg tablets also available as Urso Forte 500 mg tablets:

i. 13 to 15 mg/kg/d in two to four divided doses.

FOLLOW-UP

A. See the client at next pain attack to reevaluate.

B. Surgical follow-up in 2 weeks.

C. Clients on oral bile acid therapy should follow up with serial ultrasound at 6 and 12 months, and 1 to 3 months after dissolution of the stone. AST and ALT should be measured at the start of therapy and every 6 months thereafter.

CONSULTATION/REFERRAL

A. For acute attack, consult a physician for possible surgical consultation referral.

B. Refer the client for elective cholecystectomy when they have documented gallstones and recurrent biliary colic or a history of complications from gallstone disease, such as pancreatitis.

C. Refer to a gastroenterologist for consideration of ERCP. The ERCP is the most common procedure for detecting and managing bile duct stones and may be performed before, during, or after gallbladder removal.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. RUQ pain in pregnancy differential includes preeclampsia, pancreatitis, and appendicitis.

2. In the absence of pancreatitis, maternal mortality should be rare and fetal loss is generally estimated to be no more than 5%. However, if secondary pancreatitis is present, maternal mortality is 15% and fetal loss is reported as high as 60%.

B. Geriatrics:

1. Signs and symptoms may be nonspecific and vague.

2. Localized tenderness may be the only presenting sign.

3. The response of Murphy sign may be diminished in older adults.

4. Persistent elevated WBC more than 15,000 suggests refractory disease and should play a central role in clinical follow-up for older adults with acute cholecystitis.

5. Early cholecystectomy is advocated for older adults with gallstone disease. The most important risk factor for postoperative morbidity and mortality is advanced age.

6. Gallstones occur in nearly 25% of females in the United States by the age of 60 and as many as 50% by age 75. Only about 20% of males have gallstones by their 70s.

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CIRRHOSIS OF THE LIVER

DEFINITION

A. Cirrhosis is the final stage of a pathologic process within the liver that leads to fibrosis and replacement of normal hepatic tissue with structurally abnormal nodules known as regenerative nodules. It is considered irreversible, but in mild cases has been shown to be reversible with treatment of the underlying condition. The scarring is most often caused by long-term exposure to toxins including alcohol and viral infections.

B. The natural history of liver cirrhosis (LC) is an asymptomatic compensated phase followed by a decompensated phase, marked by the development of overt clinical signs; the most frequent are ascites (5%–10%), bleeding, encephalopathy, and jaundice. Following the first appearance of any of these, the disease usually progresses more rapidly toward death or need for liver transplantation. The development of hyponatremia (serum sodium concentration <130 mmol/L in clients with cirrhosis) carries an ominous prognosis, as it is associated with increased morbidity and mortality.

C. Once decompensation has occurred, cirrhosis becomes a systemic disease, with multiorgan/system dysfunction. Cirrhosis also leads to an increased risk of developing hepatocellular carcinoma (HCC).

D. The concept of diagnosis of cirrhosis is changing from the documentation of histologic F4 fibrosis to the identification of clients truly at risk of developing complications.

1. Prevention of cirrhosis can be accomplished by the primary care provider by screening for alcohol abuse, viral hepatitis, and risk factors for development of nonalcoholic steatohepatitis (NASH).

2. Management of cirrhosis involves identifying preexisting hepatic disease, avoiding further injury to the liver, and managing the complications.

INCIDENCE

A. In the United States, chronic liver disease (CLD) and cirrhosis are the 12th leading cause of death, with about 1.5% of deaths per year and with a higher incidence in males. The transition from compensated asymptomatic cirrhosis to decompensated cirrhosis occurs at a rate of about 5% to 7% per year. The three most common causes of cirrhosis are chronic alcoholism, chronic hepatitis C virus (HCV), and nonalcoholic fatty liver disease (NAFLD).

B. Alcoholic liver disease (ALD) is one of the main causes of CLD worldwide and accounts for up to 48% of cirrhosis associated deaths in the United States.

1. Clients with ALD are at increased risk of developing HCC, with a lifetime risk of 3% to 10% and an annual risk of about 1%. Alcohol is also a frequent cofactor in clients with other type of liver disease where it accelerates hepatic fibrosis. Long-term heavy alcohol users remain at ▶

risk for advanced liver disease with alcoholic steatohepatitis (ASH), cirrhosis, and HCC.

2. Diagnosis of ALD requires documentation of chronic heavy alcohol use and exclusion of other causes of liver disease. Approximately 1 in 12 adults have alcohol use disorder (AUD), defined as consumption of more than three drinks per day in males and more than two drinks in females, or binge drinking defined by the National Institution of Alcoholism and Alcohol Abuse as more than five drinks in males and more than four drinks in females consumed over a 2-hour period.

3. Obesity and cigarette smoking are risk factors for HCC in clients with ALD.

4. Most clients with ALD present for medical care after they have developed jaundice or complications of cirrhosis.

C. NAFLD is a condition of excessive fat storage; there are two types: simple fatty liver and NASH.

1. Simple fatty liver (nonalcoholic fatty liver [NAFL]) has the presence of fat but little or no inflammation or liver cell damage. Simple fatty liver typically does not progress to cause liver damage or complications.

2. NASH causes inflammation and liver cell damage in addition to fatty liver. NASH may lead to cirrhosis or HCC.

D. Cirrhosis is complicated by bleeding from portal hypertension and also by portal vein thrombosis (PVT). PVT occurs in approximately 20% to 50% of clients with cirrhosis, particularly advanced cirrhosis. PVT is a hallmark of poor outcome. PVT vascular complications include increase of portal hypertension and bleeding risk, and higher complications in the early postliver transplantation period. At this time, clinical and laboratory variables to adequately assess the thrombotic risk in LC are limited. A meta-analysis shows than anticoagulants are efficacious and safe for treatment of PVT in clients with cirrhosis, but suggest the need of planning interventional clinical trials.

PATHOGENESIS

A. CLD resulting from various underlying etiologies causes death of hepatocytes, triggering an increase in hepatic enzymes. This process results in chronic inflammation, which over time leads to fibrosis of the liver. Fibrosis is a precursor to cirrhosis, or the replacement of normal liver parenchyma with regenerative nodules. Activation of hepatic stellate cells (HSCs) is a pivotal event in fibrosis. Defenestration and capillarization of liver sinusoidal endothelial cells are major contributing factors to hepatic dysfunction in LC. Activated Kupffer cells destroy hepatocytes and stimulate the activation of HSCs. Repeated cycles of apoptosis and regeneration of hepatocytes contribute to pathogenesis of cirrhosis.

B. The liver's ability to metabolize bilirubin is compromised, leading to elevated bilirubin levels. The liver's ability to synthesize clotting factors is reduced, creating elevated bleeding times. As the liver becomes increasingly fibrotic, the pressure in the portal system increases the risk of esophageal varices and gastrointestinal (GI) bleeding.

PREDISPOSING FACTORS

A. Toxicity:

1. Alcohol abuse.

2. Medications:

a. Methotrexate use.

b. Acetaminophen.

B. Inflammation:

1. Hepatitis C.

2. Hepatitis B.

3. Autoimmune hepatitis.

4. Sarcoidosis.

5. Schistosomiasis, syphilis, brucellosis.

C. Genetic:

1. NASH.

2. Hemochromatosis.

3. Primary biliary cirrhosis.

4. Primary sclerosing cholangitis (PSC).

5. Alpha-1 antitrypsin deficiency.

6. Wilson disease.

7. Hereditary hemorrhagic telangiectasia.

8. Polycystic liver disease.

9. Celiac disease. (Liver disease and Crohn's disease (CD) share widespread risk factors. Liver disorders such as autoimmune hepatitis, elevation of liver enzyme levels, primary biliary cirrhosis, nonspecific hepatitis, PSC, and NAFLD have been reported in clients with CD.)

D. Congestive heart failure (CHF): right-sided.

E. Budd–Chiari syndrome: a rare condition through which the venous outflow from the liver is blocked through either narrowing of the vessels or by thrombotic occlusion.

F. Idiopathic portal fibrosis.

COMMON COMPLAINTS

A. Clients can present with symptoms of cirrhosis or complications from cirrhosis.

1. Symptoms of cirrhosis include the following:

a. Abdominal distention.

b. Jaundice.

c. GI bleeding.

2. Complications include the following:

a. HCC.

b. Hepatic encephalopathy (HE): caused by the inability of the liver to metabolize toxic products; can vary in severity from mild barely perceptible symptoms (grade 1) to coma (grade 4).

c. Ascites.

d. Acute GI bleeding.

e. Bacterial peritonitis.

f. Hepatorenal syndrome.

g. Hepatopulmonary syndrome.

h. Hepatic hydrothorax.

i. Portopulmonary hypertension.

OTHER SIGNS AND SYMPTOMS

A. Nonspecific: anorexia, weight loss, weakness, and fatigue.

B. Pruritus.

C. Digital clubbing.

D. Dark-colored urine.

E. Sleep disturbances: indicative of encephalopathy.

F. Due to alteration in hormones, females develop anovulation and amenorrhea, whereas males develop hypogonadism and gynecomastia.

G. Decreased blood pressure (BP): sometimes noted with a previously hypertensive client.

SUBJECTIVE DATA

A. Inquire regarding duration of symptoms, associated factors, progression of symptoms, location of pain, severity of symptoms, and any exacerbating factors noted by the client or family. ▶

B. Review the client's medical history with attention to immunization status, prior infections, and hepatic disease.

C. Has the client had a liver biopsy? If yes, when and what were the results?

D. Has the client been treated for hepatitis B or hepatitis C?

1. How long ago was the client treated?

2. Did the client complete therapy? If not, why?

3. What was the client's response to therapy (e.g., nonresponder, relapser)?

E. Review the client's medication list including prescription and over-the-counter (OTC) medications. Note any hepatotoxic drugs, particularly methotrexate. See Table 14.2 for a nonexhaustive list of herbal and nutritional supplemental products that the client may be using for cirrhosis.

F. Review the client's surgical history.

G. Does the client have a history of varices? If yes, has any procedure been done including banding or a transjugular intrahepatic portosystemic shunt (TIPS)?

H. Review the client's social history including alcohol use, sexual preference and behavior, drug use (particularly history of intravenous [IV] drug use), international travel, and dietary history.

I. Review the client's family history, particularly for inheritable disease.

J. Review for presence of HE:

1. Moderate encephalopathy signs and symptoms include difficulty thinking, personality changes, poor concentration, confusion, forgetfulness, poor judgment, problems with handwriting or other small hand movements, and fetor hepaticus breath odor.

2. Severe encephalopathy signs and symptoms include lethargy, altered mental status, confused speech, seizures, severe personality changes, and extrapyramidal signs (Parkinson-like state).

K. Perform a thorough review of systems because decompensated cirrhosis affects many other organ systems.

TABLE 14.2 HERBAL AND NUTRITIONAL SUPPLEMENTS USED IN LIVER DISEASE

Sho-saiko-to (Xiao-Chai-Hu-Tang)	Bupleurum (*Bupleurum chinense, Bupleurum kaoi*)
Salvianolic acid B	Oxymatrine
Milk thistle (*Silybum marianum*)	MitoQ (Coenzyme Q10)
Licorice root (*Glycyrrhiza glabra*)	Long pepper (*Fructus Piperis Longi*)
Dandelion (*Taraxacum officinale*)	Japanese knotweed (*Polygonum cuspidatum*)
Asian ginseng (*Panax ginseng*)	Cordyceps (*Cordyceps sinensis*)
Chinese privet (*Ligustrum lucidum*)	Angelica (*Angelica archangelica*)
Reishi mushroom (*Ganoderma lucidum*)	Centella (*Centella asiatica*)
Kudzu (*Pueraria lobate*)	Fringe tree (*Chionanthus virginicus*)
SAMe (nutritional supplement)	Holy basil (*Ocimum sanctum L.*)
Phosphatidylcholine (nutritional supplement)	Liver Dr.

PHYSICAL EXAMINATION

A. Check BP, pulse, respirations, height, and weight to calculate body mass index (BMI). Observe for decrease in weight or reduction in BP.

B. Screening tests:

1. Substance abuse screening.

a. CAGE questionnaire for detecting alcoholism is available at www.hepatitisc.uw.edu/page/substance-use/cage.

b. Alcohol Use Disorders Inventory Test (AUDIT) at every visit. The AUDIT tool is available at www.hepatitisc.uw.edu/page/substance-use/audit-c.

2. Screen for depression at each visit. The Patient Health Questionnaire-9 (PHQ-9) calculator is located at www.mdcalc.com/phq-9-patient-health-questionnaire-9.

3. Screen for generalized anxiety disorder (GAD) for clients with ALD. The GAD questionnaire is available at adaa.org/screening-generalized-anxiety-disorder-gad.

4. Cognitive testing for mental status/signs of encephalopathy includes the following:

a. The clock-draw test (CDT); refer to Section II: Procedures, "Clock-Draw Test."

b. Clinical Hepatic Encephalopathy Staging Scale (CHESS): nine items ranging from normal (0) to deep coma (9). A copy of the CHESS is available at www.liver.ca/wp-content/uploads/2018/02/HE-Staging-Scale-CHESS-ENG.pdf.

c. The Stroop Color and Word Test (SCWT). In the most common version, clients are required to read three different tables as fast as possible. Clients read names of colors (color-words) printed in black (W) and name different color patches (C). Conversely the name color-word (CW) are printed in inconsistent color ink (e.g., "red" is printed in green ink). In this incongruent condition, the client is required to name the color of the ink instead of reading the word. The EncephalApp Stroop Test is available for download for Apple iOS from www.encephalapp.com and is available for Android systems through Google Play.

C. Inspect:

1. Observe overall appearance including gait and coordination, including evaluation for fetor hepaticus breath odor noted with HE, described as a distinctive strong, musty smell like a combination of rotten eggs and garlic.

2. Observe skin for jaundice and telangiectasia.

3. Inspect eyes for scleral icterus.

4. Check palms for presence of palmar erythema.

5. Inspect distal extremities for peripheral edema and clubbing.

6. Assess for gross asymmetries across the abdomen. Note any abdominal distention or protrusion.

7. Assess chest wall for presence of gynecomastia.

8. Inspect abdominal wall for presence of dilated vessels.

9. Inspect for gynecomastia and small testes.

D. Auscultate:

1. Evaluate presence of bowel sounds in all quadrants.

2. Listen over the area of the liver for and bruits or venous hums.

E. Palpate:

1. Palpation of the abdomen can be significant for a firm nodular liver; hepatomegaly may or may not be present.

2. Palpate for splenomegaly. ▶

3. Assess the abdominal distention for ascites, which can occur with a fluid wave. A positive test is noted when "a shock wave" of fluid moves against the fingertips pressed along the flank of the abdomen as the fluid is pushed from one side of the abdomen to the other by the force of a tap along the opposite flank:
   a. Grade 1, mild ascites, is only detectable by ultrasound.
   b. Grade 2, moderate ascites, manifests by moderate symmetrical distension of the abdomen.
   c. Grade 3, large or gross ascites, is manifested by marked abdominal distension.
4. Palpate the scrotum to evaluate size of the testes.

F. Percuss:
1. Percuss the costal margins for dullness. Begin percussion over the lungs and move from the area of resonate lung sounds to the areas of dullness. Mark the area of change. Start in the right lower quadrant (RLQ), repeating the same process. Measure the vertical distance from the top to the bottom. You can also use palpation to determine the lower border.

G. Neurologic exam:
1. Cranial nerve examination (refer to Section II: Procedures, "Neurologic Examination") for testing of sensations, temperature, and vibratory sensation.
2. Test for asterixis: Asterixis is the involuntary flapping of the wrist in a symmetric or asymmetric pattern with wrist extension. This test can also be elicited by the examiner pressing the client's wrists into hyperextension and then removing the pressure.

DIAGNOSTIC TESTS

A. Liver biopsy is still considered the gold standard to identify the typical features of cirrhosis. However, alternative diagnostic methods have been validated in comparison with liver biopsy and have a good diagnostic accuracy for the diagnosis of cirrhosis:
1. Currently, the most important and frequent scenario that requires a mandatory liver biopsy is the differentiation between severe alcoholic hepatitis and decompensated alcoholic cirrhosis, because at the present time there are no specific clinical signs or noninvasive methods to differentiate between the two conditions.
2. Liver biopsy remains key in the case of suspected NASH-related liver disease and in cholestatic and autoimmune CLD.
3. Noninvasive testing:
   a. Ultrasound-based techniques.
      i. Transient elastography (Fibroscan).
      ii. Point shear-wave elastography.
      iii. Two-dimensional (2D) shear wave elastography.
      iv. Ultrasound and Doppler.
   b. Clinical calculators:
      i. Fibrosis-4 (FIB-4) calculator: (www.hepatitisc.uw.edu/page/clinical-calculators/fib-4).
      ii. Echosense FibroMeter.
      iii. Aspartate aminotransferase (AST)/aspartate platelet ratio index (APRI): www.hepatitisc.uw.edu/page/clinical-calculators/apri.

B. Platelet count, although never used alone, is used to look for portal hypertension.

C. AST/alanine transaminase (ALT) index: elevated but may be normal in cases of advanced liver disease.

D. AST/platelet ratio index.

E. Serum prothrombin time (PT)/international normalized ratio (INR): Elevation indicates a decline in the liver's ability to produce clotting factors.

F. Complete blood count (CBC): anemia, leukopenia, thrombocytopenia.

G. Total bilirubin: elevation.

H. Alkaline phosphatase (ALP): elevated two to three times upper limit of normal.

I. Gamma-glutamyl transpeptidase (GGT): elevated.

J. Albumin: hypoalbuminemia.

K. Sodium: hyponatremia.

L. Creatinine: elevated.

M. Alpha-fetoprotein: marker for HCC. Used for monitoring CLD and development of HCC.

N. Urine drug screen (if indicated).

O. Imaging:
1. Ultrasound: noted earlier.
2. Abdominal contrast-enhanced CT scan. (Not routinely used for diagnosis of cirrhosis as similar information is obtained with ultrasound without exposure to excessive radiation.)
3. MRI:
   a. Abdominal contrast-enhanced MRI.
   b. Magnetic resonance elastography.
   c. Magnetic resonance angiography (MRA).

P. Endoscopy to visualize presence of varices. Noninvasive methods especially transient elastography are also being done to stratify the risk of varices.

Q. A diagnostic paracentesis is warranted to rule out spontaneous bacterial peritonitis.

R. Child–Pugh score for cirrhosis mortality estimates cirrhosis severity using total bilirubin, albumin, INR, presence/absence of ascites, and presence/absence of encephalopathy. An online calculator is available at www.hepatitisc.uw.edu/page/clinical-calculators/ctp.

S. Model of End-stage Liver Disease (MELD) score is used to estimate the severity and likely survival of clients awaiting liver transplantation. Factors include creatinine, bilirubin, INR, sodium, and recent dialysis. A score of 10 or greater indicates need for referral to hepatology. MELD exceptions (meaning these conditions are automatically given a score of 22 or greater) include HCC, hepatopulmonary syndrome, portopulmonary hypertension, and hepatic artery thrombosis. An online calculator is available at www.hepatitisc.uw.edu/page/clinical-calculators/meld.

DIFFERENTIAL DIAGNOSES

A. Cirrhosis.

B. HCC.

C. Budd–Chiari syndrome.

D. PVT.

E. Splenic vein thrombosis.

F. Inferior vena cava obstruction.

G. Sarcoidosis.

H. Schistosomiasis.

PLAN

A. Immunizations:
1. Clients should be up to date on all immunizations including hepatitis A and hepatitis B, if they are not already immune. Pneumococcal pneumonia and influenza are also recommended by the Centers for Disease Control and Prevention (CDC). ▶

B. Client teaching: avoidance of hepatotoxins:

1. Alcohol: Long-term mortality is related to severity of underlying liver disease and is dependent on abstinence from alcohol. Prolonged abstinence is the most effective strategy to prevent disease progression and treatment of the underlying AUD. Active alcoholism and previous alcohol use with less than 6 months of abstinence may be a contraindication to liver transplantation center's policy.

2. Clients with obesity or chronic HCV should avoid consumption of alcohol.

3. Counsel clients to stop smoking.

4. Counsel clients that OTC medications, including Tylenol or medications containing Tylenol, should not be taken.

5. Nonsteroidal anti-inflammatory drugs (NSAIDs) should not be used in clients with ascites.

6. Supplements: Many supplements are hepatotoxic.

7. Clients with hypovolemic hyponatremia have a fluid restriction (1,000 mL/d is recommended).

C. Pharmaceutical therapy: Because cirrhosis is the result of an underlying process, pharmaceuticals are directed at the causative agent (if applicable) or are used for supportive care of the complication:

1. The client's medications should be dose-adjusted for hepatic impairment.

2. The use of diuretics, antibiotics, beta-blockers, and anticoagulants is indicated in the treatment of ascites, bacterial peritonitis, portal vein hypertension and PVT, respectively. Lactulose and rifaximin therapy are used in the management on HE.

3. The use of proton pump inhibitors (PPIs) is generally avoided if possible due to their association with increased rates of bacterial peritonitis.

4. Angiotensin-converting enzyme (ACE) inhibitors, angiotensin II antagonists, or alpha-1 adrenergic receptor blockers should not generally be used in clients with ascites due to increased risk of renal impairment.

5. Aminoglycosides are discouraged in the presence of ascites; they are associated with an increased risk of acute kidney injury (AKI).

D. Diet: Refer to a dietician:

1. A moderate restriction of sodium intake (4.6–6.9 g of salt) is recommended in clients with moderate, uncomplicated ascites. This is generally equivalent to a no added salt diet with avoidance of preprepared meals.

2. Diets with a very low sodium content should be avoided because this is associated with diuretic-induced complications and can endanger a client's nutritional status.

3. Four to seven small frequent meals per day due to anorexia and nausea.

4. Ensure the diet contains adequate protein from a variety of sources.

EMERGENT ISSUES/INSTRUCTIONS

A. Clients presenting with acute GI bleeding should be hospitalized and undergo an urgent endoscopy.

B. Clients with moderate or severe alcohol withdrawal should be hospitalized. Severe forms of alcohol withdrawal include delirium tremens, generalized seizures, coma, cardiac arrest, and death.

FOLLOW-UP

A. Follow-up visit frequency is dependent on the severity of cirrhosis as determined by the MELD score:

1. Less than 10: every 6 to 12 months.

2. 11 to 18: every 3 months.

3. 19 to 24: every month.

4. 25 or greater: every week.

B. Monitor:

1. Lab: Monitor liver enzymes, renal function, electrolytes, blood count, and alpha fetoprotein:

a. Development of HCC with ultrasonography every 6 months.

2. Encephalopathy: This is indicated by personality change, change in consciousness, and sleep disturbance.

3. Ascites: A therapeutic paracentesis is needed for symptom relief of tense ascites.

4. Esophageal varices and acute GI bleeding through frequent endoscopy: Frequency is determined by severity of varices.

5. Bacterial peritonitis.

CONSULTATION/REFERRAL

A. Hepatology consultation:

1. Indicated for all clients whose diagnosis remains unclear after history, physical, serologic tests, and imaging.

2. Indicated for clients with cirrhosis in which the causative agent requires treatment, such as hepatitis C.

3. Clients in need of secondary prophylaxis of esophageal variceal hemorrhage and treatment of refractory ascites, or those for whom paracentesis is ineffective, should be evaluated for a TIPS insertion.

4. Indicated for clients who would benefit from liver biopsy. Liver biopsy is avoided if the biopsy will not change the management of condition, such as a late-stage diagnosis.

5. MELD score should be calculated at the time of the diagnosis and recalculated frequently during management. Referral to a transplant center is indicated for a MELD score 10 or greater.

B. Clients with cirrhosis are best managed by an interdisciplinary team including drug and alcohol addiction specialists.

C. Dietary consultation.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Older clients are at greater risk for delirium tremens with alcohol withdrawal.

RESOURCES

U.S. Department of Veterans Affairs: www.hepatitis.va.gov MD Calc: www.mdcalc.com Support Groups: www.liverfoundation.org/resources

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COLORECTAL CANCER SCREENING

DEFINITION

A. Screening for colorectal cancer (CRC) has increased early detection and has allowed for early intervention of premalignant localized cancer. There are multiple screening guidelines. ▶

Table 14.3 outlines current recommendations for colorectal screening for adults at average risk from the U.S. Preventive Task Force (USPTF), the American Cancer Society (ACS), and the U.S. Multi-Society Task Force (MSTF) on CRC. There are two categories of screening:

1. Stool-based testing.
2. Direct visualization tests.

INCIDENCE

A. In the United States, CRC is the second leading cause of cancer-related death in both males and females.
B. Americans have a 5% lifetime risk of CRC.
C. CRC is rare before age 40 years.
D. Ninety percent of the cases of CRC occur after 50 years. The majority of cases are diagnosed between 65 and 74, with the average age of diagnosis at 68 and the average age of death at 73.
E. African Americans have a higher overall incidence rate and younger mean age at onset of CRC:

1. Lower screening rates.
2. Higher CRC incidence rate.
3. Earlier mean age at onset.
4. Worse survival and late-stage presentation.
5. Higher proportion of cancers before age 50.

F. Up to 20% of CRC in the United States are associated with smoking.
G. Obesity is associated with colon cancer but not increases in rectal cancer. Abdominal obesity is a stronger risk factor than truncal obesity or body mass index (BMI).

PATHOGENESIS

A. The usual pathogenesis is the development of a slow-growing adenomatous polyp followed by onset of dysplasia and finally cancerous cells.

PREDISPOSING FACTORS

A. Age: 50 years and older.
B. Male.
C. African American.
D. Family history/genetic: accounts for 20% of all CRCs:

1. Familial adenomatous polyposis (FAP).
2. Nonpolyposis CRC: Lynch syndrome.
3. MUTYH-associated polyposis (MAP).
4. Family history outside of genetic factors.

E. Smoking and moderate to heavy alcohol use (on average three drinks or more daily).
F. Obesity.

TABLE 14.3 CRC SCREENING RECOMMENDATIONS FOR ADULTS AT AVERAGE RISK

	U.S. Preventive Services Task Force: 2016	American Cancer Society: 2018	U.S. MTSF[a] on CRC: 2017
Ages for screening	Start screening at age 50. Stop screening at 75. The decision to be screened after 75–85 should be made on an individual basis taking into account the client's overall health and screening history. Screening after age 85 is not recommended.	Start screening at age 45. For adults with average CRC risk, in good health, and with a life expectancy of more than 10 years, screening should continue through age 75 years. The decision to be screened for clients aged 76–85 years should be individualized to client preference, life expectancy, health status, and history of screening. Screening after 85 is not recommended.	• Start screening at age 50. • Limited evidence supports screening African Americans at 45 years. • Stop screening at age 75 or have <10 years of life expectancy. • Persons without prior screening should be considered for screening up to age 85, depending on age and comorbidities. • Colonoscopy and FIT are recommended as the cornerstones of screening regardless of how screening is offered.
Highly sensitive gFOBT	Annual	Annual	No recommendation
Highly sensitive FIT	Annual	Annual	Annual
FIT fecal DNA	Every 1 or 3 years[b]	No recommendation	Every 3 years
MT-s DNA test		Every 3 years	No recommendation
FSIG	Every 5 years OR the FSIG and FIT DNA recommendation	Every 5 years	Every 5–10 years
FSIG and FIT DNA	FSIG every 10 years plus FIT every year	No recommendation	No recommendation
Colonoscopy	Every 10 years	Every 10 years	Every 10 years[c]
CT colonography (virtual colonoscopy)	Every 5 years	Every 5 years	Every 5 years

[a]MSTF represents the American College of Gastroenterology, the American Gastroenterological Association, and the American Society for Gastrointestinal Endoscopy.

[b]FIT DNA testing suggested by manufacturer.

[c]Colonoscopy offered first, FIT should be offered to clients who decline colonoscopy.

CRC, colorectal cancer; FIT, fecal immunochemical test; FSIG, flexible sigmoidoscopy; gFOBT, guaiac-based fecal occult blood test; MT-s DNA, multitargeted stool DNA; MTSF, Multi-Society Task Force.

G. Diet high in red meat and fats.
H. Personal history of prior adenomatous polyps or isolated CRC.
I. Inflammatory bowel disease (IBD: Crohn's disease [CD] and ulcerative colitis [UC]).
J. Diabetes/insulin resistance.
K. History of renal transplantation.
L. History of abdominal radiation.

COMMON COMPLAINTS
A. Asymptomatic screening.

SUBJECTIVE DATA
A. Review the client's age and risk factors to discuss screening for CRC.
B. Review family history of CRC.
C. Review smoking history and alcohol consumption.
D. Review the client's diet, evaluating red meat, processed meats, and lack of grains, fruits, and vegetables.
E. Review all medications currently being taken, including over-the-counter (OTC) and herbal products.

PHYSICAL EXAMINATION
A. Examinations are not required for discussion on colorectal screening testing.
B. A physical examination and vital signs should be taken as indicated for other presenting complaints.

DIAGNOSTIC TESTS
A. Stool-based testing:
1. Guaiac-based fecal occult blood test (gFOBT).
2. Fecal immunochemical test (FIT).
3. FIT fecal DNA.
4. Multi-targeted stool DNA test (MT-sDNA).

B. Direct visualization tests:
1. Flexible sigmoidoscopy.
2. **Colonoscopy: The American College of Gastroenterology (ACG) recommends the colonoscopy as the preferred strategy for screening.**
3. CT colonography (virtual colonoscopy).

C. Serum, urine, and capsule endoscopy are NOT recommended for CRC screening.

DIFFERENTIAL DIAGNOSES
A. None related to screening.

PLAN
A. Client teaching:
1. Educate client about modifying controllable risk factors with diet, exercise, smoking cessation, and low or no consumption of alcohol (on average one or less drinks per day).
2. Discuss the procedures and the preparation needed for each test.

B. Pharmaceutical therapy:
1. Bowel prep for flexible sigmoidoscopy and colonoscopy depends on the test, client's age, and other comorbidities.
2. Refer to Chapter 2, Table 2.2 for the U.S. Preventive Services Task Force (USPSTF) recommendations for aspirin use to prevent cardiovascular and colorectal disease recommendations by age range.

FOLLOW-UP
A. Follow-up CRC screening is determined by family history, underlying risk factors, results of the prior screening test, and the test used for screening.
B. Clients with classic FAP (>100 adenomas) should be advised to have genetic counseling.
C. Nonsteroidal anti-inflammatory drugs (NSAIDs) have been associated with a decrease in the risk of developing CRC. There is insufficient evidence to recommend the use of NSAIDs as a prevention strategy.

CONSULTATION/REFERRAL
A. Refer to a gastroenterologist and/or surgeon as indicated.

INDIVIDUAL CONSIDERATIONS
A. Geriatrics:
1. Medicare Part B covers colorectal screening tests. See Chapter 2, "Healthy Living for the Adult-Geriatric Client," for the full Medicare coverage guidelines.

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CONSTIPATION

DEFINITION
A. Constipation means infrequent and difficult defecation of hard stools and a sensation of incomplete evacuation or straining. Constipation may also refer to a decrease in the volume or weight of stool, and the need for enemas, suppositories, or laxatives to maintain bowel regularity. Constipation is a symptom, not a disease.
B. The lower limit of normal stool frequency is three bowel movements (BMs) a week. The Rome consensus criterion defines constipation as two or more of the following occurring over a period of 3 months: two or fewer stools weekly, sensation of straining, sensation of incomplete evacuation or sensation of obstruction or blockage, and/or the need for digital removal of stool.
C. Classification of constipation includes the following:
1. Primary constipation:
   a. Normal-transit constipation (most common).
   b. Functional constipation (slow transit).
   c. Defecatory disorders.
2. Secondary constipation:
   a. Dietary deficiency.
   b. Structural obstruction.
   c. Systemic: related to endocrine, neurologic, or connective tissue disorder.

INCIDENCE
A. The incidence of constipation is 16% among all adults and 33% in clients older than 60 years of age. Constipation is commonly self-reported. Constipation occurs in more than 50% of clients with colorectal cancer (CRC); it is usually a symptom of advanced disease, but it may be the presenting complaint.

PATHOGENESIS
A. Constipation can be caused by an alteration of the filling of the rectum by colonic transportation and/or reflex defecation of stool.
B. Lack of exercise and prolonged bedrest decrease propulsion of bowel contents.
C. During pregnancy, progesterone has a relaxing effect on the muscles of the gastrointestinal (GI) tract and causes a decrease in peristalsis. The compression of the intestines by the enlarging uterus causes constipation during pregnancy. ▶

D. Habitual use of laxatives is associated with impaired colonic motor activity and has the potential of producing hypokalemia.
E. Hypokalemia can produce a generalized ileus and is most often seen in clients who take diuretics.
F. Psychiatric disease and psychosocial distress have important roles. The exact mechanisms by which emotional difficulties lead to constipation remain unclear, but their contribution is widely recognized.
G. Drugs (Table 14.4).

PREDISPOSING FACTORS

A. Insufficient nutrition:
  1. Low-fiber diet.
  2. Low fluid intake.
B. Neurologic causes:
  1. Spinal cord injury.
  2. Parkinson disease.
  3. Multiple sclerosis.
  4. Aganglionosis (Hirschsprung disease).
  5. Sacral nerve trauma/tumor.
C. Sedentary lifestyle/impaired mobility.
D. Laxative abuse.
E. Travel.
F. Ignoring urge to defecate.
G. Drug use (individual medications and polypharmacy).
H. Pregnancy, especially third trimester.
I. Psychosocial problems:
  1. Depression.
  2. Sexual abuse.
  3. Unusual attitudes to food and bowel function.
  4. Obsessive/compulsive.
J. Extremes of ages: Infants and geriatrics.
K. Hypothyroidism.
L. CRC.
M. Irritable bowel syndrome (IBS).
N. Pelvic floor disorders:
  1. Impaired function of the pelvic floor and/or external sphincter.
  2. Pelvic floor obstruction.
  3. Rectal prolapse.
  4. Enterocele and/or rectocele.
  5. Rectal intussusception.

TABLE 14.4 DRUGS AND CLASSIFICATIONS THAT CAUSE AND INCREASE CONSTIPATION

Anticholinergics (atropine, antidepressants, neuroleptics, antiparkinsonian drugs, antispasmodics)	Opiates
Antipsychotics	Cholestyramine (binds bile salts)
Anticonvulsants	Antacids (aluminum hydroxide and calcium carbonate)
Antihistamines	Iron supplements
Antihypertensives (calcium channel blockers, clonidine, hydralazine, MAOIs, methyldopa)	NSAIDs
Diuretics	Ganglionic blockers

NSAIDs, nonsteroidal anti-inflammatory drugs; MAOIs, monoamine oxidase inhibitors.

COMMON COMPLAINTS

A. Hard, infrequent stools.
B. Straining.
C. Inability to defecate when desired.
D. Need for digital manipulation to facilitate evacuation.

OTHER SIGNS AND SYMPTOMS

A. Hard, pebbly, rocklike stools.
B. Painful defecation.
C. Abdominal pain/distention.
D. Weight loss.
E. Blood in stools.

POTENTIAL COMPLICATIONS

A. A rectal prolapse of the mucosa is pink and looks like a doughnut or rosette. Complete prolapse involving the muscular wall is larger and red, and has circular folds.
B. Anal fissure.
C. Hemorrhoidal disease.
D. Pelvic floor damage in females.
E. Rectal ulceration.
F. Fecal impaction.
G. Bowel obstruction.
H. Fecal incontinence (FI).
I. Megacolon.
J. Volvulus.
K. Urinary retention.
L. Syncope.
M. Anal fistula.

SUBJECTIVE DATA

A. Review the onset, duration, and course of symptoms:
  1. Is constipation a chronic or acute problem?
  2. If there has been a change in bowel habits, was it gradual or sudden?
  3. What feature does the client rate most distressing?
B. Review bowel habits:
  1. Does the client have a regular time for defecation?
  2. Review size, color, consistency, and frequency of stools.
  3. Is there any blood?
  4. Are there any periods of diarrhea?
  5. How often are laxatives being used and what doses?
  6. Are suppositories and enemas also required?
  7. Does the client have the urge to defecate?
  8. Does the client have a sensation of incomplete evacuation?
  9. Does the client need to digitally remove stool?
C. Review the client's daily diet and fluid intake. Has there been any dietary change?
D. Review the client's medication history: prescription and over-the-counter (OTC; refer to Table 14.4).
E. Review the client's daily physical activity.
F. Review the client's psychosocial history of stress, depression, anxiety, and coping mechanisms.
G. Review the client's other health problems such as diabetes, depression, hypothyroidism, and hypercalcemia.
H. Review family history of constipation and CRC.
I. Review surgical history.

PHYSICAL EXAMINATION

A. Check pulse, respirations, blood pressure (BP), and weight. Check temperature if indicated.
B. Inspect:
  1. General overall assessment of nutritional status.
  2. Examine the skin for pallor and signs of dehydration and hypothyroidism. ▶

3. Evaluate for presence of hernias.
4. Inspect anus, including the position, presence of perianal erythema, hemorrhoids, and skin tags.
5. Observe for signs of fecal soiling.
6. Examine the lower back to rule out spinal lesions: hairy or hyperpigmented patches, gluteal fold asymmetry, cutaneous dimples, sinus tracts, and lipomas.
7. The American Gastroenterology Association (AGA) notes that the client should be in the left lateral position with the buttocks separated to evaluate:
   a. Observe the descent of the perineum during simulated bowel evacuation.
   b. Observe the elevation during a squeeze aimed at retention of stool.
   c. During the simulated defecation, the anal verge should be observed for any patulous opening or prolapse of anorectal mucosa.

C. Auscultate:
1. Auscultate abdomen for bowel sounds. Auscultation of the abdomen should precede any palpation or percussion due to the changes in intensity and frequency of sounds after manipulation. Bowel sounds may be high pitched or absent in the presence of constipation.

D. Percuss:
1. Percuss the abdomen.

E. Palpate:
1. Palpate the abdomen for masses, tenderness, distension, and fecal mass.
2. Palpate the liver and spleen.

F. Digital rectal exam:
1. Examine the rectum for masses, hemorrhoids, fissures, fistula, prolapse, inflammation, and anal warts.
2. Evaluate for impaction, hard stool in ampulla.
3. Perform an anal "wink" reflex test by a light pinprick or scratch.
4. Evaluate sphincter tone:
   a. Disordered innervation of the anus is indicated by finding that the anal canal opens wide when the puborectalis muscle is pulled posteriorly.
   b. Evaluate the resting tone of the sphincter and squeezing effort.
   c. Instruct the client to "expel the examination finger" to evaluate the force of expulsion.

G. Neurologic examination:
1. Perform a neurologic examination for tone, strength, and reflexes to search for focal deficits and delayed relaxation phase of the ankle jerks, suggestive of hypothyroidism.

H. Pelvic examination:
1. Perform pelvic examination to evaluate a prolapse or rectocele. Evaluate when the client is at rest and with straining.

I. Mental status:
1. Perform mental status examination, checking for signs of depression and somatization.

DIAGNOSTIC STUDIES

A. No tests are required for the diagnosis of chronic constipation.

B. Because laboratory tests have demonstrated low sensitivity for indicating a cause for constipation, unless there are alarm symptoms (as defined as follows), initial treatment may consist of alterations in the diet and activity level, and addition of laxatives. The following are alarm symptoms:
1. Weight loss of greater than 10 lbs.
2. Rectal bleeding or positive test for occult blood.
3. Abdominal pain.
4. Inability to pass flatus.
5. Vomiting.
6. Change in bowel habits in a client older than 50 years.
7. Positive family history of CRC or inflammatory bowel disease (IBD).

C. If a trial of lifestyle modifications and/or laxatives is unsuccessful, further investigation is warranted.

D. Laboratory tests:
1. Complete blood count (CBC).
2. Thyroid studies.
3. Potassium and calcium. **Clients taking diuretics should have serum potassium checked. Hypokalemia may reduce bowel contractility and produce an ileus.**
4. Serum glucose to rule out diabetes.
5. Stool for occult blood.

E. Radiographs:
1. Plain abdominal radiographs may be useful to evaluate stool burden and assess for obstruction or megacolon.
2. Barium enema (BE) to evaluate megacolon and redundant sigmoid colon.

F. Endoscopic:
1. Flexible sigmoid.
2. Colonoscopy (especially if the client is older than 50 or has had abrupt onset of constipation).
3. CT colonography.

G. Anorectal function tests if there is no response to a trial of laxatives:
1. Balloon expulsion.
2. Colonic transit.
3. Anorectal manometry.
4. Defecography.
5. Electromyography (EMG): anal surface.

DIFFERENTIAL DIAGNOSES

A. Constipation.
B. Intestinal obstruction: Acute onset of constipation requires ruling out an ileus, especially when accompanied by abdominal discomfort.
C. Hypothyroidism.
D. Psychosocial dysfunction.
E. Fecal impaction.
F. Neurologic disorders.
G. Multiple sclerosis.
H. Spinal cord injury.
I. Cancer.
J. Drug use.
K. Celiac disease: Constipation is often the presenting complaint in Crohn's disease (CD).
L. Diabetes (chronic dysmotility).
M. IBS: constipation predominant.

PLAN

A. Client teaching: *See Client Teaching Guide for this chapter, "Tips to Relieve Constipation."*
1. Encourage the client to exercise. Both exercise and dietary fiber stimulate the natural wavelike contraction of the colon that trigger the urge to defecate.
2. Reassure the client that recommended dietary changes and exercise help with constipation.
3. Aim to reduce dependency on laxatives. Warn chronic laxative users that it may take 4 to 6 weeks before spontaneous BMs return.
4. Provide education regarding potential complications of long-term constipation.
5. Encourage defecation after meals, especially in the morning hours when colonic motility is most pronounced. ▶

6. Ask the client to keep a stool diary to bring to their next appointment. A BM record is available at www.nhsggc.org.uk/media/241191/nhsggc-bristol-stool-chart.pdf.

B. Dietary management:

1. See Appendix B, "High-Fiber Diet."

C. Medical management of idiopathic constipation:

1. Treatment of constipation is symptomatic and should begin with lifestyle and dietary changes:

a. Direct relationship between fiber intake, water intake, and fecal output. Fiber goal is 20 to 35 g daily.

b. Use of bulk-forming laxatives encouraged first. These agents include psyllium (Metamucil), methylcellulose (Citrucel), calcium polycarbophil (Fibercon), and wheat dextrin (Benefiber). Of these choices, psyllium or methylcellulose produce the best subjective results. All bulk-forming agents produce inconsistent objective results.

c. Nonbulk-forming laxatives may be initiated:

i. Surfactants: Colace: generally used short term.

ii. Osmotics: polyethylene glycol (PEG; MiraLax): may be used long term but may produce electrolyte imbalances in the older adults or clients with renal disease.

iii. Stimulants: bisacodyl, senna: may be added every other day or every third day to the previous choices. The intensity of the action of stimulant laxatives is dose-related. A large dose of any stimulant laxative can produce serious adverse effects including severe cramps, dehydration, and hypokalemia. There is concern that chronic, long-term use of stimulant laxatives can lead to loss of colon function (cathartic colon).

2. Evaluate and stop medications, if possible, that cause constipation.

3. Mineral oil, also known as liquid paraffin, should be used with caution if the client is at risk for aspiration or accidental inhalation secondary to the risk of aspiration pneumonia. Mineral oil interacts with fat-soluble vitamins and anticoagulants.

D. Medical management of severe or refractory constipation:

1. Suppository: bisacodyl or glycerin; used for defecatory dysfunction since the products liquify stool and relieve obstruction.

2. Fecal impaction may require manual fragmentation followed by enema to relieve the situation. Enemas should not be given routinely to treat constipation because they disrupt normal defecation reflexes and the client becomes dependent. After manual disimpaction, mineral oil/warm water enema is used to soften the remaining stool and evacuate the rectum. PEG solution may then be administered orally daily with the intention of stimulating one BM every day to every other day. Investigation to ascertain the initial cause of constipation should be undertaken.

3. Manual removal of fecal impaction can stimulate the vagus nerve and cause syncope and tachycardia. It is contraindicated in the following conditions:

a. Pregnancy.

b. After genitourinary, rectal, perineal, abdominal, or gynecologic surgery.

c. Myocardial infarction (MI), coronary insufficiency, pulmonary embolism (PE), congestive heart failure (CHF), or heart block.

d. GI or vaginal bleeding.

e. Blood dyscrasias or bleeding disorders.

f. Hemorrhoids, fissures, and rectal polyps.

TABLE 14.5 LAXATIVES

Bulk laxatives	Psyllium (Konsyl, Metamucil) polycarbophil Malt soup extract (Maltsupex) Methylcellulose (Citrucel) Calcium polycarbophil (FiberCon) Wheat dextrin (Benefiber)
Saline laxatives	Magnesium hydroxide (Milk of Magnesia) Magnesium citrate Fleet Phospho-Soda
Lubricating agents (emollient)	Magnesium citrate Mineral oil
Stimulant laxatives	Docusate Bile acids Bisacodyl (Dulcolax) Castor oil Senna Cascara Aloes
	Rhubarb Ex-Lax Senokot Correctol
Osmotic agents	Magnesium and phosphate salts Lactulose (Kristalose) Sorbitol Polyethylene glycol (Colyte, Glycolax, MiraLax)
	Glycerin suppositories

E. Pharmaceutical therapy:

1. Bulk-forming agents (Table 14.5) reduce abdominal pain and improve stool consistency. These should be used if an increase in dietary fiber does not work. They act by causing retention of fluid and increasing fecal mass. They must be taken with plenty of fluids to prevent formation of an obstructing bolus. Flatulence and abdominal distension may occur, but long-term use is safe.

2. Stimulant laxatives (see Table 14.5) act by directly stimulating the colonic nerves. Suppositories are faster (20–60 minutes) versus oral laxatives (8–12 hours).

3. Osmotic laxatives (see Table 14.5) act by retaining fluid in the bowel by osmosis, changing the water distribution in the feces. Good hydration is important. Clients who need to limit their sodium intake, such as those with CHF, renal disease, and hypertension, should not use osmotic laxatives that contain sodium.

4. Newer agents:

a. Lubiprostone (Amitiza): chloride channel activator; indicated for use in IBS with constipation predominant (IBS-C), opioid-induced constipation (OIC), and chronic idiopathic constipation (CIC). Dosing adjustments are required for hepatic impairment. The effectiveness of Amitiza has not been established for a diphenylheptane opioid (e.g., methadone).

b. Linaclotide (Linzess): guanylate cyclase C agonist; indicated for IBS-C and CIC.

c. In 2018 the U.S. Food and Drug Administration (FDA) accepted a new drug application for prucalopride (Resolor). Prucalopride, a high affinity, selective serotonin type 4 (5-hydroxytryptamine [5-HT4]) receptor agonist, is a GI prokinetic agent that stimulates colonic peristalsis, increasing bowel motility associated with CIC. ▶

5. When severe depression requires the use of antidepressants, the least constipating agent should be selected (i.e., one with minimal anticholinergic activity).

6. Treat other identified causes, for example, hypothyroidism.

F. Multidisciplinary referrals:

1. Pelvic floor physiotherapy may be offered. The pelvic floor retraining involves teaching the client to relax their pelvic floor muscles during straining and to correlate relaxation and pushing to achieve defecation.

2. Biofeedback has been effective for short-term treatment of intractable constipation and for treatment of FI. Biofeedback has been shown to improve rectoanal coordination during defecation and symptoms of constipating despite reduced laxative use.

FOLLOW-UP

A. Repeat assessment in 4 to 6 weeks. Ask the client to keep a stool diary and bring it in for subsequent appointments.

B. If there is no evidence of obstruction, anemia, or occult blood loss, follow the client expectantly for a few weeks on a conservative program that includes increased dietary fiber and increased exercise, and follow stool guaiac.

C. Failure to improve may indicate a serious underlying cause and need for referral.

EMERGENT ISSUES/INSTRUCTIONS

A. Acute onset of constipation is considered a red flag in the geriatric population.

B. Fever, abdominal pain, inability to pass flatus, and abdominal distention may be a sign of bowel obstruction and require immediate evaluation.

CONSULTATION/REFERRAL

A. Consider consultations with a gastroenterologist, gynecologist, or psychologist/psychiatrist as indicated.

B. Surgical consultation may be required for slow-transit constipation. Total colectomy with ileorectal anastomosis may be required when there is failure of aggressive, prolonged trials of laxatives, fiber, and prokinetic agents.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Constipation is very common in pregnancy secondary to progesterone and the enlarging uterus (see "Pathogenesis").

2. Constipation that results from iron supplementation can be avoided by increasing the intake of fluid and high-fiber foods, and increasing physical activity such as walking.

3. Bulking agents and lactulose will not enter breast milk. Senna, in large doses, will enter breast milk and may cause diarrhea and colic in infants.

B. Adults:

1. The most common cause of chronic constipation in adults is failure to initiate defecation.

2. Diabetics should avoid stimulant laxatives such as lactulose and sorbitol. Their metabolites may influence blood glucose levels.

C. Geriatrics:

1. Constipation in older individuals is not a result of aging; instead it is considered a geriatric syndrome. Nurse practitioner (NPs) should practice special consideration of this symptom.

2. Constipation is usually related to an increase in constipating factors such as chronic illnesses, immobility, dietary factors, medications, neurologic factors, and psychiatric conditions.

3. An important diagnostic concern in older adults is the possibility of constipation being due to a colonic neoplasm. More than 25% of clients with colorectal carcinomas present with constipation.

4. Check for a fecal impaction, especially in older adults with a history of chronic constipation. Fecal impaction ranks as one of the major sources of anorectal discomfort among older adults and bedridden. Chronic, incomplete evacuation leads to formation of an obstructing bolus of desiccated hard stool in the rectum.

5. Diarrhea rather than constipation is sometimes the only complaint due to the collection of liquid stool distending the proximal colon and passing around the obstructing bolus.

6. Beers Criteria caution: A full review of medications, herbals, and OTC drugs should be undertaken to identify medications that should be avoided in the geriatric population, duplicate medications, and medications that are able to be decreased or deprescribed. (Refer to section on deprescribing in Chapter 3, "Adult-Geriatric Assessments"):

a. Refer to Appendix C: Beers Criteria, Table C.1 to identify drugs that have highly anticholinergic side effects and are potentially inappropriate medications for use in older adults, including the following:

i. First-generation antihistamines.
ii. Antispasmodics.
iii. Antidepressants.
iv. Opioids.
v. Calcium channel blockers.
vi. Anticonvulsants.
vii. Muscle relaxants.

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CROHN'S DISEASE

DEFINITION

A. Crohn's disease (CD) is a chronic, progressive, destructive inflammatory bowel disorder with both intestinal and extraintestinal manifestations. It produces focal, asymmetric, transmural inflammation, ulceration, and fibrosis, resulting in malabsorption. CD can involve any segment of the gastrointestinal (GI) tract from the mouth to the anus; the terminal ileum and colon are the most common sites:

1. CD is subclassified based on age, location (ileal, colonic, ileocolonic, or upper GI), and clinical presentation (nonstricturing/nonpenetrating to penetrating) using the Montreal classification.

2. Older adults with inflammatory bowel disease (IBD) can be subdivided into two groups:

a. Older adults with onset of IBD at a late age (late-onset IBD).

b. Older adults with long-standing IBD; first diagnosed as having IBD at a younger age (long-standing IBD).

B. The disease is chronic, relapsing, and incurable. CD is characterized by episodes of remission and exacerbation. The most frequent cause of death in persons with IBD is the primary disease, followed by malignancy and thromboembolic disease. Symptoms of CD do not correlate well with the presence of active inflammation and therefore should not be the sole guide for therapy. The diagnosis is usually established by objective evaluation with endoscopic finding in a client with a compatible clinical history. Objective evidence for disease activity should be sought before administering medication that may have significant adverse effects.

C. Up to 80% of clients with CD undergo surgery at some point during their clinical course. Indications for surgery include stricture, intractable or fulminant disease, anorectal disease, and intra-abdominal abscess. Surgical procedures include bowel resection and ileal pouch–anal anastomosis (IPAA). The 10-year cumulative risk of a major abdominal surgery in CD is 30% in the biologic era of treatment. The 10-year risk of a second resection after the first is 30% to 35%.

D. The lifetime risk of fistula development is 20% to 40%.

E. The incidence of adenocarcinoma in clients with CD is higher than in the general population. Lymphoma is also increased, especially in clients with IBD treated with azathioprine (AZA; 6-mercaptopurine [6-MP]).

INCIDENCE

A. The incidence rate ranges from 3.1 cases per 100,000 to 20.2 cases per 100,000. An estimated one to two million people in the United States have ulcerative colitis (UC) or CD. The incidence of IBD has been reported to be highest in Jewish populations. IBD is very prevalent among the American Jewish population—four to five times that of the general population.

B. The peak incidence of CD is most common in late adolescence to the third decade of life. Another peak occurs in persons aged 50 to 80 years. It is arguable if the increased incidence later in life is due to prior misdiagnosis or actual development of new disease. Ten to fifteen percent of cases of IBD are diagnosed in clients older than 60 years of age.

C. CD may involve the entire GI tract; note the incidence according to the location:

1. 80% have small bowel involvement, usually in the distal ileum. In severe cases of ileitis, complications may include fistulas or an abscess in the right lower quadrant (RLQ) of the abdomen.
2. 50% have ileocolitis (involving both the ileum and colon). This type is associated with significant weight loss.
3. 20% have disease limited to the colon, with roughly one-half having sparing of the rectum.
4. A small percentage has predominant involvement of the mouth (aphthous ulcers) or gastroduodenal area; fewer have involvement of the esophagus (odynophagia and dysphagia) and proximal small bowel.
5. One-third have perianal disease (perianal pain, drainage from large skin tags, anal fissures, perirectal abscesses, and anorectal fistulae).
6. 15% to 20% have arthralgias. Arthritis is the most common complication.

PATHOGENESIS

A. Pathogenesis is multifactorial and remains unclear. Dysregulation of the immune system, changes in the composition of intestinal flora, and presence of genetic susceptibility combine to produce inflammation of the mucosal lining of the intestinal tract, resulting in ulceration, edema, bleeding, and fluid and electrolyte loss.

B. The 2018 American Gastroenterology Association (AGA) guidelines note genetic testing is not indicated to establish the diagnosis of CD. There are currently 200 genetic foci that are being studied to determine their role in the pathogenesis of IBD. CD and UC share approximately 70% of their genes, indicating a close relationship between the two processes.

PREDISPOSING FACTORS

A. Age between 15 and 35 years and another peak at 50 to 80 years.

B. Genetic predisposition/family history of CD:

1. First-degree relative 5- to 20-fold increased risk.
2. 70% incidence in identical twins versus 5% to 10% in nonidentical twins.
3. Jewish populations.

C. Smoking exacerbates disease activity and accelerates disease recurrence.

D. Obesity: unclear if there is increased risk but complications are more severe and frequent.

E. Physical activity: lower risk in active individuals.

F. Dysbiosis: antibiotic use (particular metronidazole and quinolones); presence of infection.

G. Breastfeeding history: three to four times increased risk in clients that were not breastfed.

H. Medications: Use of tretinoin, nonsteroidal anti-inflammatory drugs (NSAIDs), and oral contraceptives have all been implicated in the development of Crohn's, although the relationship remains unclear.

I. History of appendectomy: Some studies show a protective benefit after appendectomy.

J. Psychosocial factors.

K. Sleep duration.

COMMON COMPLAINTS

A. The following hallmark/cardinal symptoms occur in about 80% of clients.

1. Abdominal pain, the classic location being in the RLQ (appendicitis-like):
   a. Abdominal pain often worsens postprandially.
2. Diarrhea, chronic or nocturnal.
3. Fatigue: very prevalent; thought to arise from inflammation, anemia, and vitamin and mineral deficiencies.
4. Constitutional symptoms at presentation:
   a. Fever.
   b. Weight loss/anorexia.
   c. Growth failure (Younger ages).

OTHER SIGNS AND SYMPTOMS

A. Symptoms vary depending on the location of the intestinal tract and extent of disease.

1. Constipation: early sign.
2. Bowel urgency and sense of incomplete evacuation.
3. Rectal bleeding.
4. Abdominal mass.
5. Night sweats.
6. Pallor/anemia.
7. Perianal discomfort or soft or semiliquid irritating rectal discharge.
8. Recurrent fissures and fistulas, abscesses sometimes extending to skin.
9. Folate deficiency.
10. Menstrual irregularities/amenorrhea.

▶

11. Extraintestinal symptoms: classic:
- a. Arthropathy (both axial and peripheral).
- b. Dermatologic:
  - i. Pyoderma gangrenosum.
  - ii. Erythema nodosum.
- c. Ocular:
  - i. Uveitis.
  - ii. Scleritis.
  - iii. Episcleritis.
- d. Hepatobiliary disease:
  - i. Primary sclerosing cholangitis (PSC).
- e. Other extraintestinal complications:
  - i. Thromboembolic (venous and arterial).
  - ii. Metabolic bone diseases.
  - iii. Osteonecrosis.
  - iv. Cholelithiasis.
  - v. Nephrolithiasis.

12. Other immune-mediated diseases associated with CD include asthma, chronic bronchitis, pericarditis, psoriasis, celiac disease, rheumatoid arthritis, and multiple sclerosis.

SUBJECTIVE DATA

A. Ask about onset, duration, and course of symptoms. Have any of the presenting symptoms occurred at any time in the past (flares of CD may have gone undiagnosed in the past)?

B. Review the client's history and extent of diarrhea, including frequency, consistency, color, quantity, and odor of stools. Evaluate if there is blood, mucus, pus, or food particles in the stools.

C. Inquire about recent travel to foreign countries.

D. Ask the client if diarrhea represents a change in bowel habits. Is there nocturnal diarrhea?

E. Ask the client what makes the diarrhea worse or better.

F. Inquire about previous GI surgery.

G. Review the client's usual weight and any history of weight loss. If weight loss has occurred, how many pounds? How is the client's appetite?

H. Review family history of CD, colon cancer, UC, and malabsorption syndrome.

I. How has the duration of current complaints affected the client's work or usual social activities?

J. Review for duration and extraintestinal symptoms, including the following:
- **1.** Urinary complications: renal calculi.
- **2.** Sclerosing cholangitis: fatigue and jaundice.
- **3.** Skin diseases:
  - a. Erythema nodosum: painful, tender, raised, purple lesion on the tibia.
  - b. Pyoderma gangrenosum: inflamed patch of skin that has progressed to ulceration.
  - c. Herpetic lesions related to immune suppression.
- **4.** Arthritic symptoms.

K. Review medications, especially antibiotics and NSAIDs.

L. Review the client's current tobacco/cigarette use.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure (BP). Measure height and weight to calculate body mass index (BMI).

B. Inspect:
- **1.** Observe general appearance, noting pallor, wasting, cachectic appearance, ecchymosis, skin ulcerations, jaundice, and signs of Kaposi sarcoma.
- **2.** Inspect the head and neck for aphthous ulcers, glossitis, stomatitis, and poor dentition.
- **3.** Inspect the abdomen for surgical scars.
- **4.** Order eye examination for uveitis.
- **5.** Inspect joints for warmth and redness.

C. Auscultate:
- **1.** Auscultate the abdomen for altered bowel sounds (obstruction).

D. Palpate:
- **1.** Palpate the neck for goiter and lymphadenopathy.
- **2.** Palpate the abdomen for distension, ascites, tenderness rebound, guarding, and masses.
- **3.** Palpate for hepatomegaly in RLQ.
- **4.** Palpate the joints for tenderness.

E. Rectal exam:
- **1.** Check anal sphincter for tags, control, and discharge.
- **2.** Palpate for masses, fissures, fistulas, and inflammation.
- **3.** Perform digital rectal examination to assess for anal strictures and rectal mass.

F. Neurologic exam:
- **1.** Assess for signs of vitamin B_{12} deficiency, including tingling sensation and numbness in hands or feet.

DIAGNOSTIC TESTS

A. Laboratory tests:
- **1.** Complete blood count (CBC) with differential.
- **2.** Electrolytes and albumin.
- **3.** Erythrocyte sedimentation rate (ESR)/C-reactive protein (CRP).
- **4.** Serum cobalamin (vitamin B_{12}).
- **5.** Serum iron studies.
- **6.** Folate.
- **7.** Liver enzymes and functioning tests (international normalized ratio [INR]) and bilirubin.
- **8.** HIV.
- **9.** Celiac antibody testing should be considered.
- **10.** Thiopurine methyltransferase (TPMT) activity should be assessed before AZA or MP.

B. Stool studies:
- **1.** Fecal calprotectin should be considered to help differential the presence of IBD from irritable bowel syndrome (IBS).
- **2.** Guaiac.
- **3.** Stool culture.
- **4.** *Clostridioides difficile* toxin assay.
- **5.** Ova and parasites.

C. Imaging:
- **1.** Abdominal flat plate radiography; assess dilated loops of bowel.
- **2.** Barium enema (BE):
  - a. Classic "string sign": narrow band of barium flowing through an inflamed or scarred area in terminal ileum; differentiates CD from UC.
  - b. "Rectal sparing": suggests CD in the presence of inflammatory changes in other parts of the colon.
  - c. "Thumbprinting": indicates mucosal inflammation (may be seen on flat plate of abdomen).
  - d. "Skip lesions": areas of inflammation with normal-appearing areas.
- **3.** Small bowel follow-through GI series.
- **4.** Fistulogram: used to guide the surgical correction.
- **5.** CT or MRI scan of the abdomen and pelvis (limited use in IBD but may detect fistulae).

D. Procedures with/without biopsy:
- **1.** Colonoscopy: Mucosa has characteristic cobblestone appearance. ▶

2. Flexible sigmoidoscopy with biopsy reveals "skip areas" in colon, significant small bowel involvement, fistulas, and granulomas.

3. Upper endoscopy: Aphthous ulcerations occur in the stomach and duodenum in 5% to 10% of clients.

4. Capsule enteroscopy: The major risk is the potential for the camera to become lodged at the point of stricture and requires operative intervention.

5. Skin biopsy.

E. Tuberculin purified protein derivative (PPD) skin test: prior to initiation of biologic therapy.

DIFFERENTIAL DIAGNOSES

A. CD.
B. UC.
C. Appendicitis.
D. Colon cancer.
E. IBS.
F. Anorexia nervosa.
G. Perianal abscess.
H. Intestinal protozoan and bacterial etiologies.
I. Food poisoning.
J. Cytomegalovirus (CMV).
K. Intestinal tuberculosis (TB).
L. Diverticulitis with colitis.

PLAN

A. General interventions:

1. CD should be managed jointly with a gastroenterologist, colorectal surgeon, and specialists such as a rheumatologist, pharmacist, and nutritionist.

B. Client teaching: *See Client Teaching Guide for this chapter, "Crohn's Disease and Ulcerative Colitis."*

1. Smokers should be counseled to stop smoking. Active smoking cessation programs should be encouraged. *See Client Teaching Guide for* Chapter 25, *"Nicotine Dependence."*

C. Dietary management:

1. Adequate nutrition is critical to promotion of healing. Sufficient protein and calories limit the stress on an inflamed and often strictured bowel.

2. Clients with cramps and diarrhea should alter the fiber content of their diets. Diet should include high fiber, low fat (see Appendix B, "Dietary Recommendations").

3. Those with steatorrhea benefit from decrease in fat intake to less than 80 g/d. Give the client a copy of the low-fat/low-cholesterol diet (see Appendix B, "Dietary Recommendations").

4. An empiric trial of restricting milk products may terminate diarrhea due to lactase deficiency.

5. Clients with severe diarrhea may require partial bowel rest, which removes the stimulus that food has on bowel motility and secretion.

6. Elemental diet preparations, such as Ensure, Sustacal, or Isocal, have been found to induce remission, improve symptoms, and decrease disease activity in clients with acute disease.

7. Total parenteral nutrition (TPN) is used when the client's oral intake is not adequate or when surgery is indicated.

8. Pretreatment screening for TB, using Mantoux (a PPD) skin testing, is needed prior to initiation of immunomodulators and thiopurines.

D. Immunization status:

1. International consensus guidelines recommend vaccinating clients on or likely to begin immunosuppressive therapy for control of their IBD.

2. Immunizations with inactivated vaccines should be brought up to date and rigorously maintained during treatment, including a yearly inactivated influenza, meningococcus, hepatitis B if not immune, and pneumococcal vaccine given periodically (5-yearly).

3. Check varicella titers prior to treatment with immunomodulators and reimmunize if titers are low.

4. If required, live vaccines (polio, rubella, and yellow fever) should be given at least 3 weeks before immunosuppressive therapy or 3 months after stopping the therapy.

E. Medical and surgical management.

1. Surgery does not cure the client and is reserved for intractable disease, perforation, obstruction, or severe bleeding.

2. The objective of surgery is to remove grossly involved bowel and to spare as much normal-appearing bowel as possible.

3. Postoperative recurrence rates are estimated at 30% to 50% per decade and are inversely related to preoperative disease duration.

F. Pharmaceutical therapy. The medical management of CD can be divided into treatment of mild Crohn's and moderate to severe Crohn's and further subdivided into management of an acute exacerbation and maintenance of remission. In acute exacerbation, triggers such as underlying infection, fistula, perforation, and other pathology must be ruled out prior to the intravenous (IV) administration of glucocorticoids. The goal of chronic therapy is the remission of bowel inflammation. Therapies include the following:

1. Vitamin, mineral, and folic acid supplements are necessary for proper healing and avoidance of secondary complications, such as bone disease and anemia:

a. Client should take a multiple vitamin containing about five times the normal daily vitamin requirements.

b. Sulfasalazine is effective in treating symptoms of CD. Folic acid supplementation is required for clients on sulfasalazine because it impairs folic acid absorption.

c. Vitamin B_{12} replacement is required for clients who have ileal surgery.

d. Vitamin D, 4,000 international unit (IU), is required for clients with steatorrhea.

2. Loperamide is recommended for symptomatic relief of diarrhea that occurs either prior to maintenance of remission or during acute flares only for mild CD.

3. Stepwise medication approach (Table 14.6):

a. Aminosalicylates (5-ASA) are a mainstay of therapy for mild CD due to their anti-inflammatory activities and rapid absorption throughout the small intestines. Several formulations are available for targeting a specific region of the bowel.

b. Corticosteroids are used if IBD fails to respond to 5-ASA. Corticosteroids should be tapered as rapidly as possible as they do not have a role in maintaining remission. The 2018 AGA guidelines recommend controlled ileal release budesonide at a dose of 9 mg once daily and is effective and should be used for induction of symptomatic remission for clients with mild-to-moderate ileocecal CD. However, budesonide should not be used to maintain remission of CD beyond 4 months.

c. Immunomodulatory agents may be initiated for moderate or severe Crohn's refractory to corticosteroids or in Crohn's that exhibits frequent flares that require steroids. These agents require monitoring of ▶

TABLE 14.6 MEDICATIONS FOR CROHN'S DISEASE AND ULCERATIVE COLITIS

5-ASA: anti-inflammatory activities and rapid absorption throughout the GI tract			
		Induction Therapy	**Maintenance Therapy**
Sulfasalazine (Azulfidine)	Treatment of mild to moderate UC, and as adjunctive therapy in severe UC; converted to mesalamine in the colon	**UC and CD** Initial 1–2 g/d in divided doses Gradual increase to 3–4 g/d in divided doses	**UC** For prolongation of the remission period between acute attacks 2–4 g/d in divided doses
Balsalazide (Colazal)	Mild to moderately active CD Release delayed until the terminal ileum and cecum; converted to mesalamine in the colon	**UC and CD** 6.75 g/d in divided doses; duration 8–12 weeks	**UC and CD** Safety and effectiveness not established beyond 12 weeks
Mesalamine (Asacol)	Targeted for release in the distal ileum and colon	**UC and CD** 2.4–4.8 g/d in divided doses	**UC and CD** Maintenance: 2.4–4.8 g/d in divided doses
Mesalamine (Pentasa)	Mild to moderate disease; released in the duodenum to the distal colon	**UC and CD** 4 g/d in divided doses for up to 8 weeks	**UC and CD** 2–4 g/d in divided doses
Mesalamine (Apriso)	Locally acting aminosalicylates 24-hour delayed and extended-release; release occurring at a pH ≥ 6, then prolonged throughout the colon	**UC** 1.5–3 g/d in divided doses	**Maintenance of Remission of UC** 1.5 g/d
Mesalamine suppository (Canasa)	Specific to the rectum and distal colon	**Ulcerative Proctitis Rectal Suppository** One 500 mg suppository rectally BID OR one 1,000 mg rectally at night; suppositories to be retained 1–3 hours to achieve maximal benefit	**Maintenance of Remission of Ulcerative Proctitis Rectal Suppository** One 500 mg suppository rectally BID OR one 1,000 mg rectally at night; suppositories to be retained 1–3 hours to achieve maximal benefit
Mesalamine retention enema (Rowasa)	Specific to the rectum and distal colon	**Retention Enema** 60 mL (4 g) at bedtime, retained overnight (8 hours)	**Retention Enema** 60 mL (4 g) at bedtime, retained overnight (8 hours)
Corticosteroids: nonspecifically suppress the immune system; mainstay of treatment for active flares			
Budesonide (Uceris; Entocort EC).	Treatment of mild to moderate CD involving the ileum and/or ascending colon; released in the distal small intestine and right colon	**UC and CD** 9 mg daily for up to 8 weeks; TAPER to 6 mg daily for 2 weeks prior to complete cessation; for recurring episodes of active CD, a repeat 8 week course can be given	**UC and CD** Maintenance of clinical remission of mild to moderate CD of ileum and/or ascending colon 6 mg/d up to 3 months; maintenance treatment beyond 3 months with no proven substantial clinical benefit
Prednisone (most common oral steroid used) Methylprednisolone (IV)	Used in acute treatment but are not preventive; IV steroids used in clients with severe disease that requires hospitalization	**UC and CD** Varied dosage and length of treatment. Steroids not to be stopped abruptly; tapering should begin concomitantly with initiating other treatments to maintain remission	**UC and CD** Not for maintenance therapy
		Induction Therapy	**Maintenance Therapy**
Hydrocortisone enemas (Cortenema) Hydrocortisone acetate foam and steroid suppositories (Cortifoam, ProctoFoam-HC)	Colonic disease: proctitis requiring topical/rectal formulations.	**UC and CD Enema**: one nightly at HS; usual length of treatment 2–3 weeks **Suppository**: one BID (a.m. and p.m.); severe proctitis may require using a suppository TID or using two suppositories BID **Foam**: one applicator once daily or Q12H for 2–3 weeks, then one applicator every other day if needed	**UC and CD** Dosage and length of treatment based on medical treatment and response to treatment

(*continued*)

TABLE 14.6 MEDICATIONS FOR CROHN'S DISEASE AND ULCERATIVE COLITIS (*CONTINUED*)

Immunomodulatory Agents: • **Thiopurines (azathioprine, 6-MP are effective and should be considered for use for steroid sparing in CD. Both are effective therapies and should be considered for treatment of CD for maintenance of remission (2018 AGA recommendations).** • **AGA recommends using MTX should be considered for alleviating signs and symptoms in clients with steroid-dependent CD and for maintaining remission. Consensus opinion currently does not favor using MTX as monotherapy.**			
6-MP (Purinethol)	Slow-onset: onset of therapy noted after 3–6 months	**CD** Weight-based dosage and response to therapy 1.5 mg/kg/d	**CD** Weight-based dosage and response to therapy 1.5 mg/kg/d
Azathioprine (Imuran): used off-label for UC and CD	Converts to its active form 6-MP; slow-acting and can take up to 3 months to work	**UC and CD** Weight-based dosage and response to therapy 2.5 mg/kg/d.	**UC and CD** Weight-based dosage and response to therapy 2.5 mg/kg/d
MTX folic acid antagonist	Improvement generally in 3–6 weeks; however, full benefit may not be seen for 12 weeks	**CD** 15–25 mg once a week by injection; clients also need to take folic acid 1 mg/d	**CD** 15 mg per week; clients also need to take folic acid 1 mg/d
Biologics: TNF-Alpha Blocker • **The AGA states that using combination therapy of infliximab with immunomodulators (thiopurines) is more effective than treatment with either immunomodulator monotherapy or infliximab monotherapy in clients who are naïve to those agents.** • **The AGA recommends using anti-TNF-alpha for CD refractory to thiopurines and MTX.** • **The AGA recommends anti-TNF-alpha be used to treat CD that is resistant to treatment with corticosteroids.**			
Infliximab (Remicade) IV infusion	Moderate to severe CD with fistulizing CD or resistance to steroids and conventional therapy	**CD** 5 mg/kg IV over 2 hours at 0, 2, and 6 weeks	**CD** 5 mg/kg every 8 weeks thereafter; if no response by week 14, consider discontinuing therapy
Antiadhesion Molecule			
Natalizumab (Tysabri) Distribution restricted and available only through a close monitored program known as TOUCH	Moderate to severe CD for clients who failed anti-TNF therapy	**CD** 300 mg every 4 weeks; given by IV infusion over 1 hour; discontinuation after 12 weeks based on individual response/ protocol	**CD** 300 mg IV monthly; restricted distribution
Antibiotics: used for treatment of bacterial infections that cause abscesses and can be helpful in treatment of fistulas			
Ciprofloxacin (Cipro)		**UC and CD** Varied dosage and length of treatment	**UC and CD** Varied dosage and length of treatment
Metronidazole (Flagyl)		**UC and CD** Varied dosage and length of treatment	**UC and CD** Varied dosage and length of treatment

5-ASA, aminosalicylates; 6-MP, 6-mercaptopurine; AGA, American Gastroenterology Association; CD, Crohn's disease; GI, gastrointestinal; HS, at bedtime; IV, intravenous; MTX, methotrexate; TNF, tumor necrosis factor; UC, ulcerative colitis.

Source: Adapted from the 2018 American College of Gastroenterology Clinical Guideline: Management of Crohn's Disease in Adults. http://gi.org.

blood counts due to hematologic toxicity. A 3% incidence of pancreatitis, allergic reactions, infections, and marrow toxicity is associated with their use. The main drawback to the use of AZA and 6-MP is their slow onset. The effect of therapy is noted after 3 to 6 months of treatment. The 2018 AGA guidelines recommend TPMT testing should be considered before initial use of AZA or 6-MP.

d. Biologic agents: Tumor necrosis factor (TNF)-alpha blocker: initial biologic. Infliximab and adalimumab are indicated for both remission induction and maintenance of remission:

i. Infliximab (Remicade).
ii. Adalimumab (Humira).
iii. Certolizumab: not indicated to induce remission.
iv. Anti-interleukin 12/23: ustekinumab (Stelara): alternative to TNF-alpha blocker.
v. Anti-integrin.
vi. Vedolizumab (Entyvio): useful in clients over 60.
vii. Natalizumab (Tysabri).

e. Antibiotics are utilized in adults for management of perianal disease or inflammatory mass (metronidazole, ciprofloxacin). Metronidazole and ciprofloxacin should ▶

not be used as primary therapy for luminal inflammatory CD.

f. **NSAIDs may exacerbate disease activity and should be avoided when possible.**

FOLLOW-UP

A. Have the client record their weight daily to monitor changes.

B. Assess frequency and consistency of stools to evaluate volume losses and effectiveness of therapy.

C. Instruct clients on self-medication and to call a physician if fever develops, diarrhea worsens, bleeding occurs, or abdominal pain becomes marked.

D. Monitoring clients on 5-ASA should include CBC at least twice a year and urinalysis at least annually.

E. Monitoring clients on 6-MP requires frequent monitoring, including CBC and aminotransferase levels (alanine transaminase [ALT] and AST) before treatment, and again at 2, 3, 8, and 12 weeks after initiating therapy. When stable, monitor every 3 months thereafter, and 2 to 3 weeks after a change in dosage.

F. Monitoring clients on methotrexate (MTX) includes a CBC and aminotransferases as with 6-MP/thiopurine therapies.

G. Periodic bone mineral density assessment is recommended for clients on long-term corticosteroid therapy (longer than 3 months). Osteopenia should be treated aggressively. The primary intervention includes dietary counseling and supplementation to ensure adequate intake of vitamin D and calcium.

H. Annual ophthalmologic examinations are recommended for clients on long-term corticosteroids.

I. Clients who are using corticosteroids should be monitored for glucose intolerance and other metabolic abnormalities.

CONSULTATION/REFERRAL

A. Refer the client to a gastroenterologist initially for evaluation. Treatment requires multidisciplinary management with a gastroenterologist and other subspecialists, including a nutritionist, surgeon, rheumatologist, ophthalmologist, and social workers.

B. Promptly hospitalize for parenteral management clients who are toxic, bleeding heavily, in severe pain, or too sick to obtain adequate nutrition orally.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Active disease at the time of conception is associated with increased incidence of miscarriage and postpartum exacerbation. It may also predispose the client to other maternal and prenatal risks, such as premature labor, small-for-gestational-age babies, and stillbirth.

2. Sulfasalazine and steroids are safe and effective during nursing or pregnancy.

3. Clients on prednisone should receive supplementary steroids during labor and delivery, as well as during other highly stressful times.

4. Counsel clients to attempt pregnancy only when the disease has been quiescent for several months.

5. Withholding sulfasalazine for 2 to 3 days before delivery may be advisable to minimize neonatal jaundice due to bilirubin displacement.

6. MTX is a U.S. Food and Drug Administration (FDA) category X drug.

B. Geriatrics:

1. Unexplained diarrhea, weight loss, and perianal disease in older adults should arouse suspicions regarding CD. Older adults have worse outcomes due to delayed presentation and comorbid conditions. The older adult is more likely to be more malnourished, anemic, and hypovolemic with higher transfusion requirements and with longer postoperative hospital stays especially after surgery. Dehydration is more common in older adults. Signs of dehydration include confusion, muscle weakness, fever, dizziness, poor skin turgor, hypotension, and tachycardia.

2. Diagnostic delay is more common in older adults. The differential diagnosis for older clients includes the following:

a. Diverticulitis and diverticular bleeding.
b. Ischemic colitis.
c. Microscopic colitis.
d. Infectious colitis.
e. NSAID-induced ulcerations, strictures, and perforation.
f. Radiation colitis (most commonly gynecologic, rectal, and prostate cancers).

3. Older adults tend to have CD confined to the distal colon, with only 40% having proctitis.

4. With an increase in cancers in older adults with CD, it is imperative to evaluate and exclude cancer before beginning immunosuppressant or biologic therapies. Older clients with CD are at increased risk for cancers, including those cancers found in the following:

a. Small intestines.
b. Pancreas.
c. Endocrine glands.
d. Kidney.
e. Stomach.
f. Lung and non-Hodgkin lymphoma (NHL) and nonmelanoma skin cancer.

5. Review CD therapies, with comorbid conditions in mind, due to side effects, interactions between medications, need for increased laboratory monitoring (e.g., INR as well as digoxin levels and phenytoin levels), and risk of infections.

6. Anti-TNF-alpha agents are contraindicated in clients with New York Heart Association (NYHA) class III and IV heart failure (HF).

7. Age is currently not a contraindication to performing an IPAA. Older adults with good anal sphincter function and adequate cognitive ability are better surgical candidates for the IPAA procedure.

8. The incidence of a deep vein thrombosis (DVT) is higher in older adults.

9. Due to polypharmacy a full review of medications, herbals, and over-the-counter (OTC) drugs should be undertaken to identify medications that should be avoided in the geriatric population, duplicate medications, and medications that are able to be decreased or deprescribed. (Refer to section on deprescribing in Chapter 3, "Adult-Geriatric Assessments.")

RESOURCE

Crohn's and Colitis Foundation of America: www.ccfa.org.

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CYCLOSPORIASIS

DEFINITION

A. Cyclosporiasis is a one-cell parasite that infects the upper small intestines. Causes of cyclosporiasis include consuming infected water or produce (fresh fruits, especially raspberries and vegetables) or exposure to the organism during travel to countries where it is endemic.

B. It manifests as protracted and relapsing gastroenteritis. The clinical syndrome consists of explosive watery diarrhea, nausea, anorexia, weight loss, fatigue, and abdominal cramps that may persist for 7 days to several weeks, with a waxing and waning course.

C. In an immunocompromised host, onset is insidious and the condition becomes chronic; the shedding of oocysts continues indefinitely. Symptoms associated with cyclosporiasis are more severe in HIV/AIDS clients.

D. The oocysts are resistant to most disinfectants used in food and water processing and can remain viable for prolonged periods.

INCIDENCE

A. Cyclosporiasis affects approximately 16,000 persons per year in the United States among 76 million cases of foodborne diarrhea. It is endemic in other regions of the world.

B. Most outbreaks in the United States and Canada have been associated with consumption of imported fresh produce. Most cases are diagnosed between May and August. Reported cases increased between summer 2016 and summer 2017.

C. It affects all ages.

D. In the United States, one-third of cases occur in international travelers, one-third of cases are acquired without travel, and one-third occur without known risk factors.

E. The highest incidences are in Guatemala and Peru, with other countries in Central America, South Americana, Asia, and the Caribbean also contributing to reported cases among travelers.

PATHOGENESIS

A. Infection is caused by an 8- to 10-μm spore-forming coccidian protozoan called *Cyclospora cayetanensis*. Transmission of oocysts is by the oral–fecal route. The incubation period ranges from 2 days to 2 weeks after excretion, depending on temperature and humidity.

PREDISPOSING FACTORS

A. Incompetent or compromised immune system (e.g., infection with HIV).

B. Travel to underdeveloped or tropical countries.

C. Ingestion of contaminated food or water.

D. Contact with animals that carry the parasite.

COMMON COMPLAINTS

A. Abrupt, profuse, malodorous, watery diarrhea.

B. Nausea.

C. Vomiting.

D. Anorexia.

E. Substantial weight loss.

F. Flulike symptoms.

G. Abdominal cramps and bloating.

OTHER SIGNS AND SYMPTOMS

A. Asymptomatic: common among individuals residing in endemic regions.

B. Low-grade fever.

C. Nausea and vomiting.

D. Profound fatigue.

E. Yellow-to-khaki-green stools.

F. Flatus.

G. Dehydration.

SUBJECTIVE DATA

A. Review onset, duration, and course of symptoms. Is diarrhea acute or chronic?

B. Question the client about travel to areas known for *Cyclospora*, such as Guatemala, Haiti, Puerto Rico, Pakistan, India, Mexico, Nepal, New Guinea, and Peru.

C. Review the client's intake of medications and other substances that can cause diarrhea, especially antibiotics, laxatives, quinidine, magnesium-containing antacids, digitalis, loop diuretics, antihypertensives, alcohol, caffeine, herbal teas, and sorbitol-containing (sugar-free) gum and mints.

D. Ask about the nature of the client's bowel movements (BMs), including frequency, consistency, volume, and presence of blood, pus, or mucus.

E. Review associated symptoms that need evaluation: fever, abdominal pain, and anorexia.

F. Ask the client if other family members or sexual contacts are also ill.

G. Establish the client's normal weight and any recent weight loss. How much weight was lost and over what period of time?

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure (BP; vital signs are normal in most cases), and height and weight to calculate body mass index (BMI).

B. Inspect:

1. Inspect general appearance for signs of illness and dehydration:
   a. Inspect mucous membranes.
   b. Note the presence of decreased skin turgor.

C. Auscultate:

1. Auscultate the abdomen for bowel sounds in all quadrants.

D. Palpate:

1. Palpate the abdomen for masses, rebound tenderness, guarding; may exhibit right upper quadrant (RUQ) pain (biliary disease).
2. Palpate lymph nodes for enlargement.

E. Rectal exam.

DIAGNOSTIC TESTS

A. Identification may be made by microscopic examination of a stool preparation by modified acid-fast staining, fluorescence microscopy, or review of wet mounts of stool by experienced microscopists. Finding large numbers of white cells suggests an inflammatory or invasive diarrhea. The following tests are done:

1. Acid-fast Ziehl–Neelsen stained slide of stool: 8 to 10 micron *Cyclospora* oocysts are visible.
2. Stool culture for ova and parasites: Parasites are passed intermittently, so three or more stools on alternating days should be examined. Specific requests to assess for *Cyclospora* is necessary.
3. Endoscopy with small bowel biopsy.

DIFFERENTIAL DIAGNOSES

A. Cyclospora infection.

B. Giardiasis.

▶

C. Malabsorption.
D. *Escherichia coli* infection: *E. coli* causes diarrhea within hours of ingesting contaminated food. Confirm by checking if others were affected.
E. Irritable bowel syndrome (IBS): Leukocyte-free mucus is the hallmark of IBS.
F. Viral diarrhea.
G. Lactose intolerance.
H. Other bacterial infections, for example, *Shigella, Salmonella*, and *Campylobacter.*
I. Cholera.
J. Inflammatory bowel disease (IBD; Crohn's disease (CD) or ulcerative colitis [UC]).
K. Cryptosporidium: retain acid-fast stain but are smaller, only 5 microns.

PLAN

A. General interventions:
1. Advise the client to tell household members and sexual contacts to seek medical examination and treatment.
2. Children and caregivers with diarrhea should be excluded from childcare centers until they become asymptomatic.
3. Fresh produce should always be washed thoroughly before it is eaten.
B. Client teaching: *See Client Teaching Guide for this chapter, "Diarrhea."*
1. Discuss safe sexual practices.
2. Teach contact precautions to those caring for diapered and/or incontinent children.
C. Dietary management:
1. Tell the client to increase fluids. Fluid replacement is the basic approach to prevent dehydration from diarrhea.
2. Tell the client to restrict milk products to rule out lactose intolerance.
3. Give the client a copy of a diet to control nausea and vomiting. See Appendix B, "Nausea and Vomiting Diet Suggestions."
D. Pharmaceutical therapy:
1. The drug of choice is trimethoprim-sulfamethoxazole (TMP-SMZ). It can reduce shedding and stop diarrhea within 2 days:
a. Adults:
i. Immunocompetent host: TMP 160 mg/SMZ 800 mg tablet orally BID for 7 to 10 days.
ii. Immunocompromised host: TMP 160 mg/SMZ 800 mg tablet orally QID for 10 days, followed by prophylaxis with TMP 160 mg/SMZ 800 mg orally three times per week.
2. For sulfa allergic clients: nitazoxanide (500 mg BID for 7 days)
3. Ciprofloxacin (Cipro): Treatment failures have been reported:
a. Adults: Cipro 500 mg orally BID for 7 to 10 days for acute infection.
b. Adults: prophylaxis in HIV: cipro 500 mg orally three times a week.

FOLLOW-UP

A. See the client in 1 week to verify continuing clinical improvement.
B. If diarrhea persists 2 weeks or more, a second evaluation is indicated.
C. Report cases of cyclosporiasis to the health department.

CONSULTATION/REFERRAL

A. Consult an infectious disease specialist and/or gastroenterologist if the client has no symptom relief after completing therapies or has a prolonged or severe case.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. TMP-SMZ is a pregnancy category C drug. Use during pregnancy if the potential benefit outweighs the risk to the fetus.
2. TMP-SMZ should be avoided near term due to the potential for hyperbilirubinemia and kernicterus in the newborn.
B. Geriatrics:
1. Use of bactrim in the geriatric adult should coincide with monitoring of renal function and after evaluation of med list to ensure no interactions.

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DIARRHEA

DEFINITION

A. Diarrhea is characterized by an abnormally high fluid content in the stool. Generally, diarrhea also involves an increase in the frequency of bowel movements (BMs), which can range from four to five to more than 20 times a day. Diarrhea may be an acute onset or chronic/persistent diarrhea.
1. Acute diarrhea is usually self-limited; the most common complication of diarrhea is dehydration and malabsorption.
2. Chronic diarrhea is defined as lasting longer than 14 days.

INCIDENCE

A. The incidence of diarrhea is unknown.
B. The incidence of *Clostridioides difficile* infection is approximately 7%, and 28% of clients who were hospitalized have positive cultures for the organism. *C. difficile*-associated diarrhea has a mortality rate as high as 25% in the frail older adults.
C. Be aware that *C. difficile* can occur among clients in the community who have been treated with antibiotic therapy or antineoplastic agents with antibacterial activity.

PATHOGENESIS

A. The increased water content in diarrhea stools is due to an imbalance in the physiology of the small and large intestinal processes. See Table 14.7 for organisms that cause diarrhea.

PREDISPOSING FACTORS

A. Enteric infections.
B. Females have a higher incidence of *Campylobacter species* infections.
C. Institutional: day care and skilled nursing facilities.
D. Food: raw or contaminated food.
E. Contaminated water or inadequate chlorinated water supply.
F. Travel.
G. Chemotherapy or radiation induced.
H. Vitamin deficiencies (niacin and folate).

TABLE 14.7 ORGANISMS THAT CAUSE DIARRHEA

Viral organisms	Rotavirus
	Norovirus
	Adenovirus
	Calicivirus
	Astrovirus
	Ebola
Invasive bacteria	*Escherichia coli*
	Klebsiella
	Clostridioides difficile
	Clostridium perfringens
	Shigella
	Salmonella
	Campylobacter
	Cholera
	Yersinia
	Plesiomonas
	Aeromonas
Parasites	*Giardia*
	Entamoeba organisms
	Cryptosporidium
	Giardia lamblia

I. Vitamin toxicity (C, niacin, and vitamin B_3).
J. Ingestion of heavy metals (copper, tin, or zinc) or toxins.
K. Ingestion of plants, mistletoe, or mushrooms.
L. Antibiotics.
M. Immunodeficiency.

COMMON COMPLAINTS

A. Frequent watery stool.
B. Foul-smelling stools (fat malabsorption).
C. Flatulence.
D. Abdominal cramping.

OTHER SIGNS AND SYMPTOMS

A. Lethargy.
B. Fever.
C. Nausea and vomiting.
D. Currant jelly stool (blood and mucus).
E. Anorexia.
F. Dehydration in adults:
  1. Thirst.
  2. Less frequent urination.
  3. Dark urine.
  4. Dry skin.
  5. Fatigue.
  6. Dizziness.
  7. Light-headedness.

SUBJECTIVE DATA

A. Review the onset of diarrhea stools. What is the normal stool pattern?
B. Review the consistency, color, volume, and frequency of the stools.
C. Review dietary intake of raw foods, contaminated food, and nonabsorbable sugars, including lactulose or lactose in lactose malabsorbers. Review the use of artificial sweeteners such as sorbitol.
D. Review any contact with others who may have the same symptoms.
E. Have any of the stools contained blood?
F. Review travel history, including camping vacations.
G. Review medication history, including vitamins, herbal production, laxatives, antacids that contain magnesium, opiate withdrawal, Olestra, and methylxanthines (caffeine, theobromine, and theophylline):
  1. Specifically inquire about the use of antibiotics in the previous 2 to 3 months (*C. difficile* may develop as late as 10 weeks after stopping antibiotics).
H. Review any food allergies and history of lactose intolerance.
I. Evaluate the presence of other symptoms such as fever and abdominal pain.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure (BP; standing and sitting), and weight.
B. Inspect:
  1. Observe the client's general overall appearance, the presence of lethargy or depressed consciousness, or grimace during exam.
  2. Evaluate muscle tone, skin turgor, reduced muscle, and fat mass.
  3. Examine mouth, lips, and mucous membranes for signs, symptoms, and severity of dehydration.
  4. Perianal examination for skin breakdown, erythema, and fissures.
C. Auscultate:
  1. Assess heart and lungs.
  2. Auscultate the abdomen in all four quadrants.
  3. Assess the presence of borborygmi (significant increase in peristaltic action that may be audible and/or palpable).
D. Percuss:
  1. Percuss abdomen.
E. Palpate:
  1. Palpate the abdomen for masses, guarding, rebound tenderness, and peritoneal signs.
  2. Palpate for lymphadenopathy.
F. Rectal examination:
  1. Perform a rectal examination, including testing of stool for occult blood.

DIAGNOSTIC TESTS

A. Stool specimens for evaluation of the following:
  1. *C. difficile*: reserved for clients having more than three loose stools per 24 hours and/or with risk factors including recent hospitalization, use of antibiotics, or advanced age:
    a. Enzyme immunolinked assay (EIA) tests: glutamate dehydrogenase (GDH) antigen and toxin detection, which detects toxin A, toxin B, or both.
    b. Nucleic acid amplification test (NAAT): polymerase chain reaction (PCR) test that detects presence of pathogenic strain of *C. difficile*.

2. Fecal leukocytes.
3. Blood tests:
a. Complete blood count (CBC): White blood cells (WBCs) may be elevated.
b. Electrolytes.
c. Albumin.
4. Bacterial culture.
5. Ova and parasites.
6. Giardia antigen.
7. Fecal calprotectin: indicates colonic inflammation.
8. Fecal alpha-1 antitrypsin levels: useful in chronic diarrhea.
9. Specific lab requests must be made to identify *Campylobacter, Aeromonas*, and *Yersinia. Listeria* should be suspected with outbreaks of nonbloody, febrile diarrhea in the setting of negative stool testing.

B. A colonoscopy for intestinal biopsy for chronic or protracted diarrhea or clients with AIDS should be done. A sigmoidoscopy alone may not reveal any abnormality.
C. Abdominal ultrasound to identify intussusception.
D. Abdominal CT.
E. Hydrogen breath test for lactose/fructose intolerance.

DIFFERENTIAL DIAGNOSES

A. Diarrhea: infectious etiology.
B. Inflammatory bowel disease (IBD):
1. Crohn's disease (CD).
2. Ulcerative colitis (UC).
C. Cystic fibrosis (CF).
D. Giardiasis.
E. Protozoan.
F. Malabsorption syndromes.
G. Intussusception.
H. Stool impaction.
I. Irritable bowel syndrome (IBS).
J. Meckel diverticulum.
K. Intolerance to lactose, carbohydrates, and protein.
L. Antibiotic-associated diarrhea.
M. Pseudomembranous colitis.
N. Toxic megacolon.
O. Appendicitis.
P. Celiac disease.

PLAN

A. Client teaching: *See Client Teaching Guide for this chapter, "Diarrhea."*
1. Examination of stools for ova and parasites should be done every other day or every 3 days.
2. Rehydration with oral fluids for each diarrhea stool. Administer small amounts at frequent intervals. Solution should contain water, salt, and sugar. Diluted fruit juices or flavored soft drinks, broths, or soups should be sufficient with mild cases of diarrhea. Gatorade-type solutions may be sufficient for mild cases, but for more severe cases oral rehydration solution (Rehydralyte or Ceralyte) should be considered. Intravenous (IV) fluids should be used for severe hypovolemia.
3. Hold foods until hydration is completed. Dietary modifications are poorly validated in studies; however, the use of saltine crackers, boiled vegetables or starches, and bananas is generally supported in clients with nausea, vomiting, or anorexia.
4. Use antibiotics (or the discontinuation of antibiotics in the case of *C. difficile)* or antiparasitic agents, depending on the etiology. Empiric use of antibiotics is not recommended as most cases of diarrhea are viral. However, in cases of severe and/or bloody diarrhea, especially with fever, the benefit of the antibiotic likely outweighs the risk.
5. The use of probiotics, *Lactobacillus GG* (I, A) and *Saccharomyces boulardii* (II, B), has been found to be effective in repopulating or maintaining the colonic gut flora. They may reduce the spread of rotavirus.
6. Encourage proper hygiene and food preparation to prevent spread and future infections.
7. Water should be boiled for at least 1 minute if contamination is suspected.

B. Pharmaceutical therapy:
1. Antiprotozoal: nitazoxanide (Alinia) 500 mg Q12H for 3 days.
2. HIV/AIDS: crofelemer (Fulyzaq) for noninfectious diarrhea in adults on antiretroviral therapy; 125 mg tablet BID.
3. Antidiarrheals.
a. Loperamide (Imodium):
i. Initially 4 mg, then 2 mg after each loose stool. Maximum dose 16 mg/d. Stop in 48 hours if diarrhea is not relieved.
ii. Chronic diarrhea: initially 4 mg, maintenance 4 to 8 mg/d; reevaluate if no improvement in diarrhea in 10 days at 16 mg/d.
b. Bismuth subsalicylate (Kaopectate) 262 mg/15 mL, take 30 mL every 30 to 60 minutes if needed; maximum four doses per day. Slower onset of action compared with loperamide; however, more useful in clients with fever and dysentery.
c. Diphenoxylate HCL (Lomotil) two tablets or 10 mL QID until diarrhea is controlled; maintenance, two tablets or 10 mL per day. Increased risk of cholinergic side effects.
4. *C. difficile* treatment:
a. Stop antibiotics.
b. Avoid antiperistaltic agents.
c. Initial occurrence:
i. Vancomycin 125 mg QID for 10 days. OR
ii. Fidaxomicin (Dificid) 200 mg orally BID for 10 days. OR
iii. Metronidazole 500 mg TID for 10 to 14 days if the other two choices are unavailable.
d. First recurrence:
i. Fidaxomicin 200 mg orally for 10 days, OR 200 mg orally BID for 5 days followed by once every other day for 20 days.
ii. Vancomycin in a tapered and pulsed regimen (i.e., 125 mg orally QID for 10–14 days, followed by 125 mg orally BID for 7 days, followed by 125 mg orally daily, followed by 125 mg orally every 2–3 days for 2–8 weeks) OR 125 mg orally QID for 10 days.
iii. Bezlotoxumab 10 mg/kg IV given once during above as adjunctive treatment.
e. Second and subsequent recurrence:
i. Fidaxomicin: 200 mg PO BID for 10 days OR 200 mg orally BID for 5 days followed by once every other day for 20 days.
ii. Vancomycin in taper/pulsed regimen (see above for example). OR
iii. Vancomycin followed by rifaximin. Vancomycin 125 mg PO QID for 10 days, then rifaximin 400 mg PO TID for 20 days.

iv. Consider fecal microbiota transplantation (FMT).

f. Fulminant disease: any occurrence or recurrence accompanied by hypotension/shock, ileus, or megacolon.

i. Consider consultation with infectious disease and surgical specialist.

ii. Vancomycin 500 mg PO QID AND metronidazole 500 mg IVQ8H.

iii. If ileus present, consider FMT or recall vancomycin.

FOLLOW-UP

A. Follow-up depends on the severity of diarrhea and the age of the client.

B. Leukocytosis of increasing severity in hospitalized clients should prompt a clinical evaluation for *C. difficile* infection.

CONSULTATION/REFERRAL

A. Evaluate the need for a surgical consultation (fulminant colitis, peritonitis, or toxic megacolon) or one with an infectious disease specialist or a gastroenterologist.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Diarrhea rather than constipation is sometimes the only complaint due to the collection of liquid stool distending the proximal colon and passing around the obstructing bolus.

2. Review any hospitalizations within the past 72 hours as a cause of diarrhea. Traditionally *C. difficile* infections occur in an institutional setting; however, community-acquired infection is not uncommon.

3. Advanced age is a risk factor for *C. difficile* infection.

4. Due to polypharmacy, a full review of medications, herbals, and over-the-counter (OTC) drugs should be undertaken to identify medications that should be avoided in the geriatric population, duplicate medications, and medications that are able to be decreased or deprescribed. (Refer to section on deprescribing in Chapter 3, "Adult-Geriatric Assessments.")

5. Beers Criteria caution:

a. Medications for constipation and those for diarrhea are on the American Geriatrics Society 2015 medications to be avoided. Refer to Appendix C, "Beers Criteria."

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DIVERTICULOSIS AND DIVERTICULITIS

DEFINITION

A. Diverticulum is the saclike protrusion of mucosa through the muscular colonic wall. Protrusions occur in weakened areas of the bowel wall. Diverticulosis is the presence of diverticula within the colon, but it does not imply a pathologic condition and may remain asymptomatic. Diverticulitis occurs when the diverticula become plugged and inflamed. Diverticular bleeding occurs due to injury of the blood vessel within the diverticulum and subsequent rupture into the lumen. Diverticular disease occurs over a continuum including simple asymptomatic diverticular disease, uncomplicated symptomatic diverticulitis, and complicated symptomatic diverticulitis. Complicated diverticulitis includes problems arising from abscess, perforation, fistula, peritonitis, and sepsis.

B. An increased risk of developing diverticular disease is associated with a diet that is high in red meat and total fat content. This risk can be reduced by a diet high in fiber content, especially with fruits and vegetables (cellulose; see Appendix B, Table B.7).

C. Diverticulosis is often diagnosed as an incidental finding on a barium enema (BE) or sigmoid/colonoscopy.

D. Recurrent attacks of diverticulitis can result in the formation of scar tissue, leading to narrowing and obstruction of the colonic lumen.

INCIDENCE

A. Diverticulosis is very common and increases with age:

1. Prevalence by age:

a. 20% by age 40.

b. 60% by age 60.

C. No significant difference in prevalence by sex. Male preponderance in diverticular clients less than 50 years old and female preponderance in clients over 70 years of age.

D. Diverticulosis becomes symptomatic in 70% of cases. It leads to diverticulitis in 5% to 25% and is associated with bleeding in 5% to 15%. The sigmoid colon is commonly affected in Western countries, whereas right-sided involvement is more commonly seen among Asian entities.

E. Diverticulitis can be separated into two groups:

1. Simple, with no complications; responds to treatment such as dietary changes without the need for surgery.

2. Complicated, with abscesses, fistula, obstruction, perforation, and peritonitis leading to sepsis; usually requires surgery.

PATHOGENESIS

A. The formation of diverticula is multifactorial. Clients with diverticula have been shown to have higher colonic segmental pressures, which cause the mucosa to herniate through the colonic wall. The increased pressure might be due to upregulation of smooth muscle receptors and, combined with production of abnormal collagen, luminal narrowing, abnormal elastin deposits within the colonic wall, leads to the development of herniation. Diverticulitis occurs due to micro- or macroperforation of the diverticular wall, which is a result of persistent increased pressure and retained food particles that erode the wall. The perforation is followed by inflammation and localized necrosis. Diverticular bleeding occurs when the blood vessel contained within the diverticulum becomes weakened due to structural repositioning and ruptures into the colon lumen.

PREDISPOSING FACTORS

A. Advanced age.

B. Obesity (84%–96%).

C. Dietary factors:

1. Low residue.

2. High red meat and total fat content.

D. Complicated diverticular disease is increased in the following:

1. Smokers.

2. Chronic nonsteroidal anti-inflammatory drugs (NSAIDs) use.

3. Acetaminophen use (especially paracetamol).

E. Physical inactivity.

F. Indication that genetics are a predisposing factor:

1. Left-sided diverticula is predominant in the United States.
2. Right-sided (cecal) diverticula is predominant in Asia.

COMMON COMPLAINTS

A. Diverticulosis is usually asymptomatic.
B. Painless rectal bleeding is the hallmark of diverticular bleeding, with intermittent passage of maroon or bright red blood.
C. Common diverticulitis symptoms:
1. Left lower quadrant (LLQ). pain.
2. Constipation.

OTHER SIGNS AND SYMPTOMS

A. Back pain.
B. Flatulence.
C. Periodic abdominal distension.
D. Borborygmi, or loud, prolonged gurgles caused by hyperactive intestinal peristalsis.
E. Diarrhea.
F. Nausea or vomiting.
G. Dysuria.
H. Tenderness on palpation, possible guarding.
I. Low-grade fever.

SUBJECTIVE DATA

A. Review onset, duration, and course of symptoms, including size, color, consistency, and frequency of stools.
B. Ask the client if constipation is a chronic or acute problem, and if it alternates with diarrhea.
C. Review the client's daily diet and fluid intake.
D. Ask the client about medication use, including iron supplements, NSAIDs, and acetaminophen, steroids, and opiates.
E. Inquire about the color, amount, and frequency of rectal bleeding. Does the client strain when having a bowel movement (BM)?
F. Review the client's history of pain with defecation.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure (BP), and weight.
B. The physical examination may be relatively unremarkable but most commonly reveals abdominal tenderness or a mass.
C. Inspect:
1. Observe the general overall appearance for signs of pain.
2. Inspect the abdomen in detail, assessing for distension.
D. Auscultate:
1. Abdomen. Absent bowel sounds suggest peritoneal inflammation.
E. Percuss:
1. Abdomen.
F. Palpate:
1. Palpate the abdomen for rebound tenderness or masses signaling possible abscess and tenderness.
2. Palpate beneath right costal arch, checking for Murphy sign or pain on deep inspiration.
G. Rectal exam:
1. Evaluate for hemorrhoids, masses, fissures, fistulas, inflammation, and stool in ampulla.

DIAGNOSTIC TESTS

A. Labs:
1. Complete blood count (CBC): Leukocytosis supports the diagnosis of diverticulitis.
2. Comprehensive metabolic profile (CMP): evaluates kidney function, liver enzymes, and electrolytes.
3. Urinalysis: Sterile pyuria may occur.
4. Stool studies are only indicated for clients with diarrhea and are rarely indicated.
5. Human chorionic gonadotropin (HCG) in all females of childbearing age.
6. C-reactive protein (CRP) to assess for inflammation.
7. Stool hemoccult or fecal immunoassay.
B. Imaging:
1. CT contrasted of the abdomen and pelvis.
2. Ultrasonography can show bowel wall thickening, abscess (with/without gas bubbles).
3. MRI is acceptable to avoid radiation.
4. Abdominal radiography shows abnormality in 30% to 50% of symptomatic clients.
5. BE after infection subsides. Caution: A BE during the acute phase may increase intraluminal pressure and cause bowel perforation.
C. Endoscopy:
1. Colonoscopy.
2. Proctosigmoidoscopy.

DIFFERENTIAL DIAGNOSES

A. Diverticulitis.
B. Diverticulosis.
C. Acute appendicitis.
D. Bowel obstruction.
E. Ischemic colitis.
F. Colon cancer.
G. Hemorrhoids.
H. Constipation or impaction.
I. Inflammatory bowel disease (IBD).
J. Urologic disorder: pyelonephritis.
K. Pelvic inflammatory disease (PID).
L. Ectopic pregnancy.

PLAN

A. General interventions:
1. Clients who have evidence of complicated diverticulitis (with perforation, fistula, obstruction, or abscess) need to be admitted to hospital and treated as inpatients. All inpatients are treated with intravenous (IV) fluid support, IV antibiotics, and pain control. Depending on the complication, other interventions may be undertaken by a specialist.
2. Clients who have uncomplicated diverticulitis but one of the following findings should also be treated as inpatients: microperforation, fever over 102.5°F, significant leukocytosis, severe pain, inability to tolerate oral intake, immunosuppression, significant comorbidities, poor support system, or outpatient treatment failure.
3. Clients with uncomplicated diverticulitis and none of the earlier characteristics may be treated as outpatients.
B. Pharmaceutical therapy:
1. Antibiotic therapy should be continued for 7 to 10 days. Choices include the following:
a. Cipro 500 mg Q12H + metronidazole 500 mg Q8H or levaquin 500 mg daily + metronidazole.
b. Bactrim DS one tablet Q12H + metronidazole 500 mg Q8H.
c. Augmentin 875 mg Q12H or Augmentin 500 mg Q8H. ▶

d. Moxifloxacin 400 mg daily if beta-lactam and/or metronidazole not tolerated.

i. Check local resistance patterns. If resistance is greater than 10% to one agent, choose alternative regimen.

C. Dietary treatment:

1. Outpatient diet: There is no evidence to support dietary restrictions during acute episode of diverticulitis.

2. Long-term dietary management:

a. High-fiber diet, including bran, beans, fruits, and vegetables.

b. Bulk agents if unable to tolerate bran.

c. There is no evidence in the research to support the avoidance of foods such as corn, seeds, or nuts.

D. Relapse:

1. Approximately one-third of clients with an initial episode of diverticulitis will experience a second episode and one-third of those clients will progress to have a third recurrence.

2. Whereas surgical management used to be considered based on the number of episodes of diverticulitis, currently surgical management is not dependent on the number of uncomplicated episodes but rather the presence of a complicating problem.

3. There is no evidence to support the idea that uncomplicated episodes progress or worsen over time.

FOLLOW-UP

A. Outpatients should be reevaluated after 3 days of therapy and every week until resolution of symptoms. Generally, reimaging is not indicated or recommended.

B. A colonoscopy should be performed from 6 weeks after recovery to evaluate the extent of the diverticulosis/rule out other manifestations such as colon cancer.

EMERGENT ISSUES/INSTRUCTIONS

A. Arrange for prompt hospitalization and surgical consultation if the client's temperature rises above 102.5°F, the client's pain worsens, peritoneal signs develop, or white blood cell (WBC) continues to rise. Surgery consultation is required for abscess, peritonitis, obstruction, fistula, or failure to improve after several days of medical management.

CONSULTATION/REFERRAL

A. Consult a physician if the client has mild diverticulitis: temperature less than 101°F; WBC less than 13,000 to 15,000.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. For the older adult with existing diverticulosis, nurse practitioner (NPs) should practice special consideration of the following *geriatric syndromes*:

a. Pain: Poorly relieved pain is an important cause of functional impairment at any age group:

i. Older adults have a diminished sensorium, allowing pathology to advance to a dangerous point prior to symptom development.

ii. Older adults with abdominal pain may present with altered mental status. Cognitive impairment can make assessment and diagnosis of pain more difficult.

iii. For dementia or noncommunicative clients, the American Medical Directors Association has endorsed the Pain Assessment in Advanced Dementia (PAINAD) Scale, which scores for the following items:

1) Breathing: Examples include normal, labored, hyperventilation.

2) Negative vocalization: Examples include moaning, negative language, crying.

3) Facial expression: Examples include smiling, frowning, grimacing.

4) Body language: Examples include relaxed, tense, rigid, or striking out.

5) Consolability: Examples include extent to which a caregiver is able to console, distract, or reassure.

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ELEVATED LIVER ENZYMES

DEFINITION

A. Liver function tests (LFTs) used to determine the health of the liver, but are not direct measures of its function. The liver has excretory, metabolic, protective, detoxification, hematologic, and circulatory functions. LFTs may be abnormal even in clients with a healthy liver. Normal laboratory chemistry values may vary according to age, gender, ethnicity, blood group, and postprandial state, as well as other factors such as exercise and pregnancy. Uses for common liver chemistry tests (Table 14.8) include the following:

1. Assessing the severity of hepatocellular injury.

2. Evaluating cholestatic problems.

3. Following the trend of hepatic disease.

4. Diagnosing for the presence of latent liver disease (e.g., differential diagnosis of ascites or hematemesis).

5. Screening for suspected cases during outbreaks of infective hepatitis.

6. Making differential diagnosis of the different types of jaundice.

7. Screening persons exposed to hepatotoxic drugs. A wide variety of drugs are associated with liver disease and require monitoring of liver function, including carbamazepine, methyldopa, minocycline, macrolide antibiotics, nitrofurantoin, statins, sulfonamides, terbinafine, chlorpromazine, and methotrexate. In addition, drugs can cause fatty liver and steatohepatitis and vascular injury. Methotrexate treatment requires special care, to prevent dose-dependent liver fibrosis, and noninvasive markers of fibrosis should be monitored. Although statins can lead to drug-induced liver injury, this is very rare, with studies demonstrating they are safe in clients with preexisting abnormal liver enzymes.

INCIDENCE

A. The incidence of elevated liver enzymes is undetermined. Abnormal elevations of serum liver chemistries may occur in 1% to 4% of the asymptomatic population.

TABLE 14.8 LIVER CHEMISTRY TESTS AND IMPLICATIONS

Liver Chemistry Test	Clinical Implication of Abnormality
ALT	Hepatocellular damage
AST	Hepatocellular damage
ALP	Cholestasis, infiltrative disease, or biliary obstruction
Albumin	Synthetic function
Alpha-fetoprotein	Cancer marker when elevated
Bile acids: urine bile salts, bile pigments, and urobilinogen	Cholestasis or biliary obstruction, impaired hepatic update or secretion, or portal systemic shunting
Bilirubin: serum total, direct and indirect bilirubin	Cholestasis, impaired conjugation, or biliary obstruction
Cholesterol, serum triglycerides	Lipoprotein production and metabolism, chronic cholestasis
Fibrinogen	Liver damage/cirrhosis, acute liver insufficiency, poisoning
GGT	Cholestasis or biliary obstruction, malignant involvement in hepatocellular disease, more sensitive than other enzymes in alcoholism
Hepatitis surface antigen, IgM, antibody, RNA, genotype, viral load	Differentiation of type of hepatitis
LDH	Hepatocellular damage not specific for hepatic disease
Total proteins, albumin globulin, (A/C ratio)	Hepatitis, advanced liver disease
PT	Synthesis function in hepatocellular disease, fulminate hepatitis
Plasma ammonia	CNS dysfunction/toxicity or end-stage liver disease
5NT	Cholestasis or biliary obstruction
Urea	End-stage liver disease

5NT, 5′-nucleotidase; A/C, albumin, creatinine; ALP, alkaline phosphatase; ALT, alanine transaminase; AST, aspartate transaminase; CNS, central nervous system; GGT, gamma-glutamyl transpeptidase; IgM, immunoglobulin M; LDH, lactate dehydrogenase; PT, prothrombin time.

PATHOGENESIS

A. Pathogenesis varies by diagnosis.

PREDISPOSING FACTORS

A. Predisposing factors are dependent on the suspected or known medical diagnosis.

COMMON COMPLAINTS

A. Asymptomatic.
B. Pruritus.
C. Jaundice.
D. Ascites.
E. Fatigue.
F. Weight loss.
G. Change in the color of urine (dark) or stools (clay-colored).
H. Abdominal pain.
I. Nausea/vomiting.

SUBJECTIVE DATA

A complete medical history is the single most important part of the evaluation of a client with elevated LFTs.

A. Due to polypharmacy, a full review of medications, herbals, and over-the-counter (OTC) drugs should be undertaken to identify medications that should be avoided in the geriatric population, duplicate medications, and medications that are able to be decreased or deprescribed.

B. Determine the duration of LFT abnormalities (if known).

C. Review the presence of accompanying symptoms, including arthralgias, myalgias, rash, abdominal pain, fever, pruritus, and changes in the color of urine or stool.

D. Has the client experienced any anorexia or weight loss? Over what period of time did weight loss occur?

E. Review parenteral exposures, including transfusions (especially if occurred prior to 1992), intravenous (IV) and intranasal drug use, tattoos, and sexual history.

F. Review the client's recent travel history: food, sexual history, regions of travel, purpose of travel, exposure to other sick contacts.

G. Review the client's exposure to people with jaundice.

H. Review the client's occupational and recreational history and possible exposure to hepatotoxins.

I. Review the client's history of alcohol consumption. Significant alcohol intake is generally considered greater than 210 g alcohol per week in a male or greater than 140 g alcohol per week in a female occurring over at least a 2-year period. Fourteen grams of alcohol are contained in a 12-oz beer, 5 oz of wine, or 1.5 oz of liquor.

J. Evaluate the gestational age of pregnancy. Hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome is generally present in the mid-second trimester or third trimester of pregnancy.

PHYSICAL EXAMINATION

A. Temperature (if infection is suspected), pulse, respirations, blood pressure (BP), and height and weight to calculate body mass index (BMI).

B. Inspect:

1. Observe for temporal and proximal muscle wasting.

2. Perform eye and mouth (mucous membranes) examination for icterus.

3. Perform skin examination for icterus, spider nevi, palmar erythema, presence of caput medusae (the presence of dilated veins seen on the abdomen; noted with cirrhosis of the liver and portal hypertension).

4. Evaluate the presence of gynecomastia.

5. Observe for the presence of jugular venous distension (JVD), a sign of right-sided heart failure (HF) that suggests hepatic congestion.

C. Auscultate:

1. Heart.

2. Lungs.

D. Percuss:

1. Abdomen.

E. Palpate:

1. Abdominal examination:

a. Evaluate the presence of hepatomegaly; focus on the size and consistency of the liver.

b. Evaluate the presence of splenomegaly; focus on the size of the spleen. An enlarged spleen is most easily appreciated with the client in the right lateral decubitus position.

c. Assess for ascites: Note presence of a fluid wave or shifting dullness.

d. Assess for an abdominal mass.

2. Lymph nodes: Evaluate lymphadenopathy.

3. Conduct testicular examination for testicular atrophy (increased estrogen/reduced testosterone).

DIAGNOSTIC TESTS

A. The particular LFT tests ordered are related to the suspected or identified medical diagnosis. Table 14.9 shows common serologic tests for viral hepatitis.

B. The pattern of elevated liver biochemistries suggests the underlying cause:

1. Elevation of aminotransferases indicates hepatocyte injury.

2. Elevation of alkaline phosphatase (ALP) indicates cholestasis.

3. Changes in albumin and/or prothrombin time (PT) indicate interruption in the synthesis process.

C. The magnitude of the elevation can indicate the underlying cause:

1. Aspartate transaminase (AST): Alanine transaminase (ALT) of greater than 2:1 indicates alcohol-related disease. If the AST:ALT is greater than 3:1, the chance of alcohol-related disease is at 96%.

2. AST and ALT normal to two times upper limit normal (ULN) indicates chronic hepatitis B or C.

3. AST and ALT less than four times ULN occurs in nonalcoholic hepatic disease.

4. AST less than eight times ULN and ALT less than five times ULN indicates alcoholic hepatic disease.

5. AST and ALT greater than 25 times ULN indicates acute viral hepatitis.

6. AST and ALT greater than 50 times ULN indicates ischemic hepatitis.

TABLE 14.9 SEROLOGIC TESTS FOR HEPATITIS

Virologic Test	Usual Clinical Implication of a Positive Test
Hepatitis A-IgM	Positive in acute hepatitis A
Hepatitis A-IgG	Positive in response to previous hepatitis A infection or vaccination
HBsAg	Positive during active hepatitis B infection
Hepatitis B surface antibody	Positive in response to previous hepatitis B infection or vaccination
Hepatitis B core antibody-IgM	Positive during active hepatitis B infection
Hepatitis B core antibody-IgG	Positive in response to current or prior hepatitis B infection
HBV-DNA	Positive during active hepatitis B infection
Hepatitis Be antigen	Positive test indicates replicative state of wild-type hepatitis B infection
Hepatitis Be antibody	Positive after replicative state of wild-type hepatitis B infection
HBV viral load	Assess hepatitis B virology
HCV-antibody ELISA	Positive during or after hepatitis C infection
HCV RIBA	Positive during or after hepatitis C infection
HCV-RNA	Positive during hepatitis C infection
HCV viral load	Assess hepatitis C virology
HCV genotype	Genotyping is used for evaluation of the length of therapy

ELISA, enzyme-linked immunosorbent assay; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; HCV, hepatitis C virus; IgG, immunoglobulin G; IgM, immunoglobulin M; RIBA, recombinant immunoblot assay.

DIFFERENTIAL DIAGNOSES

A. Table 14.10 shows differential diagnoses with elevated liver enzymes.

PLAN

A. General interventions:

1. The clinical significance of any liver chemistry test abnormality must be interpreted in the context of the clinical situation. All abnormalities should be confirmed before additional tests are added. Follow-up blood chemistries can vary according to the nature of the initial elevation:

a. An elevated ALP should be rechecked with additional testing with gamma-glutamyl transpeptidase (GGT) or fractionated ALP to determine if the elevation is related to hepatic disease or from an outside location such as bone.

b. An elevated bilirubin should be fractionated to determine if it is conjugated or unconjugated.

c. Serology for viral hepatitis, viral infections, autoimmune disease, muscular disease, Wilson disease, and pregnancy may also be added according to the client's history and exam.

d. Abdominal ultrasound should be undertaken.

e. If the source is not obvious by laboratory testing and ultrasonography, liver biopsy is recommended.

2. Clients with marked abnormalities of liver tests, or with signs and symptoms of chronic liver disease (CLD) or hepatic decompensation (e.g., ascites, encephalopathy, coagulopathy, or portal hypertension), should be evaluated and treated in a more expeditious manner than asymptomatic clients.

B. Client teaching:

1. Lifestyle modifications, including discontinuance of medications and alcohol, weight loss, and dietary changes, can be recommended as appropriate.

FOLLOW-UP

A. Follow-up testing for elevated LFTs, including abdominal/liver ultrasonography, CT, MRI, and liver biopsy, is dependent on the risk factors for disease, symptoms and history, and physical finding of the suspected or identified medical diagnosis. A liver chemistry test that is normal does not ensure that the client is free of liver disease. If a laboratory error is suspected, the laboratory test should be repeated.

CONSULTATION/REFERRAL

A. Refer to gastroenterology or hepatology any client with persistent elevation of aminotransferases greater than two times ULN or of ALP greater than 1.5 times ULN.

B. Referral to infectious disease specialist as indicated.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. Pregnancy: HELLP syndrome is a severe form of pregnancy-induced hypertension (PIH or preeclampsia). It may occur anywhere from the mid-second trimester to immediate postpartum. HELLP syndrome occurs in 0.2% to 0.6% of pregnancies. The etiology is unknown. The presence of laboratory abnormalities confirms the diagnosis. Treatment of HELLP syndrome will not be covered. However, the laboratory abnormalities that are noted include the following:

a. Hemolytic anemia.

b. Proteinuria.

c. Serum AST level (>70 IU/L).

d. Low platelet count (<10,000 cells/mL).

e. Serum lactate dehydrogenase (LDH) level (>600 IU/L).

f. Total bilirubin level (>1.2 mg/dL).

B. Geriatrics:

1. Elevated liver enzymes are common in older adults:

a. One in six of the general population aged 75 and above have at least one elevated liver test of AST, ALP, or bilirubin. ▶

TABLE 14.10 DIFFERENTIAL DIAGNOSIS WITH ELEVATED LIVER ENZYMES

Infiltrating diseases of the liver • Sarcoidosis • TB • Fungal infection • Amyloidosis	• Lymphoma • Metastatic malignancy • Hepatocellular carcinoma
Acute viral hepatitis (A-E, EBV, CMV, herpes)	Wilson disease (genetic disorder of biliary copper excretion)
Cholestasis disease	Acute bile duct obstruction
Chronic hepatitis B and C	Hemolysis
Steatosis/NASH	Rhabdomyolysis
Hereditary hemochromatosis	Thyroid disorders
Medication/herbal-induced	Strenuous exercise-induced changes
Alpha-1 antitrypsin deficiency	Pregnancy: HELLP syndrome
Cirrhosis	Toxin(s) exposure
Celiac disease	Acute Budd–Chiari syndrome
Alcohol-related liver injury	Anorexia nervosa

EBV, Epstein–Barr virus; CMV, cytomegalovirus; HELLP syndrome, hemolysis, elevated liver enzymes, and low platelets; NASH, nonalcoholic steatohepatitis; TB, tuberculosis.

2. Elevated liver enzymes levels (mainly ALT, GGT, and ALP but not AST) are positively associated with the prevalence of metabolic syndrome in older adults.

3. A history of diabetes and an assessment of dementia were associated with both elevated AST and ALP, with a history of heart attack associated only with increased ALP.

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FECAL INCONTINENCE

DEFINITION

A. Fecal incontinence (FI) is the involuntary loss of bowel function, ranging from an occasional leakage of mucus or stool when passing flatus to complete loss of bowel control. There are three classifications of FI:

1. Passive incontinence: characterized by impaired awareness of the need to defecate prior to the incontinent episode.

2. Urge incontinence: characterized by the desire to retain fecal material followed by an incontinent episode.

3. Fecal seepage: leakage of fecal material after normal evacuation of stool:

a. Overflow incontinence secondary to fecal impaction.

b. Reservoir secondary to diminished colonic or rectal capacity.

c. Rectosphincteric dysfunction: structural or neurologic damage to the anal sphincter.

INCIDENCE

A. Incidence is dependent on the population studied. Overall incidence is 7.7%, with a range from 2% to 21%. The American College of Gastroenterology (ACG) estimates that more than 18 million Americans have FI. Due to the embarrassing nature of the problem, it is often underreported.

B. Up to 21% of institutional clients have FI. It is the second most common cause of nursing home placement.

C. Incidence increases with age, female gender, and institutionalization.

D. Literature notes 7% incidence of coexisting fecal and urinary incontinence among females and a 4% incidence of FI alone.

PATHOGENESIS

A. FI is a multifactorial problem, and generally a deficiency in one area does not lead to incontinence. Stool continence requires the ability to hold stool (sphincter function), the ability to sense the presence of stool in the rectum (anorectal sensation), the ability to relax and hold the stool (rectal compliance), the mental capabilities to recognize the urge, and the physical ability to go to the toilet, as well as colonic transit of a stool with volume and consistency.

PREDISPOSING FACTORS

A. Fecal impaction/constipation.

B. Diarrhea (viral/bacteria etiology).

C. Obstetrics:

1. Forceps delivery.

2. Episiotomy.

3. Vaginal tears.

4. Large baby (>9.5 lbs).

D. Neurologic/spinal etiology of rectosphincter dysfunction:

1. Lumbar disc herniation with subsequent cauda equina syndrome.

2. Spinal cord injury.

3. Multiple sclerosis.

4. Spina bifida.

5. Meningocele/myelomeningocele.

6. Imperforated anus.

7. Diabetes.

8. Stroke.

E. Trauma.

F. Hemorrhoids/hemorrhoidectomy.

G. Rectocele.

H. Rectal prolapse.

I. Dementia.

J. Radiation for rectal/pelvic cancer.

K. Inflammatory bowel disease (IBD).

L. Inactivity (constipated-related bowel control problems).

M. Vitamin B_{12} deficiency.

N. Hypothyroidism.

O. Severe learning disability.

P. Urinary incontinence.

Q. History of postmenopausal hormone use.

COMMON COMPLAINTS

A. Leakage of mucus/stool when passing flatus.

B. Uncontrolled passing of stool.

OTHER SIGNS AND SYMPTOMS

A. Inability to get to the toilet in time.

SUBJECTIVE DATA

A. Ask about the following bowel-related issues:

1. When did the FI start and how often does the client have bowel control problems?

2. Is the client incontinent of mucus, liquid, and/or solid stools? Do they leak a little or have full incontinence?

3. Is the client able to control passing gas?

4. Does the client have an urge to have a bowel movement (BM) or just have loss of control?

5. Are hemorrhoids present?

6. What foods make the FI worse?

7. Does the client have to do a self-rectal evacuation of stool? How often?

8. Does the client have anal intercourse? How often? Use of penetrating objects?

9. Is there a history of impaction?

10. Does the client wear a pad or an adult diaper?

B. Obtain a detailed obstetrical history:

1. Number of vaginal deliveries.

2. Use of forceps.

3. Infant birth weights and pelvic floor trauma.

4. Need for episiotomy for each vaginal delivery.

5. Presence of vaginal tears.

6. Stool incontinence after deliveries.

C. Has the client been diagnosed with Crohn's or ulcerative colitis (UC)? Is the client currently under treatment? Is the client compliant with their treatments?

D. Review full medical history, especially for diabetes, pelvic radiation, neurologic disease, and back injury. ▶

E. Inquire about urinary incontinence.
F. Review surgical history, including pelvic/rectal surgeries.
G. Review all medications currently being taken, including over-the-counter (OTC) and herbal products. Specifically, evaluate for laxative abuse and recent antibiotic use.
H. Are any other family members ill?
I. Has the client recently traveled or gone camping, which would identify infectious causes? (Refer to entries on diarrhea and infectious diseases.)

PHYSICAL EXAMINATION

A. Check temperature (if applicable), pulse, respirations, blood pressure (BP), and weight.
B. Inspect:
1. Inspect general appearance.
2. Examine the back and lower limbs.
3. Inspect the perianal area for fecal matter, dermatitis, excoriation, fistula, abscess, rectal prolapse, hemorrhoids, and scars.
4. Evaluate if the anal sphincter is open or closed at rest.
C. Auscultate:
1. Auscultate the abdomen.
D. Palpate:
1. Palpate all quadrants of the abdomen to evaluate distension, tenderness, and masses.
E. Anorectal examination: A negative digital rectal examination does not rule out proximal impaction:
1. Evaluate anal stenosis, resting tone.
2. Check presence of impacted stool.
3. Assess the puborectalis muscle by palpating the posterior anal canal:
a. Ask the client to bear down as if having a BM. (An intact puborectalis displaces the examining finger anteriorly.)
b. Ask the client to squeeze the provider's finger.
4. Stroke the perianal skin bilaterally to check for an anal "wink" anocutaneous reflex.
F. Pelvic examination:
1. Perform pelvic examination to evaluate the presence of a cystocele, rectocele, enterocele, vaginal prolapse, and fistula.
H. Mental status:
1. Make a cognitive assessment, such as the Mini-Mental State Examination (MMSE).
2. Assess for depression with Patient Health Questionnaire-2 (PHQ-2) or Patient Health Questionnaire-9 PHQ-9) screening tool.

DIAGNOSTIC TESTS

A. Stool studies to rule out infectious etiology (refer to section "Diarrhea" in this chapter).
B. Abdominal x-ray.
C. Sigmoidoscopy/colonoscopy: Flexible sigmoidoscopy is reserved for clients younger than age 40.
D. Rectal manometry.
E. Anorectal ultrasound.
F. Defecography/proctography.
G. MRI.
H. Anal electromyography (EMG).
I. Balloon expulsion test.
J. Thyroid function tests.
K. Hydrogen breath tests to evaluate lactose/fructose intolerance.

DIFFERENTIAL DIAGNOSES

A. FI.
B. Constipation/fecal impaction.
C. Diarrhea secondary to infectious etiology.
D. IBD.
E. Postradiation therapy.

PLAN

A. General interventions:

Optimal treatment of FI is dependent on identifying the underlying cause of the symptoms. The goal of therapy is to reduce the number of stools and increase the consistency. The best prevention of FI is an empty rectum.

B. Client teaching: *See Client Teaching Guide for this chapter, "Fecal Incontinence."*
1. Discuss keeping a stool diary for a week. A BM record is available at www.hct.nhs.uk/media/1067/bristol-stool-chart.pdf.
2. Discuss keeping a food diary for a week to assist in identifying any foods that cause, trigger, or worsen FI such as coffee, fatty or greasy foods, dairy products (lactose intolerance), sugar-free gum/artificial sweeteners, and cured/smoked meats.
3. Discuss a high-fiber diet. Give client a dietary sheet on a high-fiber diet (see section "High-Fiber Diet" of Appendix B).
4. Discuss the need for additional fluids, 8 to 10 glasses of water, to help prevent constipation. Caffeine, carbonated beverages, alcohol, and milk may cause diarrhea.
5. Discuss the need for manual disimpaction.
6. Remove physical barriers that prevent getting to the bathroom; if mobility an issue, consider bedside commode.
7. Discuss tips to help deal with bowel incontinence/hygienic measures:
a. Use the toilet before leaving home.
b. Identify public restrooms before they are needed.
c. Carry a bag of supplies and change clothes as soon as possible.
d. Pants with elastic are easier to pull down than pants that have buttons.
e. Wear disposable underwear/absorbent pads.
f. Use water without soap; soap dries and irritates.
g. Use baby wipes.
h. Use a moisture-barrier cream, such as zinc oxide.
i. Use fecal deodorant to disguise odor.
C. Conservative treatment:
1. Bowel retraining program:
a. Schedule defecation throughout the day, especially after meals.
2. Biofeedback:
a. **Biofeedback requires the ability to comprehend and follow directions, motivation, and cooperation:**
i. Auditory or visual feedback for reeducation of the pelvic floor muscles.
ii. Rectal sensitivity training utilizing a rectal balloon.
iii. Anal sphincter exercises.
3. Pelvic floor exercises (Kegel exercises).
4. Anal plug can be irritating and is most effective for clients with decreased anal sensation.
D. Dietary management:
1. Counsel the client on nutrition and low-fat, low-cholesterol, and low-sodium diets. Give diet handouts. ▶

E. Pharmaceutical therapy:

1. Solutions containing magnesium, phosphate, or citrate should be used with caution or avoided, especially with clients who have heart failure (HF), or chronic renal failure secondary to the absorption of these electrolytes increases fecal impaction:

2. Mainstay of treatment: loperamide hydrochloride (Imodium): 2 to 4 mg two or three times a day to control symptoms. Maximum daily dose for loperamide is 16 mg.

3. Diphenoxylate hydrochloride/atropine (Lomotil):

a. Contains an opioid Schedule V medication under the Controlled Substance Act. May produce sedation and dizziness.

b. Two tablets QID until diarrhea is controlled, or if liquid Lomotil 10 mL up to four times a day.

c. Maintenance dose two tablets per day.

4. Bulking agents to increase formed stools (used for diarrhea and constipation): may worsen incontinence in clients with decreased rectal compliance but is useful in clients with low-volume loose stools:

a. Methylcellulose (Citrucel): one to two tablespoons daily.

b. Psyllium (Metamucil): Dose varies per client; start with one scoop powder or six pills per day.

5. Fecal impaction:

a. Polyethylene glycol (MiraLax).

b. Enemas (predictable and have a timely response).

c. Suppositories (e.g., glycerine).

d. Laxatives.

6. Manage and treat underlying disease(s) if present, including *Clostridioides difficile* and IBD (see related entries).

F. Medical management:

1. Sacral nerve stimulator: In 2013, the U.S. Food and Drug Administration (FDA) approved the Medtronic InterStim Therapy System.

2. Injectable nonabsorbable bulking agent: hyaluronic acid/dextranomer (Solesta)-tissue bulking agent given by submucosal injection:

a. Indicated as an option for FI treatment for clients older than 18 years of age having failed conservative treatment or not good surgical candidate.

b. Administered by trained providers who hold a certification on the Solesta procedure.

G. Surgical management:

1. Surgical management is dependent on the findings of the history, physical, diagnostic testing, failure of conventional therapies, and client consent. Options include sphincteroplasty or colostomy.

FOLLOW-UP

A. Follow-up is determined by client's needs, severity of acute FI, and whether complications are present.

CONSULTATION/REFERRAL

A. Refer to a gastroenterologist for an endoscopy and if diagnosis is still in question.

B. Refer to a colorectal surgeon.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. FI is the second leading cause of institutionalization of older adults.

2. Polypharmacy with constipating drugs and laxative overuse are common reasons for FI in older adults. Conduct a full review of medications, herbals, and OTC drugs should be undertaken to identify medications that should be avoided in the geriatric population, duplicate medications, and medications that are able to be decreased or deprescribed. (Refer to section on deprescribing in Chapter 3, "Adult-Geriatric Assessments.")

3. Many older adults experience FI as a result of fecal impaction and subsequent overflow. Reduced fluid intake is a common factor for constipation in older adults:

a. Impaired thirst perception.

b. Urinary incontinence.

c. Limited access to liquids due to immobility.

d. Removal of fecal impaction involves a three-step program:

i. Disimpaction by manual removal or with use of enema.

ii. Titration to appropriate dose of oral polyethylene glycol to promote one BM at least every other day.

iii. Maintenance therapy to reduce the risk of reoccurrence.

4. For the older adult with existing FI, a nurse practitioner (NP) should practice special consideration of the following *geriatric syndromes*:

a. Delirium is a common early sign of acute illness or infection in older adults. It is more common in clients with existing cognitive impairment. Dehydration is more common in older adults. Signs of dehydration include confusion, muscle weakness, fever, dizziness, poor skin turgor, hypotension, and tachycardia. Diminished thirst mechanism and decreased body water exacerbate dehydration.

b. At a minimum, clients and family should be asked if the client has experienced episodes of altered mental status:

i. The Brief Confusion Assessment Method (bCAM) is a well-documented assessment for delirium. It is available at www.mnhospitals.org/Portals/0/Documents/ptsafety/LEAPT%20Delirium/HELP%20Program%20CAM%20Flowsheet.pdf.

c. Falls: Consistently assess for falls. Discuss the risk of dizziness or light-headedness when standing:

i. At the minimum, older adults should be asked how many times they have fallen since their last visit. A reported fall should trigger questions about whether the fall led to injury, a visit to urgent care or hospital ED, or a hospital admission:

1) Many falls occur en route to the bathroom when the client hurries and may not be able to remove their pants and undergarments in time.

ii. Clients who use pads or adult diapers should be informed of risks associated with infrequent changing. Because of the high cost of absorbent pads and undergarments, infrequent changing is common and contact dermatitis and candidal rashes may develop. If these occur, clients should be encouraged to change pads/garments more frequently, and avoid wearing absorbent garments overnight and instead should use mattress/bedding pads.

iii. Safety measures to reduce falls in the home include removing small area carpets, keeping night lights along the path to the bathroom, and installing a toilet seat or nearby grab bars. ▶

d. Depression: Anxiety and depression are common in FI and are factors that lead to isolation and decreased health-related quality of life. A depression screen should be done at each office visit:

i. An online PHQ-9 is available at www.mdcalc.com/phq-9-patient-health-questionnaire-9.

ii. The Geriatric Depression Scale, Short Form, a 15-item questionnaire that can be quickly applied in the clinical setting, is available at https://www.apa.org/depression-guideline/patient-health-questionnaire.pdf.

e. Weight loss/appetite suppression: Weight loss is a predictor of mortality. Clinically significant weight loss is considered to be 5% loss of usual body weight in 3 months or 10% in 6 months:

i. Due to gastrointestinal (GI) distress, abdominal discomfort, and diarrhea, older adults are at increased risk of avoiding eating.

ii. Monitor weight patterns/body mass index (BMI). (Caution: BMI is not used to identify loss of muscle mass.)

iii. Monitor both serum albumin and anemia: Hypoalbuminemia and anemia are not necessarily associated with low bodyweight. They can be caused by decreases in liver protein synthesis:

1) Serum albumin concentration is commonly recommended.

2) Hemoglobin (Hgb), prealbumin, and transferrin.

3) Cholesterol.

5. Beers Criteria caution:

a. Diphenoxylate/atropine predisposes older adults to significant side effects, including falls, mental status changes, and urinary retention.

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GASTROENTERITIS, BACTERIAL AND VIRAL

DEFINITION

A. Bacterial and viral gastroenteritis is an acute inflammation of the gastrointestinal (GI) mucosa of the middle or lower intestine. It is primarily an acute, self-limited illness that resolves in a week or less. The illness is usually accompanied by nausea, vomiting, diarrhea, fever, and generalized abdominal pain, but can occur with diarrhea alone. Immunocompromised clients can develop unremitting or fatal symptoms from gastroenteritis.

INCIDENCE

A. Gastroenteritis is very common, occurring in all age groups. Epidemic outbreaks of bacterial gastroenteritis occur in groups who have ingested contaminated food. Viral excretion can begin before symptoms. Gastroenteritis is responsible for an estimated eight million healthcare visits and 250,000 hospitalization a year. There are 19 to 20 million annual gastroenteritis illnesses in the United States annually.

PATHOGENESIS

A. Gastroenteritis is commonly due to infectious agents: viruses, bacteria, and parasites (Table 14.11). There are four viral agents: rotavirus, norovirus, enteric adenovirus, and astrovirus. Exotoxins produced by some organisms induce hypersecretion or increased peristalsis, resulting in diarrhea or vomiting. Bacteria such as *Escherichia coli* and *Salmonella* penetrate and invade the gastric mucosa and lead to diarrhea accompanied by fever and fecal leukocytes. Viruses destroy enterocytes of the upper jejunal villi, often producing secondary lactose intolerance.

PREDISPOSING FACTORS

A. Travel to areas where cholera or *Giardia* is epidemic:

1. Ingestion of raw or undercooked seafood or drinks containing cholera-contaminated ice or water.

2. Ingestion of *Giardia*: contaminated water supplies.

B. Ingestion of food contaminated with *Salmonella* or *Shigella.* Foods that are often implicated are domestic fowl and eggs, custard-filled pastries, processed meats, foods warmed on steam tables, poultry, red meat, raw seafood, raw milk, rice, and bean sprouts due to the following:

1. Inadequate cooking time and temperatures.

2. Poor hygiene, lack of handwashing.

3. Improper storage of food.

4. Ingestion of fruits and vegetables contaminated by an infected person or by animal products.

C. Infection by person-to-person spread:

1. Day-care centers: Rotavirus can be found on toys and hard surfaces.

2. Overcrowded environment, inadequate healthcare or education.

3. Schools/dormitories. ▶

TABLE 14.11 INFECTIOUS AGENTS CAUSING GASTROENTERITIS

Causative Agent	Incubation Period
Noroviruses	12 hr–2 d
Escherichia coli	24–72 hr
Campylobacter	2–5 d
Staphylococcus	1–6 hr
Shigella	8–24 hr
Botulism	12–36 hr
Giardia lamblia	7–21 d
Salmonella	6–72 hr
Rotaviruses	1–3 d
Adenovirus	5–8 d
Clostridium perfringens	10–12 hr
Clostridioides difficile	Variable
Listeria species	20 hr

4. Nursing homes.
5. Banquet halls, cruise ships.

D. Contact with *Salmonella*-infected turtles, iguanas, and other reptiles kept as pets.

E. Seasonal outbreaks:
1. Rotavirus and astrovirus occur from October to April.
2. Adenovirus occurs throughout the year.
3. Norovirus occurs throughout the year but tends to increase in cooler months (December–February).

COMMON COMPLAINTS

A. Abrupt onset of nausea and vomiting.
B. Abrupt onset of diarrhea.
C. Fever, sometimes.

OTHER SIGNS AND SYMPTOMS

A. Explosive flatulence.
B. Cramping abdominal pain.
C. Abdominal tenderness.
D. Frequent watery diarrhea.
E. Mucoid stools with or without blood.
F. Tenesmus.
G. Myalgia.
H. Headache.
I. Weakness.
J. Malaise.

SUBJECTIVE DATA

A. Review onset, duration, and course of symptoms, including presence of abdominal pain and frequency of bowel movements (BMs). Ask the client if anyone else in the family or coworkers have the same symptoms.

B. Ask the client about travel history, including travel by cruise ships or travel to foreign countries and camping with ingestion of water from streams, springs, or untreated wells. Inquire regarding exposure to animals.

C. Ask the client about crowded or unsanitary living conditions, use of day-care centers, and institutional living.

D. Take a 24-hour diet history, including ingestion of prunes or beans.

E. Review diarrhea history:
1. How many stools?
2. How frequent are the diarrhea stools?
3. What color is the stool?
4. Does the stool contain mucus?
5. Has there been blood in the stool?
6. Does the client have tenesmus (constant feeling for the need to pass stool)?

F. Inquire about other symptoms, such as fever or respiratory problems.

G. If the client is an infant, ask caregiver about activity level, irritability, sleep pattern, fluid intake, and number of wet diapers.

H. Review drug history intake, including laxatives, antacids, antibiotics, quinine, or anticancer medications.

I. Has the client been vaccinated with the rotavirus vaccine?

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure (BP), and weight:
1. Bacterial infections: temperatures between 101°F and 102°F.
2. Viral infections: temperatures of 103°F and above.
3. Note hypotension and tachycardia.

B. Inspect:
1. Inspect general appearance; note if the client is very ill.
2. Assess hydration status. Signs of dehydration:
   a. Mild: slightly dry buccal mucous membranes, increased thirst, decreased urine output.
   b. Moderate: sunken eyes, loss of skin turgor, dry buccal mucous membranes, decreased urine output.
   c. Severe: signs of moderate dehydration and one or more of the following: rapid thready pulse, tachypnea, lethargy, and postural hypotension.

C. Auscultate:
1. Abdomen for bowel sounds; note hyperactive bowel sounds, absent or hypoactive bowel sounds (common with botulism), and borborygmi.

D. Palpate:
1. Abdomen for diffuse tenderness, slight distension, masses, rebound tenderness, and spasm.

E. Rectal exam:
1. Check for masses, fissures, inflammation, perianal erythema, or stool in ampulla.

F. Neurologic exam:
1. Check for dizziness, difficulty swallowing, and other neurologic signs.
2. Neurologic signs and symptoms indicate botulism and require emergency intervention.

DIAGNOSTIC TESTS

A. No immediate lab tests are required if dehydration is absent or mild and the client feels well except for frequent diarrhea.

B. Complete blood count (CBC) with differential: Serologic studies may fail to differentiate between bacterial and viral pathogens.

C. Sedimentation rate: elevated with infections or inflammation

D. Electrolytes, sodium, chlorides, potassium, and blood urea nitrogen (BUN).

E. Blood gases to assess acid–base balance, if indicated.

F. Blood cultures, if indicated.

G. Stool for guaiac, leukocytes, ova, and parasites; test specimens three times, every other day. Stool guaiac is usually negative in viral infections, positive in invasive bacterial infections. Large numbers of white cells in stool suggest inflammatory or invasive diarrhea, such as occurs with *Shigella, Salmonella, Campylobacter*, invasive *E. coli*, or *Entamoeba.* Mononuclear cells in stool are characteristic of salmonellosis.

H. Stool culture if blood or mucus, fever more than 24 hours, or leukocytes are present.

I. Special cultures for *Campylobacter* and cholera are required.

J. Urinalysis: excludes urinary tract infection (UTI) as cause of nonspecific diarrhea; urine-specific gravity to assess dehydration.

K. Sigmoidoscopy: Skip bowel prep with presentation of gross blood, large numbers of leukocytes in stool, or severe illness.

L. Culture food from suspected foci for *Salmonella.*

M. Real-time reverse transcriptase-polymerase chain reaction (RT-qPCR) is the most widely used assay for detecting norovirus in stool, vomitus, and environmental specimens. The best detection is in stool specimens.

DIFFERENTIAL DIAGNOSES

A. Gastroenteritis, bacterial and viral.
B. Acute viral hepatitis.

▶

C. Acute appendicitis.
D. Cholecystitis.
E. Irritable bowel disease (IBD; ulcerative colitis [UC]/Crohn's disease [CD]).
F. Pelvic inflammatory disease (PID).
G. Bowel obstruction for other causes.
H. Colorectal cancer (CRC).
I. Irritable bowel syndrome (IBS).
J. Microscopic colitis.
K. Malabsorption syndrome.
L. Postcholecystectomy syndrome.
M. Medication-induced.
N. Laxative abuse.

PLAN

A. General interventions:

1. Meticulous handwashing is the single most important measure to decrease transmission. Hand sanitizers are an option when access to soap or clean water is limited.

2. Advise the client to begin bedrest with progression to regular activities.

3. If the client is diapered and/or incontinent, tell the client or caregiver to adhere strictly to contact precautions. Alcohol-based handwashing may decrease the spread.

4. Diaper-changing areas should be separate from food preparation areas.

5. Chlorine-based disinfectants inactivate rotavirus and may prevent disease transmission from contact with environmental surfaces.

B. Client teaching: *See Client Teaching Guide for this chapter, "Diarrhea."*

C. Dietary management:

1. There are no specific dietary restrictions for clients diagnosed with acute gastroenteritis. As tolerated, food intake should be advised. Small meals and low-residue foods are better tolerated. Soup, saltines, broiled or boiled salted starches (oats/cereal/potatoes), and bananas are good choices.

2. Hydration is one of the most important factors in the prevention of complications. For most clients, use of sports drinks, diluted juices, or broth is sufficient. Oral rehydration solutions may be implemented for moderate to severe cases.

3. See section "Nausea and Vomiting Diet Suggestions" of Appendix B.

D. Pharmaceutical therapy:

1. The primary treatment for viral gastroenteritis is fluid replacement. There are no specific antiviral pharmaceutical therapies. Intravenous (IV) rehydration of fluids and electrolytes may be required in severe dehydration as evidenced by dry mucous membranes, hypotension, tachycardia, or mental status changes.

2. Antibiotics may or may not be prescribed according to the bacterial source. Antimicrobial therapy is not indicated for uncomplicated (noninvasive) gastroenteritis because therapy does not shorten duration of the disease and can prolong duration of excretion of *Salmonella* organisms.

3. Antidiarrheal therapy delays transit time and can reduce the severity and duration of abdominal cramping; however, it may prolong the course of some bacterial diarrhea such as *Shigella* and *E. coli*. In otherwise healthy clients younger than 65 years of age, if diarrhea is moderate to severe or if clinical signs of dehydration are evident, short-term (1–2 days) use of loperamide may be used to facilitate oral rehydration.

4. An antiemetic such as ondansetron (Zofran), which does not cause sedation, may be useful for 1 to 2 days if vomiting is severe enough to prevent oral rehydration.

5. Review all medications, herbal, and over-the-counter (OTC), to decrease or deprescribe other medications that may be triggering diarrhea.

EMERGENT ISSUES/INSTRUCTIONS

A. Refer clients to a hospital immediately if they have dehydration, rebound tenderness, severe abdominal pain, neurologic symptoms, and intussusception.

FOLLOW-UP

A. Tell the client to return if the condition worsens or if signs and symptoms have not abated in 48 to 72 hours. However, diarrhea due to *Salmonella* may be expected to persist for up to 2 weeks.

B. If diarrhea persists for 1 or more weeks, have the client return for a secondary evaluation. Stool studies, white blood cell (WBC), renal function, and electrolytes are warranted at this time.

C. Twenty-five percent of affected clients will have long-lasting bowel changes after recovering from gastroenteritis. A small percentage of these clients will develop postinfectious IBS.

D. Reporting surveillance systems:

1. In 2009, the Centers for Disease Control and Prevention (CDC) launched the National Outbreak Reporting System (NORS) and updated in 2017 to collect information on outbreaks of foodborne, waterborne, and enteric disease that is spread from person to person, animals, environmental surfaces, and other ways by public health agencies.

2. In 2009, the CDC developed CaliciNet for public health and food regulator laboratories to submit outbreaks to a national database.

3. New Vaccine Surveillance Network (NVSN).

4. Foodborne Disease Active Surveillance Network (FoodNet).

CONSULTATION/REFERRAL

A. Refer any client with diarrhea longer than 7 days that has had no response to usual treatment.

B. Refer an immunocompromised client.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. Those with concomitant chronic debilitating disease are at a higher risk of mortality. Closely monitor clients with immunodeficiency, IBD, valvular heart disease, diabetes mellitus, renal impairment, rheumatoid arthritis, systemic lupus erythematosus (SLE), and clients taking immunosuppressants, corticosteroids, or diuretics.

B. Geriatrics:

1. Older adults are at a higher risk of mortality secondary to dehydration. Diminished thirst mechanism and decreased body water exacerbate dehydration. Signs of dehydration include confusion, muscle weakness, fever, dizziness, poor skin turgor, hypotension, and tachycardia.

a. At a minimum, clients and their family should be asked if the client has experienced episodes of altered mental status.

i. The Brief Confusion Assessment Method (bCAM) is a well-documented assessment for delirium. It is available at deliriumnetwork.org/wp-content/uploads/2018/05/bCAM.pdf.

2. Due to polypharmacy, a full review of medications, herbals, and OTC drugs should be undertaken to identify medications that should be avoided in the geriatric ▶

population, duplicate medications, and medications that are able to be decreased or deprescribed. (Refer to section on deprescribing in Chapter 3, "Adult-Geriatric Assessments.")

3. New residents to nursing/group homes should be isolated from ill clients.

4. For the older adult with existing diarrhea, nurse practitioner (NPs) should practice special consideration of the following *geriatric syndromes*:

a. Falls:

i. Consistently assess for falls. Discuss the risk of dizziness or light-headedness when standing due to orthostatic hypotension and due to weakness.

ii. Many falls occur en route to the bathroom when the client hurries and may not be able to remove their pants and undergarments in time.

iii. At the minimum, older adults should be asked how many times they have fallen since their last visit. A reported fall should trigger questions about whether the fall led to injury, a visit to urgent care or hospital ED, or a hospital admission.

iv. Safety measures to reduce falls in the home include removing small area carpets, keeping night lights along the path to the bathroom, and installing a toilet seat or nearby grab bars.

v. Durable medical equipment such as a bedside commode may be considered.

vi. Clients who use pads or adult diapers should be informed of risks associated with infrequent changing. Because of the high cost of absorbent pads and undergarments, infrequent changing is common and contact dermatitis and candidal rashes may develop. If these occur, clients should be encouraged to change pads/garments more frequently, and avoid wearing absorbent garments overnight and instead should use mattress/bedding pads.

b. Delirium is a common early sign of dehydration, acute illness, or infection in older adults. It is more common in clients with existing cognitive impairment.

c. Weight loss/appetite suppression: Weight loss is a predictor of mortality. Clinically significant weight loss is considered to be 5% loss of usual body weight in 3 months or 10% in 6 months:

i. Monitor weight patterns/body mass index (BMI). (Caution: BMI is not used to identify loss of muscle mass.)

ii. Monitor both serum albumin and anemia: Hypoalbuminemia and anemia are not necessarily associated with low body weight. They can be caused by decreases in liver protein synthesis:

1) Serum albumin concentration is commonly recommended.

2) Hemoglobin (Hgb), prealbumin, and transferrin.

3) Cholesterol.

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GASTROESOPHAGEAL REFLUX DISEASE AND DYSPEPSIA

DEFINITION

A. The American College of Gastroenterology (ACG) defined gastroesophageal reflux disease (GERD) as symptoms or complications resulting from the reflux of gastric contents into the esophagus or beyond, into the oral cavity (including larynx) or lung.

B. GERD is considered a normal physiologic process in healthy infants, children, and adults. Most episodes last less than 3 minutes, and most often occur 30 to 60 minutes after meals and with reclining positions. GERD is present when the episodes, more than twice a week, cause troublesome symptoms or complications.

C. The ACG and Canadian Association of Gastroenterology (CAG) Clinical Guideline: Management of Dyspepsia was released in 2017 and updated in 2021. Dyspepsia is defined as predominant epigastric pain lasting at least a month. This can be associated with any other upper gastrointestinal (GI) symptoms such as epigastric fullness, nausea, vomiting, or heartburn, provided epigastric pain is the client's primary concern. Functional dyspepsia (FD) refers to clients with dyspepsia where endoscopy and other tests, where relevant, have ruled out organic pathology that explains the client's symptoms:

1. A very large population of clients will present after self-medicating with antacids, bicarbonate soda, and over-the-counter (OTC) medications. Proton pump inhibitors (PPIs) are currently available by prescription and OTC (Table 14.12).

INCIDENCE

A. GERD is very common; the estimated global presence is 8% to 33%. GERD affects all ages and both genders. Daily heartburn typically occurring postprandially has been estimated to affect 17% to 65% of the normal adult population. ▶

TABLE 14.12 PROTON PUMP INHIBITORS (PPIS)

PPI	Available Dosages
Omeprazole (Prilosec)	Capsules: 10, 20, and 40 mg
Lansoprazole (Prevacid)	Capsules: 15, 20, and 30 mg delayed-release Oral suspension, capsules, and SoluTab
Rabeprazole (Aciphex)	Tablets: 20 mg delayed-release Sprinkles 5 and 10 mg capsules
Pantoprazole (Protonix)	Capsules: 20 and 40 mg Oral suspension: 30 mg
Esomeprazole (Nexium)	Capsules: 20 and 40 mg Oral suspension: 20 and 40 mg
Dexlansoprazole (Dexilant)	Sustained-release tablets: 30 and 60 mg
Omeprazole + sodium bicarbonate (Zegerid)	Capsules: omeprazole 20 and 20 mg plus 300 mg Na+ Oral suspension: omeprazole 20 and 40 mg plus 460 mg Na+

Reflux esophagitis affects over 50% of females at some time during pregnancy. It is estimated that 30% to 90% of asthmatics have GERD. Barrett esophagus, which affects fewer than 1% of adults, is commonly associated with GERD.

B. Approximately 40% of the population have symptoms of dyspepsia globally. Dyspepsia is more common in females, smokers, and those taking nonsteroidal anti-inflammatory drugs (NSAIDs).

PATHOGENESIS

A. GERD is relaxation or incompetence of the lower esophagus persisting beyond the newborn period. Relaxation of the lower esophageal sphincter (LES) allows reflux of gastric acid and pepsin into the distal esophagus. Heartburn occurs when reverse peristaltic waves cause regurgitation of acidic stomach contents into the esophagus. Anatomic abnormalities such as a hiatal hernia predispose persons to GERD. Improper diet and nervous tension are also precipitating factors.

B. GERD has been identified as a trigger for asthma, possibly by the activation of vagal reflexes and/or microaspiration. Asthma may promote GERD, and GERD may provoke asthma. Some asthma medications may reduce LES tone, further complicating the picture. Conversely, a client with GERD may experience pulmonary disease as a response to the esophageal acid exposure.

PREDISPOSING FACTORS

A. Obesity.
B. Consuming large meals.
C. Pregnancy.
D. Emotional stress.
E. Increased abdominal pressure from tight clothes, straining to lift or defecate, or swallowing air.
F. Ingesting drugs that promote LES relaxation; slow gastric emptying can directly affect the mucosal lining (see Table 14.12).
G. Foods that promote LES relaxation:
  1. Alcohol.
  2. Chocolate.
  3. Peppermint.
  4. Garlic and onions.
  5. Fatty, spicy, and fried foods.
H. Smoking: increases stomach acid and LES pressure.
I. Ingestion of caustic agents such as lye.
J. Infection by agents, such as *Candida*, herpes simplex, or cytomegalovirus (CMV), which directly attack the esophageal mucosa.
K. Compromised immunity, from AIDS, diabetes, or chemotherapy.
L. Asthma.

COMMON COMPLAINTS

A. Heartburn.
B. Regurgitation of fluid or food.
C. Chest pain.

OTHER SIGNS AND SYMPTOMS

A. Retrosternal aching or burning.
B. Nocturnal aspiration, water or "acid" brash.
C. Harsh taste in the mouth upon awakening.
D. Chronic cough, especially at bedtime.
E. Hoarseness.
F. Globus sensation.
G. Nausea.
H. Dental erosion.

SUBJECTIVE DATA

A. Review onset, duration, and course of heartburn or other symptoms.
B. Review medication history, including OTC medication and herbals:
  1. Has the client been taking OTC antacids, H2 blockers, or OTC PPIs?
  2. How long has the client been using these OTCs?
  3. Is the client taking drugs that induce symptoms (refer to Table 14.13)?
C. Ask the client about alleviating and aggravating factors.
D. Review the client's habits, including smoking and alcohol intake.
E. Inquire about other symptoms, such as weight loss, dysphagia, blood loss, regurgitation, and diarrhea.
F. Establish the client's usual weight to determine extent of problem.
G. Ask the client about any history of asthma.
H. Ask the client about any history of Crohn's disease (CD; mucosal damage).
I. Review the client's dietary history for bulimia.
J. Ask about any history of celiac disease or family history of autoimmune disorders.

PHYSICAL EXAMINATION

A. Check pulse, respirations, and blood pressure (BP), and measure height and weight to calculate the client's body mass index (BMI).
B. General observation of respiratory distress.
C. Inspect:
  1. Examine throat and evaluate mouth for dental erosion.
  2. Assess swallowing ability.
D. Auscultate:
  1. Evaluate the presence of wheezing in the lungs.
  2. Auscultate the heart.
  3. Evaluate the abdomen in all four quadrants.
E. Palpate:
  1. Palpate abdomen for the presence of hepatosplenomegaly and masses.
  2. Assess the abdomen for tenderness or distension.
F. Rectal exam (if indicated for any history of hematemesis or melena).

DIAGNOSTIC TESTS

A. Clinical findings and history alone usually confirm the diagnosis in the vast majority of reflux sufferers.
B. *Helicobacter pylori* testing.

TABLE 14.13 COMMONLY USED MEDICATIONS THAT CAN IMPEDE ESOPHAGEAL FUNCTION, RETARD GASTRIC EMPTYING, OR CAUSE DIRECT ESOPHAGEAL MUCOSAL INJURY

Antibiotics	Bisphosphonates
Anticholinergics	Dopamine agents
Anticonvulsants	Ferrous sulfate
Antihypertensives	Lithium
Antiretrovirals	Nicotine
Ascorbic acid	Nitrates
Aspirin and NSAIDs	Potassium chloride
Benzodiazepines	Progesterone
Beta agonists	Xanthines (Theophylline)

NSAIDs, nonsteroidal anti-inflammatory drugs.

C. The 2021 ACG and CAG clinical guidelines for dyspepsia related to endoscopy state:

1. Clients 60 years or over with dyspepsia should have an endoscopy to exclude upper GI neoplasia.

2. For clients under age 60, an endoscopy should not be done to investigate alarm features for dyspepsia to exclude upper GI neoplasia.

3. Clients younger than 60 years with dyspepsia should have a noninvasive *H. pylori* test and treatment if possible. (Refer to section "Peptic Ulcer Disease" in this chapter for *H. pylori* treatment.)

D. The 2021 American Gastroenterology Association (AGA) guidelines for GERD state:

1. Endoscopy is not required for the presence of typical GERD symptoms but is recommended for the presence of alarm symptoms or for screening clients at high risk for complications.

2. Endoscopy with biopsy is usually the first diagnostic tool in cases of caustic ingestion or suspected infectious etiology.

3. The ACG does not recommend an endoscopy to establish the diagnosis of GERD-related asthma, chronic cough, or laryngitis.

E. The Lyon Consensus (2020) statement suggests various questionnaires present similar sensitivity and specificity compared with accurate gastroenterologic evaluations. Related to endoscopy, Lyons Consensus notes when GERD symptoms do not respond to empiric PPI an endoscopy is advised to both evaluate for GERD complications and to detect potential alternative diagnoses that might redirect therapy. Barrett esophagus or peptic stricturing is considered confirmatory evidence of GERD.

1. Motility and pH monitoring:

a. The ACG and CAG guidelines on motility studies state the following:

i. Recommend against routine motility studies for clients with FD.

ii. Suggest motility studies for selected clients with FD where gastroparesis is strongly suspected.

b. The AGA states ambulatory 24-hour pH monitoring: Prolonged monitoring is the best clinical tool for diagnosing GERD in asthmatics. However, it is very expensive and not universally available.

c. The Lyon Consensus states reflux monitoring demonstrates GERD pathology, evident as either excessive esophageal acid exposure time (AET) or reflux episodes, rather than the mechanics. However, wireless pH monitoring is expensive, limiting its availability.

F. Upper GI series or barium contrast radiography is not used to diagnose GERD but rules out anatomic abnormalities of the upper digestive tract.

G. Guaiac test for occult blood: Bleeding may accompany reflux esophagitis and be slow and chronic, resulting in iron deficiency anemia, or brisk, resulting in hematemesis. GERD may not be obvious to the clinician when obtaining a client history, especially in an asthma client with confounding respiratory symptoms.

DIFFERENTIAL DIAGNOSES

A. GERD.
B. Dyspepsia.
C. Cancer: gastric or esophageal.
D. Peptic ulcer.
E. Eosinophilic esophagitis (EE).
F. Esophageal spasm.
G. Myocardial infarction (MI)/angina.
H. Gallbladder disease.
I. Infections: CMV, herpes simplex virus, and *Candida.*
J. Ingestion of caustic substance.
K. Self-induced vomiting/bulimia.
L. Pyloric stenosis.
M. Autoimmune skin disorders affecting the esophagus.
N. Food allergy.

PLAN

A. General interventions:

1. Management depends on cause and severity of symptoms.

B. Client teaching: *See Client Teaching Guide for this chapter, "Gastroesophageal Reflux Disease (GERD)."*

C. Dietary management:

1. Weight loss is advised for overweight or obese clients with GERD symptoms.

2. At present, there are no supporting data for special dietary precautions; however, an elimination diet of key trigger foods helps to identify triggers.

D. Medical and surgical management:

1. The Nissen fundoplication is a surgical procedure used to treat GERD in asthmatics. It improves the antireflux barrier and provides a lasting solution. Because it is not always successful, it is reserved for severe cases. The Nissen operation is often performed as a laparoscopic procedure.

2. Surgical therapy is not recommended for clients who do not respond to PPI therapy.

3. The ACG guidelines note that surgical therapy is as effective as medical therapy for carefully selected clients with chronic GERD when performed by an experienced surgeon.

4. Clients on NSAIDs who experience upper gastric pain including reflux need to be referred for endoscopy as soon as possible.

E. Pharmaceutical therapy:

PPIs are used for dyspepsia, GERD, and EE and are considered the "gold standard" of treatments.

1. Guidelines related to PPI therapy:

a. The AGA and CAG guidelines related to PPI therapy:

i. Dyspepsia clients under the age of 60 should have empirical PPI therapy if they are *H. pyloric*-negative or remain symptomatic after *H. pylori* eradication therapy.

ii. Tricyclic antidepressants (TCA) or prokinetic therapies for clients younger than 60 with dyspepsia can be tried if a trial of PPI is ineffective or the clients did not respond to *H. pylori* eradication therapy.

iii. The ACG and CAG guidelines recommend PPI, TCA, and prokinetic therapy in this order for clients who fail therapy or are *H. pylori*-negative.

b. The AGA guidelines state the following:

i. Histamine-2 receptor antagonists (H2 blockers) are effective in managing milder, infrequent GI symptoms. Tolerance occurs with chronic use of H2 blockers. Several H2 blockers are currently available as prescription and OTC: nizatidine (Axid), famotidine (Pepcid), and cimetidine (Tagamet).

ii. Rule out cardiac/noncardiac chest pain before institution of PPI therapy (see section "Chest Pain" in Chapter 13).

iii. The target of pharmaceutical therapy is improvement in quality of life by the reduction/relief of symptoms and healing of erosive esophagitis.

iv. Initiation of a PPI should be prescribed once a day, before the first meal of the day. For maximal pH control, the traditional delayed-release PPI should be administered 30 to 60 minutes before a meal.

v. Management of GERD and dyspepsia should be tailored to the frequency, severity, and duration of symptoms. On-demand or self-directed therapy has been shown to be effective; however, a client's use and response should be evaluated.

vi. Long-term use of acid suppression therapy without a diagnosis is not advised.

vii. Long-term therapy should be titrated down to the lowest effective dose based on symptom control.

2. Adverse side effects of PPIs:

a. The potential adverse effects of acid suppression include the increased risk of community-acquired pneumonia, osteoporosis/osteopenia, vitamin B_{12} deficiency and hypomagnesium, and GI infections, including *Clostridioides difficile*-associated diarrhea.

b. Avoid the concomitant use of clopidogrel (Plavix) with PPIs due to the significant reduction of the antiplatelet activity of clopidogrel.

c. PPIs interact with warfarin, phenytoin, and diazepam:

i. Monitor the therapeutic effect of warfarin and phenytoin.

ii. Clients may experience a relapse in their GERD symptoms after discontinuance and therefore may need to be tapered off or use a stepdown approach with an antacid or an H2 blocker.

3. The ACG guidelines do not recommend the use of integrated and alternative medicines.

FOLLOW-UP

A. Noncardiac chest pain due to GERD should have a diagnostic evaluation before institution of therapy.

B. Empiric treatment with a PPI may be attempted for a short period except for clients presenting with any alarm symptoms. Schedule a return visit in 1 to 2 weeks to evaluate the relief of symptoms.

C. Clients having frequent relapses and failure to adequately respond to long-term OTC H2 blockers and PPI use should have an endoscopic evaluation.

D. The need for prescribed long-term PPI treatment or the presence of alarm symptoms requires a gastroenterology consultation.

E. Consider bone density studies for clients with long-term PPI use.

EMERGENT ISSUES/INSTRUCTIONS

A. Alarm symptoms:

1. Dysphagia (difficulty swallowing).
2. Unintentional weight loss.
3. Predominant upper abdominal pain.
4. Hematemesis (vomiting blood).
5. Melena (black feces/blood stool).
6. Odynophagia (painful swallowing).
7. Severe symptoms.

B. Clients on NSAIDs who experience upper gastric pain including reflux need to be referred for endoscopy as soon as possible.

CONSULTATIONS

A. Referral is necessary if the client fails to improve after trying two different medications, or if the client has dysphagia, recent weight loss, or blood loss.

B. PPI nonresponders need to be referred for evaluation.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Underlying causes in pregnancy are diminished gastric motility and displacement of stomach by enlarging uterus.

2. Rule out pregnancy-induced hypertension (PIH); evaluate the client immediately for signs and symptoms of sudden-onset discomfort with no relief from antacids (PIH or HELLP [hemolysis, elevated liver enzymes, and low platelets] syndrome).

B. Geriatrics:

1. GERD prevalence increases with age and may be associated with a hiatal hernia.

2. Prolonged reflux results in esophagitis and may lead to stricture development. Chronic recurrence may develop into Barrett syndrome.

3. Treatment of GERD is the same as for general adults; however, do diagnostic testing in short time sequence secondary to stricture and cancer in the older adults.

4. Atypical symptoms are common presentations in the older adults:

a. Vomiting.
b. Anorexia.
c. Dysphagia.
d. Respiratory symptoms.
e. Belching.
f. Hoarseness.
g. Postprandial fullness.

5. Beers Criteria cautions for PPIs:

a. Due to polypharmacy, a full review of medications, herbals, and OTC drugs should be undertaken to identify medications that should be avoided in the geriatric population, duplicate medications, and medications that are able to be decreased or deprescribed. Evaluate if the client has difficulties with chewing or if the client is altering medications by crushing due to swallowing difficulties. Some medications should not be crushed because it alters the absorption or stability of the medication, causes an irritant effect, or has unacceptable taste. (Refer to section on deprescribing in Chapter 3, "Adult-Geriatric Assessments.")

b. **When prescribing to older adults, use the lowest dose, for the shortest period of time.**

c. PPIs interact with medications that are commonly taken in the older population, including warfarin, phenytoin, and diazepam. PPIs also reduce the antiplatelet effect of clopidogrel:

i. Monitor the therapeutic effect of warfarin and phenytoin.

d. PPIs are on the American Geriatrics Society 2019 Beers Criteria for potential inappropriate medication use in older adults. (Refer to Appendix C, Table C.1.) Avoid scheduled use of PPIs for greater than 8 weeks unless needed for high-risk clients. PPIs are associated with the following:

i. Osteoporosis/osteopenia, prolonged exposure may increase of fractures.
ii. Community-acquired pneumonia.
iii. *C. difficile* infections and diarrhea.
iv. Vitamin B_{12} deficiency and hypomagnesium.

e. See Appendix D for the PPI deprescribing algorithm from deprescribing.org.

RESOURCES

American Gastroenterological Association: The 2021 guidelines for the diagnosis and management of gastroesophageal reflux disease are available online at: gastro.org/clinical-guidance/personalized-approach-to-the-evaluation-and-management-of-gastroesophageal-reflux-disease-gerd/

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GIARDIA INTESTINALIS

DEFINITION

A. *Giardia intestinalis* is the leading parasitic cause of diarrhea. Infestation can lead to malabsorption by coating large areas of the small bowel, particularly the lower duodenum and upper jejunum. Most people infected with *G. intestinalis* remain asymptomatic, and most infections are self-limited.

INCIDENCE

A. Giardiasis has a worldwide distribution and is especially prevalent in the United States and overseas. It is common in areas where water supplies are contaminated by human sewage. The age-specific prevalence of giardiasis is highest in children 1 to 5 years and in adults 35 to 39 years of age. The peak onset occurs annually during early summer through early fall.

PATHOGENESIS

A. *G. intestinalis* is a flagellated protozoan. The infective form is the cyst. Humans are the principal reservoir of infection, but *Giardia* can infect dogs, cats, beavers, and other animals that can contaminate water with feces containing cysts.

B. People become infected either directly, by hand-to-mouth transfer of cysts from feces of an infected person (e.g., childcare), or indirectly, by ingestion of fecal-contaminated water or food. Most community-wide epidemics result from contaminated water supplies.

C. The incubation period is 1 to 3 weeks, with an average of 7 to 10 days. The infective form is the cyst with infection limited to the small intestine and the biliary tract. Disease is communicable for as long as the infected person excretes cysts.

D. Symptoms may last 2 to 6 weeks in otherwise healthy people.

PREDISPOSING FACTORS

A. 50% to 75% of outbreaks occur in childcare settings.
B. Travel to endemic areas.
C. Subjection to unsanitary food handling.
D. Exposure to contaminated water supplies (lakes, rivers, springs, ponds, streams, or wells):
1. Swallowing water from a swimming pool that is underchlorinated.
E. Hikers and campers.
F. Oral/anal and oral/genital sexual contact.
G. Immunocompromised individuals are at high risk.

COMMON COMPLAINTS

A. Acute complaints:
1. Explosive, foul-smelling diarrhea.
2. Mucus in stools, bulky stools.
3. Upper abdominal pain or discomfort.
4. Flatulence.
5. Nausea.
6. Anorexia.
7. Weight loss.

OTHER SIGNS AND SYMPTOMS

A. Chronic complaints:
1. Intermittent loose stools (but not diarrhea).
2. Steatorrhea.
3. Increased flatulence or distension.
4. Vague abdominal discomfort.
5. Fatigue related to anemia.
6. Profound weight loss (10%–20% of body weight).
7. Malabsorption.
8. Urticaria.

SUBJECTIVE DATA

A. Review onset, duration, and course of symptoms. Is diarrhea acute or chronic?
B. Ask the client about travel to areas known for giardiasis.
C. Work exposure such as day-care or orphanage worker.
D. Review recreational exposure to water sources, swimming pools, lakes, ponds, rivers, and so forth.
E. Review the client's intake of medications and other substances that can cause diarrhea, especially antibiotics, laxatives, quinidine, magnesium-containing antacids, excess alcohol, caffeine, herbal teas, digitalis, loop diuretics, antihypertensive agents, and sorbitol-containing (sugar-free) gums and mints.
F. Review the nature of the client's bowel movements (BMs), including frequency, consistency, volume, and presence of blood, pus, or mucus.
G. Does diarrhea have any relationship to meals? Onset of diarrhea within hours of ingesting a potentially contaminated food is suggestive of bacterial infection such as *Escherichia coli;* this is confirmed by checking if others were similarly affected.
H. Ask the client about associated symptoms that need evaluation, such as fever, abdominal pain, or rash.
I. Ask the client if other family members or sexual contacts are also ill.
J. Establish the client's normal weight, and if any weight has recently been lost, review amount and over what period of time.

PHYSICAL EXAMINATION

A. The physical examination may reveal no specific finding. ▶

B. Check temperature, pulse, respirations, blood pressure (BP). Measure height and weight to calculate body mass index (BMI).
C. Inspect:
1. General appearance for signs of dehydration; include evaluation of mucous membranes.
D. Auscultate:
1. Abdomen for bowel sounds in all quadrants.
E. Palpate:
1. Palpate the abdomen for masses, tenderness, guarding, and rebound. Clients with periumbilical or right lower quadrant (RLQ) pain and copious volumes of watery stool are likely to have a small bowel etiology.
2. Palpate lymph nodes for enlargement.
F. Rectal exam.

DIAGNOSTIC TESTS

A. Routine blood tests, complete blood count (CBC), and electrolytes are usually normal.
B. The Centers for Disease Control and Prevention (CDC) recommends collecting three stool specimens from clients over several days for more accurate test results to rule out other pathogens. **Parasites are passed intermittently, so examine stools on alternating days.**
C. Test of choice is direct fluorescence antibody (DFA); also available: enzyme immunolinked assays (EIAs) kit, microscopy with trichrome staining.
D. Mucus stool for leukocytes: Mucus free of leukocytes is the hallmark of irritable bowel syndrome (IBS); a large number of white cells suggests inflammatory or invasive diarrhea.
E. Endoscopy to identify cyst in duodenal fluid or small bowel tissue.

DIFFERENTIAL DIAGNOSES

A. Giardiasis.
B. Malabsorption.
C. *E. coli* infection.
D. IBS.
E. Viral diarrhea.
F. Lactose intolerance.
G. Other bacterial infections, such as *Shigella, Salmonella*, and *Campylobacter.*
H. Crohn's disease (CD).
I. Sprue, celiac disease, gluten intolerance.

PLAN

A. General interventions:
1. Advise adult workers with diarrhea to stay away from day-care centers until they become asymptomatic.
2. Advise the client's household and sexual contacts to seek medical examination and treatment.
B. Client teaching: *See Client Teaching Guide for this chapter, "Diarrhea."*
1. Discuss safe sexual practices.
2. Recommend contact precautions for duration of illness for diapered and/or incontinent children.
3. People with diarrhea caused by *Giardia* should not use recreational water venues, including swimming pools, for 2 weeks after symptoms resolve.
4. Avoidance of alcohol during treatment should be avoided secondary to disulfiram-like effect.
C. Dietary management:
1. Instruct the client or caregiver to prevent dehydration from diarrhea by increasing fluids.
2. Advise restricting milk products to rule out lactose intolerance. Post-*Giardia* lactose occurs in up to 40% of clients.
3. Instruct backpackers, campers, and people likely to be exposed to contaminated water to avoid drinking directly from streams. To make water safe for drinking, boil the water, or use chemical disinfection or filtration. Boiling water is the most reliable method to make water safe for drinking.
D. Pharmaceutical therapy:
1. Treatment of asymptomatic carriers is not generally recommended.
2. Tinidazole and nitazoxanide are the drugs of choice for treatment:
a. Tinidazole (Tindamax):
i. Adults: 2-g single dose.
ii. Only available in tablet form; it may be crushed by a pharmacist and mixed with flavored syrup.
b. Nitazoxanide (Alinia) has the advantage of treating other intestinal parasites:
i. Age 12 years and older: 500 mg tablet Q12H or 25 mL oral suspension Q12H with food for 3 days.
3. Metronidazole (Flagyl): duration of treatment 5 to 7 days:
a. 500 mg orally BID for 5 to 7 days. OR
b. 250 mg orally TID for 5 to 7 days.
c. For lactating females, alternative dosing is a 2-g single dose, with interruption of breasting feeding for 12 to 24 hours.
d. Available in tablets; oral suspension may be prepared by a pharmacist.
4. Albendazole:
a. 400 mg daily for 5 days.
b. Available in tablets or suspension.
c. Active against other parasites.
5. Paromomycin (Humatin), a nonabsorbable aminoglycoside, is recommended for treatment of symptomatic infection in pregnant females in the second and third trimesters:
a. Adults: 10 mg/kg orally TID for 5 to 10 days.
6. Furazolidone: 100 mg orally QID for 7 to 10 days.
7. Quinacrine: 100 mg orally TID for 5 days.

FOLLOW-UP

A. Relapses after treatment are common, especially in immunocompromised clients:
1. Persistent symptoms may be attributed to drug resistance, noncompliance with dosing, or immunosuppression.
B. If diarrhea persists for 2 weeks or more, secondary evaluation is indicated. Stools should be examined again for blood, leukocytes, and parasites.
C. A repeat course of antimicrobials may be needed for clients with an established diagnosis of giardiasis.
D. Schedule follow-ups at 6 weeks and 6 months after treatment, as indicated.
E. Giardiasis is a nationally notifiable disease. Cases should be reported to the local or state health departments.

CONSULTATION/REFERRAL

A. Consult a physician if the client has no relief of symptoms after completion of therapies.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Treatment of clients during pregnancy is recommended. Giardiasis in pregnancy is associated with dehydration, malabsorption, or severe symptoms.

2. Malabsorptive symptoms may persist because regeneration of functioning intestinal mucosa requires time.

3. Medications to treat giardiasis in pregnancy would include metronidazole and nitazoxanide; the agents that should not be used is Tindamax (first trimester) and no use of quinacrine, Humatin, albendazole, and use with caution furazolidone.

B. Geriatrics:

1. Anemia, weight loss, and anorexia have been the most prominent signs in older adults.

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HEMORRHOIDS

DEFINITION

A. Hemorrhoids are clusters of vascular tissues, smooth muscle, and connective tissue of the anal canal. Internal hemorrhoids are above the anorectal line and covered by rectal mucosa. Hemorrhoids can be found at any position of the rectum. Internal hemorrhoids are graded by severity (Table 14.14).

B. External hemorrhoids are below the anorectal line, covered by anal skin, and appear as painless, flaccid skin tags (Figure 14.2).

C. Complications of hemorrhoids include thrombosis, secondary infection, ulceration, abscess, and incontinence.

D. Although rectal bleeding is commonly associated with hemorrhoids, it may be a symptom of other disease processes, such as colorectal cancer (CRC), inflammatory bowel disease (IBD), other colitides, diverticular disease, and angiodysplasia.

INCIDENCE

A. The incidence of hemorrhoids is estimated at 4.4% worldwide. Clients tend to present after utilization and failure of over-the-counter (OTC) treatments. Hemorrhoids are common in people between 45 and 65 years of age. They are uncommon in people younger than age 20, except secondary to pregnancy.

TABLE 14.14 SEVERITY OF HEMORRHOIDS USED TO GUIDE TREATMENT OPTIONS

Grade	Severity
I	The hemorrhoids bleed but do not prolapse.
II	The hemorrhoids prolapse upon defecation but reduce spontaneously.
III	The hemorrhoids prolapse upon defecation and must be reduced manually.
IV	The hemorrhoids are prolapsed and cannot be reduced manually.

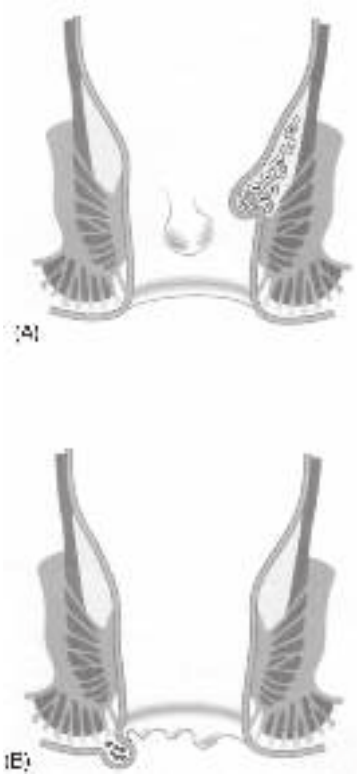


FIGURE 14.2 Internal and external hemorrhoids. (A) Internal hemorrhoid: Covered by a thin sheet of tissue called mucous membrane, an internal hemorrhoid bulges into the rectal opening and may sink a bit during bowel movements. (B) External hemorrhoid: Covered by skin, an external hemorrhoid protrudes from the rectum.

PATHOGENESIS

A. External hemorrhoids develop from the ectoderm and are covered by squamous epithelium.

B. Internal hemorrhoids develop from the embryonic endoderm and are lined with columnar epithelium of anal mucosa.

C. Mechanism is unknown. Prolapse may be initiated by shearing force from passage of large firm stool, by increased venous pressure from portal hypertension or pregnancy, or by straining that occurs with lifting or defecation.

PREDISPOSING FACTORS

A. Pressure and straining with stool associated with constipation.
B. Chronic diarrhea.
C. Pelvic congestion.
D. Poor pelvic musculature.
E. Pregnancy.
F. Episiotomy.
G. Portal hypertension, cirrhosis.
H. Low-fiber diet.
I. Sedentary jobs.
J. Loss of muscle tone due to advanced age.
K. Anal intercourse.
L. Obesity.
M. Colon malignancy.
N. Rectal surgery.
O. Inflammatory bowel disease.

COMMON COMPLAINTS

A. Cardinal features:
  1. Bleeding: painless, bright red bleeding with defecation (internal).
  2. Anal pruritus.
  3. Prolapse.
  4. Pain related to thrombosis.

OTHER SIGNS AND SYMPTOMS

A. Visible prolapsed mass.
B. Incomplete defecation.
C. Leakage of feces (internal hemorrhoids).
D. Excessive moisture.
E. Weakness or fatigue, with anemia.

SUBJECTIVE DATA

A. Review onset and duration of symptoms, especially history of rectal bleeding, prolapse, issues of hygiene, and pain.
B. Review the quantity, color, and timing of any rectal bleeding (bright red, dripping, streaks on toilet paper).
C. Review the client's history of hemorrhoids and treatments, including surgery.
D. Ask the client about recent pregnancy, liver disease, and constipation.
E. Inquire about the client's job and level of daily activity.
F. Review the client's sexual practices for anal intercourse.
G. Review the client's dietary history for fluid intake and sources/amount of fiber.
H. Ask about bowel habits, including frequency, consistency, and ease of evacuation.
I. Review a detailed family history, with emphasis on intestinal disease.
J. Review client's history of coagulation and immune status.

PHYSICAL EXAMINATION

A. Check temperature (if indicated), pulse, respirations, and blood pressure (BP). Measure height and weight to calculate body mass index (BMI).
B. Inspect:
  1. Observation of rectal area for skin tags, prolapse, irritation, fissures, and condyloma:
    a. Internal hemorrhoids are usually not visible unless prolapsed.
    b. External hemorrhoids protrude with straining or standing.
    c. In order to observe and evaluate hemorrhoids/prolapse, ask the client to strain down like they are having a bowel movement.
  2. Using anoscopy, visualize internal rectum for hemorrhoids, masses, or fissures (approximately 20% have concomitant anal fissures).
C. Palpate:
  1. Palpate abdomen for masses and tenderness.
  2. Internal hemorrhoids are usually not palpable unless thrombosed.
D. Rectal examination:
  1. Perform digital rectal examination. (Topical anesthetics may be needed to reduce discomfort from the examination.)
    a. Evaluate rectal tone.
    b. Palpate the prostate.
E. Neurologic examination:
  1. Evaluate the presence of an anal wink (reflexive contraction of the external anal sphincter upon stroking of the skin).

DIAGNOSTIC TESTS

Full evaluation of the large bowel with colonoscopy is recommended for clients with significant abdominal symptoms, weight loss, change in bowel habits, age older than 50, or other risk factors for colonic malignancy.

A. Complete blood count (CBC; hematocrit [Hct] and hemoglobin [Hgb] to evaluate anemia if bleeding is present and also evaluates presence of infection).
B. Anoscopy reveals internal hemorrhoids as bright red to purplish bulges. **Digital rectal examination alone can neither diagnose nor exclude internal hemorrhoids; anoscopy is mandatory.**
C. Sigmoidoscopy and/or flexible sigmoidoscopy.
D. Colonoscopy or CT colonography is reserved for bleeding atypical from hemorrhoids.
E. Air-contrast barium enema (BE) for atypical bleeding.

DIFFERENTIAL DIAGNOSES

A. Hemorrhoids.
B. Condyloma acuminata.
C. Rectal prolapse.
D. Rectal bleeding due to one of the following:
  1. Colorectal cancer.
  2. Polyps.
  3. Anal fissure.
  4. Fistula.
  5. Perianal abscess.
  6. IBD, including ulcerative colitis (UC) and Crohn's disease (CD).
  7. Diverticulitis.
  8. Pelvic tumor.

PLAN

A. General interventions:
  1. No treatment is necessary if the client is asymptomatic, except for maintaining regular bowel habits and performing comfort measures.
B. Client teaching: *See Client Teaching Guides for this chapter, "Hemorrhoids" and "Tips to Relieve Constipation."*
C. Dietary management:
  1. High-fiber diet and an adequate fluid intake are recommended by the American College of Gastroenterology (ACG) as the first-line treatment. High-fiber diet and adequate fluid intake should be continued indefinitely to maintain a soft bulky stool that can be passed without straining (see Appendix B, "High-Fiber Diet").
D. Medical and surgical management:
  1. Use warm sitz baths up to three times a day for irritation and pruritus.
  2. Conservative treatment for thrombosed hemorrhoid includes lying prone and applying ice pack to area.
  3. Incision and evacuation of thrombosis or clot may be performed under local anesthesia. Other treatments for thrombosed hemorrhoids noted in clinical trials have included the following:
    a. Topical nitroglycerin 0.2% topical ointment for temporary analgesia. The most common side effect was headache.
    b. Topical nifedipine: Use with caution due to hypotension.
    c. Injection of botulinum toxin has been used for treatment of chronic anal fissures if no response to conservative or pharmacologic treatment.

4. Symptomatic grade I, grade II, and some grade III hemorrhoids may be treated by the following:

a. Rubber band ligation is the treatment of choice for grades I and II hemorrhoids. Rubber band ligation is the most widely used and is associated with fewer complications than surgery:

i. Rubber band ligation is generally contraindicated in clients with advanced cirrhosis due to the risk of profound secondary bleeding following the procedure. Injection sclerotherapy and hemorrhoidectomy are used in refractory cases.

ii. Rubber band ligation is not recommended in clients with current use of anticoagulant or antiplatelet drugs due to the risk of secondary bleeding. Conservative therapy is the mainstay.

b. Bipolar, infrared, and laser coagulation (may require more than one treatment).

c. Sclerotherapy and cryotherapy are used infrequently.

d. Stapled hemorrhoidopexy can be performed in clients with grade III hemorrhoids.

5. External hemorrhoids usually do not require surgical therapy except in cases of thrombosis. For selective grade III and grade IV internal and strangulated hemorrhoids that fail medical and nonoperative therapies, surgical treatment is required:

a. **Stapled hemorrhoidopexy has a faster recovery but has a higher recurrence rate.**

b. **Hemorrhoidectomy is the treatment of last choice because it requires hospitalization and an extended recovery period, and it risks compromising competence of the anal sphincter.**

6. Hemorrhoidectomy complications include the following:

a. Urinary retention.
b. Urinary tract infection (UTI).
c. Fecal impaction.
d. Pain.
e. Hemorrhage.
f. Stricture formation (1%) or sphincter damage (rare).
g. Nonhealing wound.
h. Fistula formation.
i. Anal leakage.

7. AIDS is a contraindication to nonsurgical procedures; conservative therapy is recommended, especially if immunosuppressed.

E. Pharmaceutical therapy:

1. Drug of choice: bulk-forming agents such as psyllium seed (Metamucil), methylcellulose (Citrucel), or calcium polycarbophil (Fibercon), one to three teaspoons in 8 oz of liquid TID. Maintenance dose is one to three teaspoons after dinner.

2. Plant-based soluble fiber, (Benefiber) containing wheat dextrose and cellulose, 2 teaspoons powder with 4 to 8 oz of water 2 to 3 x a day or 3 tablets up to three times a day.

3. Stool softener: docusate sodium (Colace) or docusate calcium (Doxidan) 100 mg TID.

4. For irritation and pruritus, topical creams and anesthetics are found in OTC products such as Anusol, pramoxine HCl (Tronolane Cream), Preparation H, and topical hydrocortisone preparations.

5. Acetaminophen is the drug of choice for pain.

FOLLOW-UP

A. None necessary if resolution occurs and the client is asymptomatic.

B. Reevaluate the client in 2 weeks for further treatment if symptoms persist.

C. Evaluate the client with an intervention in 7 to 10 days.

EMERGENT ISSUES/INSTRUCTIONS

A. The onset of urinary retention and fever immediately after an office-based procedure may be the initial sign of perianal sepsis and mandates emergent client evaluation.

CONSULTATION/REFERRAL

A. Refer the client with acute thrombosis of external hemorrhoids to a physician.

B. Refer the client to a colorectal surgeon if hemorrhoids bleed repeatedly, prolapse, produce intractable pain, or are thrombosed, and if 3 to 5 consecutive days of treatment do not provide relief.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Hemorrhoids are common in pregnancy. Most clients revert to their previous asymptomatic state after pregnancy.

2. Conservative treatment is recommended with excision of thrombosed external hemorrhoids only if necessary.

3. Surgical treatment is contraindicated in pregnancy due to the risk of inducing labor.

4. Labor, which results in pressure on the pelvic floor by the presenting part of the fetus and the expulsive efforts of the mother, may aggravate hemorrhoids, causing protrusion and inflammation during the puerperium. Hemorrhoids may be pushed back after delivery to prevent them from becoming swollen and painful.

B. Geriatrics:

1. Hemorrhoids are common in older adult with intermittent and low-volume hematochezia, which often only coats the stool.

2. Prolapse of rectal mucosa is more common in older adults.

3. Older adults are at an increased risk of lower gastrointestinal (GI) bleeding secondary to the increased incidence of comorbid illness and more polypharmacy (anticoagulants and nonsteroidal anti-inflammatory drugs [NSAIDs]). Colonoscopy is recommended in the geriatric population to exclude malignancy or other underlying disease.

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HEPATITIS A

DEFINITION

A. Hepatitis A is an acute self-limited illness with inflammation of the liver caused by a viral infection. Hepatitis A virus (HAV) is spread by viral shedding. Acute cases of hepatitis A are reportable to the public health department.

B. Case definition: an acute illness with a discrete onset of any sign or symptoms consistent with acute viral hepatitis (e.g., fever, headache, malaise, anorexia, nausea, vomiting, diarrhea, ▶

and abdominal pain) AND either jaundice or elevated serum alanine aminotransferase (ALT) or aspartate transaminase (AST) levels.

C. The average incubation period is 28 days (range 15–50 days).

D. The highest titers of HAV in the stool of infected clients occur 1 to 2 weeks before onset of illness (jaundice or elevation of liver enzymes), during which time clients are most likely to transmit infection. Risk subsequently diminishes and is minimal in the week after onset of jaundice. Up to 70% of clients will have jaundice. Persons with chronic liver disease (CLD) are at risk for fulminant hepatitis.

E. Duration of HAV infection is typically 8 weeks, but prolonged disease, as long as 6 months, can occur in 10% to 15% of symptomatic clients.

F. The major methods of prevention include improved sanitation of water sources, improved hygiene practice prior to food preparation and diaper changes, immunization with the hepatitis A vaccine, and administration of immune globulin (IG).

G. Hepatitis A does not reoccur because immunoglobulin G (IgG) antibodies to HAV provide lifelong protection.

INCIDENCE

A. From 30% to 35% of acute hepatitis cases in the United States are due to HAV. In developing countries, where infection is endemic, most people are infected during the first decade of life. Multistate HAV outbreaks have recently occurred in the United States:

1. September 2021: Famous Anthony's restaurants, 14 infected people ate at three of the sites according to the Virginia Department of Health.

2. August 2016: multiple states, linked to frozen strawberries and in Hawaii linked to raw scallops.

3. March 2017 to 2018: multiple states among people who used drugs and/or people who are homeless.

B. Outbreak in custodial institutions accounts for about 10% to 15% of reported HAV in the United States.

C. The incidence of mortality from HAV is 0% to 2%. The single most important determinant of illness severity is age; a direct correlation between increasing age and the likelihood of adverse events is present. Most deaths from acute HAV occur in persons older than 50 years.

PATHOGENESIS

A. HAV is a small, RNA enterovirus classified as a member of the picornavirus group. Viral replication depends on hepatocyte uptake and synthesis, and assembly occurs exclusively in liver cells. Transmission of HAV is person-to-person, primarily by the fecal–oral route and parenterally. The incubation period is 15 to 50 days, with an average of 25 to 30 days.

B. Common-source, food- and waterborne epidemics have occurred, including several caused by shellfish contaminated with human sewage. Nosocomial outbreaks have occurred as a result of shedding of HAV from infected, asymptomatic neonates, children, or adults.

PREDISPOSING FACTORS

A. Primarily, infections in the United States result from travel to another country where HAV transmission is common.

B. Person-to-person transmission contact through the fecal–oral route:

1. Contact with a child who attends a childcare center (especially with children in diapers).

2. Sex among men who have sex with men (MSM).

3. Vertical transmission from mother to fetus (limited).

4. Personal contact with a newly arriving international adoptees with high or intermediate hepatitis A endemicity.

5. Injection and noninjection drug users.

C. Exposure to contaminated food or water:

1. Uncooked foods contaminated with HAV.

2. Cooked foods that are not heated to temperatures that are capable of killing the virus.

3. Foods contaminated after cooking (infected food handlers).

4. Freezing does not inactivate HAV.

5. Waterborne outbreaks from poor sanitation. In the United States, outbreaks of HAV caused by flooding have not been documented.

COMMON COMPLAINTS

A. Malaise.

B. Anorexia.

C. Nausea.

D. Low-grade fever.

E. Jaundice: icteric phase (70% of older children and adults):

1. Tea-colored urine.

2. Clay-colored stool.

3. Abdominal pain.

4. Pruritus.

5. Enlarged liver.

F. Fatigue.

G. Joint pain.

OTHER SIGNS AND SYMPTOMS

A. Adults: severe, prolonged course with fatigue, headache, vomiting, and symptoms noted earlier.

B. Relapsing hepatitis A is more common in older adults. There generally has been a protracted course of symptoms and a relapse of symptoms following an apparent resolution.

SUBJECTIVE DATA

A. Review duration, onset, and severity of symptoms, including specifics about urine or stool color changes.

B. Ask the client about family members and sexual contacts with similar symptoms.

C. Review the client's history of blood transfusions, intravenous (IV) drug use, and alcohol abuse.

D. Inquire about occupational exposure.

E. Ask the client about recent international travel, eating off street carts potentially not sanitized or exposure to newly arrived international adoptees.

F. Review immunization status.

G. Review medications for a possible Tylenol overdose or ecstasy use as a cause of acute drug-induced liver injury.

PHYSICAL EXAMINATION

A. Check temperature (acute illness), pulse, respirations, blood pressure (BP), and weight.

B. Inspect:

1. Note general appearance.

2. Inspect the skin for slight jaundice or rash.

3. Inspect mucous membranes and nail beds.

4. Inspect eyes for yellow sclera.

C. Auscultate:

1. Lung fields, all quadrants of the abdomen.

2. Heart.

▶

D. Percuss:
  1. Abdomen.
E. Palpate:
  1. Palpate all quadrants of the abdomen for masses, liver tenderness, and hepatosplenomegaly (about 10% of cases).

DIAGNOSTIC TESTS

A. Nucleic acid testing (NAT) is the gold standard for diagnosis of viremic stages of hepatitis infection.
B. Because the clinical characteristics are the same for all types of acute viral hepatitis, HAV must be confirmed by a positive serologic test for immunoglobulin M (IgM) antibody to HAV (anti-HAV) or the case must meet the clinical case definition and occurs in a person who has an epidemiologic link with a person who has laboratory-confirmed hepatitis A (e.g., household or sexual contact with an infected person during the 15–50 days before the onset of symptoms):
  1. Serum IgM presents at onset of illness and disappears within 4 months, generally indicating current or recent infection. However, it may persist for 6 months or longer.
  2. Presence of IgG anti-HAV antibodies without virus-specific IgM indicates past infection and immunity.
C. Liver function studies, including ALT, AST, lactate dehydrogenase (LDH), and alkaline phosphatase (ALP).
D. Bilirubin, direct and indirect.
E. Complete blood count (CBC).
F. Prothrombin time (PT).
G. Imaging studies are usually not indicated for hepatitis A infection. An ultrasound may be used to exclude other pathology.

DIFFERENTIAL DIAGNOSES

A. HAV.
B. Mononucleosis.
C. Cancer.
D. Obstructive jaundice.
E. Alcoholic hepatitis or cirrhosis.
F. Hepatotoxic drug use:
  1. Drug-induced liver injury (e.g., excessive Tylenol, Ecstasy).
  2. Drug-induced hypersensitivity reaction (e.g., sulfasalazine hypersensitivity).
G. Food poisoning.
H. Exclusion of other hepatitis types.
I. Cytomegalovirus (CMV).
J. Acute HIV infection.

PLAN

A. General interventions:
  1. Contact precautions are recommended for diapered and/or incontinent clients for 1 week after onset of symptoms.
  2. Children and adults with acute HAV infection should be excluded from school, work, and childcare centers for 1 week after onset of illness.
  3. Hepatitis A is self-limiting and does not require therapy. Treatment is supportive:
    a. Limit activities secondary to malaise.
    b. Alcohol consumption is not advised.
    c. Adults who work as food handlers should not work for 1 week after onset of the illness.
  4. Encourage strict handwashing.
B. Client teaching: *See Client Teaching Guide for this chapter,"Jaundice and Hepatitis."*
  1. Teach the client that major methods for prevention are improved sanitation (e.g., of water sources and in food preparation) and personal hygiene.
  2. Food and travel precautions include the following:
    a. Avoid uncontrolled water resources: Use bottled water, boil water, or add iodine to inactivate the virus.
    b. Avoid raw shellfish.
    c. Avoid uncooked foods.
    d. All fruits should be washed and peeled.
  3. HAV can be killed by cleaning with a freshly prepared solution of 1:100 dilution of bleach to water.
C. Dietary management:
  1. Encourage optimum nutrition.
D. Pharmaceutical therapy:
  1. HAV vaccine preexposure is preferred in all populations unless contraindicated.
  2. Immune globulin (IG; GamaSTAN S/D): preexposure administration is 0.02 mL/kg, two doses intramuscularly (IM).
  3. Travel preexposure prophylaxis to endemic areas:
    a. Up to 1 month of travel: 0.1 mL/kg.
    b. Up to 2 months of travel: 0.2 mL/kg.
    c. 2 months of travel or longer: repeat dose of 0.2 mL/kg every 2 months.
  4. Persons who have been exposed recently to HAV and who have not been vaccinated should be administered one dose of single-antigen hepatitis A vaccine or IG as soon as possible, **within 2 weeks after exposure:**
    a. Healthy persons aged ≥12 months who have been exposed to HAV within the prior 14 days and have not previously completed the two-dose HepA vaccine series should receive a single dose of HepA vaccine as soon as possible. In addition to HepA vaccine, IG (0.1 mL/kg) may be administered to persons aged >40 years depending on the providers' risk assessment. For long-term immunity, the HepA vaccine series should be completed with a second dose at least 6 months after the first dose; the second dose is not necessary for postexposure prophylaxis (PEP).
    b. Persons aged ≥12 months who are immunocompromised and persons with chronic liver disease who have been exposed to HAV within the prior 14 days and have not previously completed the two-dose HepA vaccine series should receive both IG (0.1 mL/kg) and HepA vaccine simultaneously in a different anatomic site as soon as possible after exposure. For long-term immunity, the HepA vaccine series should be completed with a second dose at least 6 months after the first dose; the second dose is not necessary for PEP.
    c. For infants aged <12 months and persons for whom vaccine is contraindicated (who are allergic to a vaccine component) should receive IG (0.1 mL/kg) instead of vaccine as soon as possible and within 2 weeks after exposure.
    d. Postexposure prophylaxis with IG is also recommended for the following:
      i. Household and sexual contacts of infected persons.
      ii. Newborn infants of HAV-infected mothers.
      iii. Childcare center staff, children, and their household contacts.
      iv. Students when transmission within school is documented.
      v. Staff in institutions and hospitals.

TABLE 14.15 DOSAGES AND SCHEDULES FOR HEPATITIS A VACCINES

HAVRIX				
Licensed dosages and schedules for HAVRIX[a]				
Age	**Dose (ELISA units)[b]**	**Volume (mL)**	**Number of Doses**	**Schedule (Months)[c]**
12 months–18 years	720	0.5	2	0, 6–12
≥19 years	1,440	1.0	2	0, 6–12
VAQTA				
Licensed dosages and schedules for VAQTA[d]				
Age	**Dose (U)[e]**	**Volume (mL)**	**Number of Doses**	**Schedule (Months)[f]**
12 months–18 years	25	0.5	2	0, 6–18
≥19 years	50	1.0	2	0, 6–18
TWINRIX				
Licensed dosages and schedules for TWINRIX[g]				
Age	**Dose (ELISA Units)[h]**	**Volume (mL)**	**Number of Doses**	**Schedule[i]**
≥18 years	720	1.0	3	Standard dosing schedule 0, 1, 6 months
≥18 years	720	1.0	4	Accelerated dosing schedule 0, 7, 21–30 days + 12 months

[a]Hepatitis A vaccine, inactivated, GlaxoSmithKline.

[b]Enzyme-linked immunosorbent assay units.

[c]0 months represents timing of the initial dose; subsequent numbers represent months after the initial dose.

[d]Hepatitis A vaccine, inactivated, Merck & Co., Inc.

[e]Units.

[f]0 months represents timing of the initial dose; subsequent numbers represent months after the initial dose.

[g]Combined hepatitis A and hepatitis B vaccine, inactivated, GlaxoSmithKline.

[h]Enzyme-linked immunosorbent assay units.

[i]This four-dose schedule enables clients to receive three doses in 21 days; this schedule is used prior to planned exposure with short notice and requires a fourth dose at 12 months.

vi. People who ingested HAV-contaminated food or water within 2 weeks of last exposure.

5. Vaccines:
- a. Two inactivated HAV vaccines, Havrix and Vaqta, are available in the United States.
- b. Twinrix is a combination hepatitis A (Havrix) and hepatitis B (Engerix-B) vaccine available in the United States for ages 18 years or older (Table 14.15).

6. HAV vaccine is recommended for the following:
- a. Children 2 years and older in defined and circumscribed communities with high endemic rates and/or periodic outbreaks of HAV infection.
- b. Clients with chronic lung disease.
- c. Homosexual and bisexual males.
- d. Users of injections and illicit drugs.
- e. Those with occupational risk of exposure, such as handlers of nonhuman primates and persons working with HAV in a laboratory setting.
- f. Travelers who need preexposure immunoprophylaxis.
- g. Clients with clotting factor disorders such as hemophilia.

7. HAV vaccine is potentially indicated for the following:
- a. Childcare center staff and children.
- b. Clients and staff in custodial care institutions.
- c. Hospital personnel.
- d. Food handlers; **however, routine HAV for food handlers is not recommended.**
- e. Clients with hemophilia.

8. HAV vaccine may be administered simultaneously with other vaccines, but it should be given in a separate syringe and at a separate site.

9. The need for an additional hepatitis A booster beyond the two-dose primary immunizations has not been established.

10. Immune response in immunocompromised clients such as those with HIV may be suboptimal. The vaccine ▶

is inactivated; therefore, no special precautions are needed when vaccinating immunocompromised clients.

11. Vaccine side effects are generally mild and may include the following:

a. Local pain at the immunization site.

b. Localized induration at the injection site.

12. Tylenol may be administered for fever and arthralgia but is strictly limited to a maximum dose of 3 to 4 g/d in adults.

13. Antiemetic agents as needed for nausea and vomiting.

FOLLOW-UP

A. Dehydration may require hospital admission.

B. Follow-up in 2 weeks for reevaluation.

C. Check for hepatitis B immunity and vaccinate.

D. Acute hepatitis A is reportable to the public health department.

E. Prolonged cholestasis may follow an acute infection.

CONSULTATION/REFERRAL

A. Consult a physician if necessary.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Pregnant females recently exposed to HAV should receive prophylactic gamma globulin.

2. HAV is an inactivated vaccine and is considered safe during pregnancy.

3. HAV infection during pregnancy is associated with increased risk of premature labor and delivery.

B. Adults:

1. In the United States, the lowest rate of HAV infection is in people older than 40.

C. Geriatrics:

1. Older adults have greater numbers of HAV antibodies, resulting in fewer cases.

2. Symptoms are usually vague. Fatigue, pruritus, and the classic symptoms of jaundice, hepatomegaly, and liver tenderness are commonly absent in older adults.

3. Diagnostic test results in older adults include elevated bilirubin, lower or normal transaminase and ALP, and normal ultrasonography.

4. Older adults with acute HAV are likely to have more profound hepatocellular dysfunction with frequent jaundice and coagulopathy, as well as a higher incidence of complications such as prolonged cholestasis, pancreatitis, and ascites.

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HEPATITIS B

DEFINITION

A. The hepatitis B virus (HBV) causes inflammation of the liver. Acute HBV infection cannot be distinguished from other forms of acute viral hepatitis on the basis of clinical signs and symptoms or nonspecific laboratory findings. Acutely infected clients may be asymptomatic or symptomatic. The likelihood of developing symptoms of acute hepatitis is age-dependent.

B. The presence of hepatitis B surface antigen (HBsAg) establishes the diagnosis of hepatitis B. Chronic HBV (CHB) versus acute infection is defined as the presence of HBsAg in serum for at least 6 months. Diagnostic criteria and clinical terms are available at the 2018 American Association for the Study of Liver Disease (AASLD) website located at www.aasld.org/publications/practice-guidelines.

C. The levels of serum alanine transaminase (ALT) and HBV DNA as well as liver fibrosis are important predictors of long-term outcome for decisions for antiviral treatment as well as treatment response.

D. Staging of liver disease severity using a liver biopsy or noninvasive tests such as elastography is important in guiding surveillance and assisting with treatment decisions.

E. Ten genotypes of HBV have been identified (A through J):

1. HBV genotypes A to H have been found in the United States, with genotypes A, B, and C being most prevalent.

F. Guidelines for monitoring clients who are CHB who are not currently on treatment as well as the management of CHB special populations are available on the AASLD website.

G. The Advisory Committee on Immunization Practices (ACIP) recommends that the following people receive hepatitis B vaccination:

1. All infants.

2. Unvaccinated children younger than 19 years.

3. People at risk for infection by sexual exposure:

a. Sex partners of HbsAg-positive persons.

b. Sexually active people who are not in a long-term, mutually monogamous relationship (e.g., persons with more than one sex partner during the previous 6 months).

c. People seeking evaluation or treatment for a sexually transmitted infection (STI).

d. Men who have sex with men (MSM).

4. People at risk for infection by percutaneous or mucosal exposure to blood:

a. Current or recent injection drug users.

b. Household contacts of people who are HbsAg-positive.

c. Residents and staff of facilities for developmentally disabled people.

d. Healthcare and public safety personnel with reasonably anticipated risk for exposure to blood or blood-contaminated body fluids.

e. Individuals on hemodialysis, including peritoneal dialysis and home dialysis.

f. People with diabetes age 19 to 59 years; persons with diabetes age >60 years at the discretion of the treating clinician.

5. International travelers to countries with high or intermediate levels of endemic HBV infection (HbsAg prevalence of ≥2%).

6. People with hepatitis C infection.

7. People with chronic liver disease (CLD; cirrhosis, fatty liver disease, alcoholic liver disease [ALD], autoimmune hepatitis, and an ALT or aspartate transaminase [AST] level greater than twice the upper limit of normal).

8. HIV-infected persons.

9. People who are incarcerated.

10. All other people seeking protection from HBV infection.

INCIDENCE

A. The majority of children and adults with CHB in the United States are immigrants, have immigrant parents, or became exposed through other close household contacts.

B. In 2016, the Centers for Disease Control and Prevention (CDC) noted the overall incidence of HBV was one case per 100,000. In 2019, 5.9 cases per 100,000 population; 47% occurred among persons aged 30 to 49 years.

C. HBV is more severe among adults older than 60 years.

D. Approximately 95% of adults recover completely from HBV infection and do not become chronically infected.

E. Approximately 25% of those who become chronically infected during childhood and 15% of those who become chronically infected after childhood die prematurely from cirrhosis or liver cancer, and the majority remain asymptomatic until onset of cirrhosis or end-stage liver disease.

PATHOGENESIS

A. HBV is a hepadnavirus. HBV-related liver injury is largely caused by immune-mediated mechanisms mediated via cytotoxic T-lymphocyte lysis of infected hepatocytes. The virus is transmitted through perinatal, percutaneous, sexual exposure, and close person–person contact body fluids, such as wound exudates, semen, cervical secretions, and saliva that are HBsAg-positive. It is not transmitted by the fecal–oral route or by water. Blood and serum contain the highest concentration of virus; saliva contains the lowest.

B. The incubation period is 45 to 160 days (2–5 months), with an average of 120 days. An infected person can infect others 4 to 6 weeks before symptoms appear and for an unpredictable time thereafter.

C. The production of antibodies against HBsAg confers protective immunity and can be detected in clients who have recovered from HBV or in those clients who have been vaccinated. The immunoglobulin M (IgM) subtype indicates an acute infection or reactivation. Immunoglobulin G (IgG) subtype indicates chronic infection.

PREDISPOSING FACTORS

A. Higher prevalence populations born in regions of high or intermediate HBV prevalence of ≥2% including (the percentage is the area of the country) Africa, Asia, South Pacific, Middle East, Eastern and Western Europe, North America/Alaskan Eskimos, Mexico and Central America, Australia, and the Caribbean.

B. United States born persons not vaccinated as an infant whose parents were born in regions with high HBV endemicity (≥8%).

C. MSM.

D. Drug use, sharing needles.

E. Occupational exposure to blood or blood-contaminated body fluids.

F. Household, needle-sharing, and sexual contacts of HbsAg-positive persons.

G. Perinatal exposure, by vertical infection.

H. Receiving blood transfusions or blood products for hemophilia.

I. End-stage renal disease, including predialysis, hemodialysis, peritoneal dialysis.

J. Staffing or residing in institutions.

K. International travel.

L. Incarceration in long-term correction facilities.

M. Percutaneous contact with inanimate objects contaminated with HBV; virus can survive 1 week or longer.

COMMON COMPLAINTS

A. The following symptoms occur in the acute phase of HBV:

1. Anicteric hepatitis: asymptomatic (majority of clients).
2. Icteric hepatitis: associated with a prodromal period:
   a. Anorexia.
   b. Nausea and vomiting.
   c. Low-grade fever.
   d. Myalgia.
   e. Fatigue.
   f. Aversion to food and cigarettes.
   g. Intermittent, mild to moderate right upper quadrant (RUQ) and epigastric pain.
   h. Tea-colored urine.
   i. Clay-colored bowel movements (BMs).
3. Hyperacute, acute, and subacute hepatitis symptoms:
   a. Hepatic encephalopathy (HE).
   b. Somnolence.
   c. Disturbances in sleep pattern.
   d. Mental confusion.
   e. Coma.

B. The following symptoms occur in the chronic phase of HBV:

1. Asymptomatic: may be healthy carriers without any evidence of active disease.
2. During the replicative state, common symptoms are:
   a. Fatigue.
   b. Anorexia.
   c. Nausea.
   d. Mild upper quadrant pain or discomfort.
   e. Hepatic decompensation.

OTHER SIGNS AND SYMPTOMS

A. Icteric phase (10 days after the appearance of constitutional symptoms and lasts for 1–3 months):

1. Jaundice of sclera and skin.
2. Tea-colored urine.
3. Clay-colored stools, often precede jaundice.
4. RUQ tenderness.
5. Hepatomegaly.

SUBJECTIVE DATA

A. Review onset, duration, course, and severity of symptoms. Ask the client for specifics about urine and stool color.

B. Ask the client about other family members and sexual contacts with similar symptoms.

C. Review family history of hepatocellular carcinoma (HCC).

D. Discuss the client's history of blood transfusions, tattoos, incarceration, intravenous (IV) drug use, and alcohol abuse.

E. Ask about high-risk sexual practices.

F. Review the client's occupational exposure.

G. Inquire about recent international travel.

H. Establish the client's usual weight; note amount of any weight lost and over what length of time.

I. Has the client ever had a liver biopsy?

J. Review for a history of variceal bleeding.

K. Ask if the client has ever been treated for any type of hepatitis?

1. How long ago was the client treated?
2. Did the client complete therapy? If not, why?
3. What was the client's response to therapy (e.g., nonresponder, relapser)?

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure (BP), and height and weight to calculate body mass index (BMI).
B. Inspect:
  1. Observe general appearance, muscle wasting, ascites, and peripheral edema.
  2. Inspect the skin for tattoos and piercings, jaundice, palmar erythema, rash, spider nevi, spider angioma, and dehydration.
  3. Inspect the eyes for yellow sclera.
  4. Inspect the mucous membranes and nail beds for clubbing and cyanosis.
  5. Evaluate for the presence of gynecomastia and small testes.
C. Auscultate:
  1. Auscultate lung fields and the heart.
  2. Auscultate all quadrants of the abdomen. Evaluate for an abdominal bruit.
D. Percuss:
  1. Abdomen.
E. Palpate:
  1. Palpate all quadrants of the abdomen for masses, liver enlargement or tenderness, characteristics of cirrhosis, and hepatosplenomegaly.
  2. Palpate the lymph nodes for lymphadenopathy.
  3. Palpate for testicular atrophy.

DIAGNOSTIC TESTS

A. Diagnostic test for HBV antigens and antibodies (Table 14.16).
B. Complete blood count (CBC) with platelets.
C. Complete liver panel:
  1. AST/ALT.
  2. Total bilirubin.
  3. Prothrombin time (PT)/international normalized ratio (INR).
  4. Albumin.
D. Alkaline phosphatase (ALP).
E. Other viral infection markers.
F. Alpha-fetoprotein (AFP; rule out other causes of liver disease).
G. Gamma-glutamyl transpeptidase (GGT).
H. Serum iron levels (screen for hemochromatosis).
I. Thyroid profile.
J. Blood glucose/A1C.
K. After HBV confirmation:
  1. HBV genotype.
  2. HBV DNA viral load.
L. Serum fibrosis panel:
  1. Aspartate platelet ratio index (APRI): AST-to-platelet ratio index used for estimating hepatic fibrosis. Online calculator can be found at: www.hepatitisc.uw.edu/page/clinical-calculators/apri.
  2. Fibrosis-4 (FIB-4) is an index for estimating hepatic fibrosis based on a calculation derived from AST, ALT, platelet concentrations, and age. Online calculator can be found at www.hepatitisc.uw.edu/page/clinical-calculators/fib-4.
  3. FibroTest (FibroSure): commercial biomarker test that uses the results of six blood markers to estimate hepatic fibrosis.
M. Imaging:
  1. Ultrasonography: FibroScan: Transient shear-wave elastography measures liver stiffness as a surrogate for fibrosis.
  2. CT or MRI to help exclude biliary obstruction.
N. Liver biopsy to assess the severity of disease.
O. Pregnancy testing before antiviral therapy.
P. Before oral antiviral therapy is introduced, all clients should be screened for HIV.
Q. Hepatitis C virus (HCV) and HIV testing to rule out coinfection.

DIFFERENTIAL DIAGNOSES

A. Hepatitis B.
B. Exclusion of other types of hepatitis (A, C, D, E, viral, or autoimmune hepatitis).
C. Infectious mononucleosis.
D. Hepatotoxic drug ingestion, for example, chloramphenicol, acetaminophen, or methyldopa.
E. Metastatic cancer to the liver.
F. Alcoholic cirrhosis.
G. Hemochromatosis.
H. Wilson disease.

PLAN

A. General interventions:
  1. Before any form of HBV therapy is started, and optimally at the first presentation, the client needs to be provided with information about the natural history of chronic ▶

TABLE 14.16 DIAGNOSTIC TESTS FOR HEPATITIS B ANTIGENS AND ANTIBODIES

Factors to Be Tested	HBV Antigen or Antibody	Indication
HBsAg	HBsAg	Detects acutely or chronically infected; antigen used in hepatitis B vaccine
Anti-HBs	Antibody to HBsAg	Identifies resolved HBV infections; determines immunity after immunization
HBeAg	HBeAg	Identifies at risk of transmitting HBV
Anti-HBe	Antibody to HBeAg	Identifies lower risk of transmitting HBV
Anti-HBc	Antibody to HBcAg; IgM (HBcAg)	Identifies acute, resolved, or chronic HBV infection; anti-HBc not present after immunization
IgM anti HBc	IgM antibody to HBcAg	Identifies acute or recent HBV infections (includes HBsAg-negative during the window phase of infection)

HB, hepatitis B; HBcAg; hepatitis B core antigen; HBeAg, hepatitis B e-antigen; HBsAg, hepatitis B surface antigen; HBV, hepatitis B virus; IgM, immunoglobulin M.

hepatitis B infection and the fact that most infections remain entirely without symptoms even in those with severe disease, so there is a need for regular lifelong monitoring.

B. Client teaching: *See Client Teaching Guide for this chapter, "Jaundice and Hepatitis."*

1. Instruct the client to avoid sexual activity until they are free of HBsAg.

2. Recommend abstinence or minimal alcohol consumption. More than seven drinks of alcohol per week for females and more than 14 drinks per week for males are associated with an increase of cirrhosis and HCC.

3. Any blood spills should be cleaned using 1:10 dilution of bleach to water for disinfecting the area. Gloves should be worn when cleaning up any blood spills.

4. There are no dietary restrictions with acute and chronic hepatitis (without cirrhosis).

5. Diets for decompensated cirrhosis, portal hypertension, or encephalopathy are prescribed:

a. Low-sodium diet (1.5 g/d).

b. High-protein diet (white-meat protein, e.g., pork, turkey, and fish).

c. Fluid restriction of 1.5 L/d in the presence of hyponatremia.

C. Pharmaceutical therapy:

1. The goal of treatment is to prevent progression to cirrhosis, hepatic failure, and hepatocellular cancer:

a. Primary prevention includes vaccination. Vaccination is up to 95% effective. Immunizations schedules are located on the CDC website. https://www.cdc.gov/hepatitis/hbv/hbvfaq.htm#D4.

i. There are three single-antigen hepatitis B vaccines (ENGERIX-B, RECOMBIVAX BH, and HEPLISAV-B).

ii. There are three combination vaccines:

1) PEDIARIX: combined hepatitis B; diphtheria, tetanus, acellular pertussis (DTaP); and inactivated poliovirus (IPV) vaccine. Cannot be administered before age 6 weeks or after age 7 years.

2) TWINRIX: combined hepatitis A and hepatitis B vaccine. Recommended for persons age ≥18 years who are at increased risk for both hepatitis A virus (HAV) and HBV infections.

3) **Vaxelis**: combination diphtheria, tetanus, acellular pertussis, IPV, *Haemophilus influenzae* type b, and hepatitis B.

iii. There is one three-antigen hepatitis B vaccine (PreHevbrio).

b. If serum HBV DNA exceeds 1,000 IU/mL, antiviral therapy is recommended. See Table 14.17 for current HBV therapy:

i. All clients need to be screened for HIV prior to antiviral therapy.

ii. Females need to have a pregnancy test prior to therapy.

iii. All antivirals require close monitoring due to side effect profiles.

iv. Dose adjustment is required in the presence of renal impairment, dialysis, and is used with caution in clients with a history of pancreatitis.

c. Orthotopic liver transplantation (OLT) is the treatment of choice for clients with fulminant hepatic failure who do not recover and for clients with end-stage liver disease.

d. Immunize against hepatitis A for individuals with CHB if not already immune.

FOLLOW-UP

A. Laboratory monitoring:

1. Screening should be performed using both HBsAg and anti-HBs.

2. Screening for anti-HBc (hepatitis B core antigen) to determine prior exposure is not routinely recommended:

a. Test in clients who have HIV infection.

b. Test persons about to initiate HCV or anticancer and other immunosuppressive therapy or renal analysis and in donated blood.

3. Monitor liver function (ALT) every 3 to 6 months for active disease.

4. Monitor HBeAg every 3 to 6 months dependent on ALT levels.

5. Monitor CBC and creatinine every month (1–2 days prior to treatment).

6. Monitor HBV DNA viral load every 3 to 6 months when the client is on treatment in the reactivation phase.

B. Laboratory, physical examination, and psychosocial evaluation are required for antiviral therapy:

1. Monitor for mental dysfunction related to interferon (IFN), including depression, psychosis, aggressive behavior, hallucinations, violent behavior, suicidal ideation, suicide attempt, and homicidal ideation (rare), even without history of psychiatric illness.

C. Risk of exposure to HBsAg ceases when antigen disappears from the bloodstream, usually within 6 to 8 weeks of infection.

D. HBV reactivation is the abrupt reappearance or rise in HBV DNA in a client with previously inactive or resolved hepatitis B. It is often accompanied by a flare in disease activity with elevation of liver enzymes with or without symptoms, and can be severe, resulting in death.

1. Clients at highest risk for HBV reactivation:

a. Clients undergoing cancer chemotherapy.

b. Clients taking immunosuppressive therapy including the following:

i. Black box warning for rituximab and other drugs that target B lymphocytes.

ii. High-dose steroids.

iii. Anti-tumor necrosis factor (TNF) agents.

c. Clients with HIV who have discontinued HBV active antiviral drugs.

d. Clients undergoing solid organ or bone marrow transplantation.

e. Clients coinfected with hepatitis C.

CONSULTATION/REFERRAL

A. Clients with persistently elevated serum transaminase concentrations (exceeding twice the upper limits of normal), as well as those with elevated serum alpha-fetoprotein concentrations or abnormal ultrasounds, should be referred to a gastroenterologist for further management.

B. Clients who are coinfected with HBV or HIV or have end-stage renal disease should be referred to and managed by a hepatologist, infectious disease provider, or gastroenterologist for treatment.

C. Clients with decompensated cirrhosis should be referred for consideration of liver transplantation.

D. HBV-infected pregnant clients with cirrhosis should be managed by a high-risk OB practice and should be treated with tenofovir disoproxil fumarate (TDF) to prevent decompensation.

TABLE 14.17 APPROVED ANTIVIRAL THERAPIES FOR ADULTS WITH CHRONIC HEPATITIS B: AASLD 2018 HEPATITIS B GUIDANCE

Drug	Dose in Adults	Monitoring on Treatment	Pregnancy Category
Preferred			
Peg-IFN-alpha-2a	180 mcg weekly	Test for HIV before treatment initiation CBC every 1–3 months TSH every 3 months Lactic acid levels if clinical concern (decompensated cirrhosis only) Clinical monitoring for autoimmune ischemia, neuropsychiatric, and infectious complications	C Avoid in pregnancy and when breast feeding
Entecavir	0.5 mg daily Dose 1 mg daily if client is lamivudine experienced of if they have decompensated cirrhosis	Test for HIV before treatment initiation CBC every 1–3 months TSH every 3 months Lactic acid levels if clinical concern (decompensated cirrhosis only) Clinical monitoring for autoimmune ischemia, neuropsychiatric, and infectious complication	C Avoid in pregnancy and when breast feeding
Tenofovir TDF	300 mg daily	Test for HIV before treatment initiation Creatinine clearance at baseline If at risk for renal impairment, creatinine clearance, serum phosphate, urine glucose, and urine protein at least annually Consider bone density study at baseline and during treatment in clients with history of fracture or risks for osteopenia Lactic acid levels if there is clinical concern	B Safe in pregnancy up to 28 weeks of pregnancy
TAF Tenofovir alafenamide	25 mg daily	Test for HIV before treatment initiation Assess serum creatinine, serum phosphorus, creatinine clearance, urine glucose, and urine protein before initiating and during therapy in all clients as clinically appropriate Lactic acid levels if there is clinical concern	Insufficient human data for assignment of drug-associated risk of birth defects and miscarriage
Nonpreferred			
Lamivudine	100 mg daily	Test for HIV before treatment initiation Amylase if symptoms of pancreatitis are present Lactic acid levels if there is clinical concern	C Not recommended during the first 3 months of pregnancy; should be used only during pregnancy if benefit outweighs the risk to fetus
Adefovir	10 mg daily	Test for HIV before treatment initiation Creatinine clearance at baseline If at risk for renal impairment, creatinine clearance, serum phosphate, urine glucose, and urine protein at least annually Consider bone density study at baseline and during treatment in clients with history of fracture or risks for osteopenia Lactic acid levels if there is clinical concern	C During pregnancy only if benefit outweighs the risk to the fetus*; avoid in breastfeeding*
Telbivudine	600 mg daily	Test for HIV before treatment initiation Creatine kinase if symptoms are present Clinical evaluation if CK and myopathy are present Lactic acid levels if there is clinical concern	B

Note: In 2015, the U.S. Food and Drug Administration replaced the pregnancy risk designation by letters A, B, C, D, and X with more specific language on pregnancy and lactation. This new labeling is being phased in gradually, and to date only TAF includes these additional data.

AASLD, American Association for the Study of Liver Disease; CBC, complete blood count; CK, creatine kinase; IFN, interferon; TAF, tenofovir alafenamide; TDF, tenofovir disoproxil fumarate; TSH, thyroid-stimulating hormone.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Clients should receive HBsAg testing during each pregnancy:

a. Those who are HBsAg-positive should have HBV DNA testing.

b. Prenatal HBsAg testing of all pregnant females is recommended to identify newborns who require immediate postexposure prophylaxis.

c. Extra care is necessary to evaluate the mother to ensure that the infant receives hepatitis B immune globulin (HBIG) and HBV vaccine within 12 hours of birth (Table 14.18).

2. Pregnancy and lactation are not a contraindication to vaccination. No adverse effect on developing fetus has been observed when pregnant females are vaccinated against HBV.

3. Antiviral therapy in the third trimester is recommended for pregnant females with serum HBV DNA > 200,000 IU/mL:

a. TDF is the preferred therapy for prevention of mother-to-child transmission.

b. Antiviral therapy given for prevention of mother-to-child transmission is recommended to be discontinued at the time of delivery or up to 4 weeks postpartum:

i. Pregnant clients who stop antiviral therapy at or early after delivery should be monitored for up to 6 months after delivery for hepatitis flares and seroconversion.

ii. Long-term follow-up should be continued to assess the need for future antiviral therapy.

c. Antiviral drug labels do not recommend breastfeeding while on therapy; however clinical studies support the safety of these drugs during breastfeeding.

B. Adults:

1. Most HBV infections are acquired in adolescence or adulthood, largely as a result of IV drug use, sexual contact, or occupational or household exposure. HBV infection is associated with other sexually transmitted diseases, including syphilis.

2. Clients who have received a blood transfusion should refrain from blood donation for 6 months, the incubation period for HBV. Blood should never be donated if the client is a hepatitis B carrier or was infected with hepatitis C.

C. Geriatrics:

1. Vaccination against HBV is recommended in older adults at risk for infection, including all nursing home residents.

2. HBV is more severe among adults older than 60 years:

a. There is substantial decrease in liver mass, in the number of hepatocytes, and in the portal blood flow, which results in alteration of metabolism. There are significant reductions in up to 30% to 40% in parenchymal volume, liver blood flow, and perfusion

b. Older adults have a higher rate of mortality than younger clients, reflecting in part a higher prevalence of comorbid conditions such as diminished immune response, metabolic derangement, nutritional deficiencies, and greater cumulative exposure to environmental hepatotoxins.

c. Those who do develop the disease have a greater tendency to deteriorate into chronic liver failure or HCC:

i. The risk of cirrhosis in highly linked to the HBV DNA level.

3. Liver transplant programs may have relative contraindication inclusion criteria for liver transplant for ages more than 65 to 70 years.

RESOURCES

Centers for Disease Control and Prevention: www.cdc.gov/hepatitis.org.

Hepatitis B Foundation: www. https://www.hepb.org/

National Institute of Diabetes and Digestive and Kidney Diseases: https://www2.niddk.nih.gov/

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HEPATITIS C

DEFINITION

A. Hepatitis C is an inflammation of the liver caused by the hepatitis C virus (HCV). HCV has signs and symptoms ▶

TABLE 14.18 POSTEXPOSURE HEPATITIS B PROPHYLAXIS AFTER PERCUTANEOUS EXPOSURE

Exposed Person	HBsAg- Negative	Unknown or Not Tested	Treatment When Source Is HBsAg-Positive
Unimmunized	Initiate hepatitis B vaccine series	Initiate hepatitis B vaccine series	Administer one dose of HBIG 0.06 mL/kg IM; then initiate hepatitis B vaccine series
Previously immunized: known responder	No treatment	No treatment	No treatment
Previously immunized: known nonresponder	No treatment	If known high-risk source, treat as if source was HBsAg-positive	Administer one dose of HBIG 0.06 mL/kg IM; then initiate reimmunization or HBIG (two doses) hepatitis B vaccine series
Previously immunized: response unknown	No treatment	Test exposed person for anti-HBs; if adequate, no treatment; if inadequate, administer vaccine booster	Test exposed person for anti-HBs and administer vaccine booster dose; two additional doses should be administered to complete three-dose reimmunization series

HB, hepatitis B; HBIG, hepatitis B immune globulin; HBsAg, hepatitis B surface antigen; IM, intramuscular.

often undistinguishable from those of hepatitis A or B (HAV or HBV). The disease tends to be asymptomatic to mild and has an insidious onset. Acute fulminate infection is rare. The major feature of HCV is its propensity to become chronic.

B. Multiple (6) HCV genotypes and subtypes exist. It is possible for a person to be infected with more than one genotype. The genotype is a major factor in the effectiveness of the client's response to therapy.

C. The development of chronic hepatitis and its complications increase with several factors, including older age at acquisition, HBV coinfection, HIV coinfection, excessive alcohol consumption, and male sex.

D. HCV is the leading cause of nonalcoholic hepatic failure and cirrhosis, and the cause of 90% of posttransfusion hepatitis. Primary hepatocellular carcinoma (HCC) also occurs in these clients.

E. No vaccine for hepatitis C is available and therefore prevention of HCV depends on reducing the risk of exposure.

F. Council of State and Territorial Epidemiologists (CSTE) Position Statements for *Acute Hepatitis C*:

1. Clinical criteria for acute HCV:

a. An illness with discrete onset of any sign or symptom consistent with acute viral hepatitis (e.g., fever, headache, malaise, anorexia, nausea, vomiting, diarrhea, and abdominal pain).

AND

i. Jaundice.

OR

ii. A peak elevated serum alanine transaminase (ALT) level greater than 200 IU/L during the period of acute illness.

2. Laboratory criteria for acute HCV:

a. A positive test for anti-HCV.

b. HCV detection test:

i. Nucleic acid testing (NAT) for HCV RNA positive (including qualitative, quantitative, or genotype testing).

ii. A positive test indicating presence of HCV antigen(s).

3. Criteria to distinguish a new case from an existing case of acute HCV:

a. A new acute case is an incident acute hepatitis C case that meets the case criteria for acute hepatitis C and has not previously been reported.

b. A new probable acute case may be reclassified as a confirmed acute case if a positive NAT for HCV RNA or a positive HCV antigen(s) test is reported within the same year.

c. A confirmed acute case may be classified as a confirmed chronic case if a positive NAT for HCV RNA or a positive HCV antigen is reported 1 year or longer after acute case onset.

d. A confirmed acute case may not be reported as a probable chronic case (i.e., HCV antibody positive, but with an unknown HCV RNA NAT or antigen status).

4. Case classification for acute HCV:

a. Probable:

i. A case that meets clinical criteria and has a positive anti-HCV antibody test, but has no reports of a positive HCV NAT or positive HCV antigen tests.

AND

ii. Does not have test conversion within 12 months or has no report of test conversion.

b. Confirmed:

i. A case that meets clinical criteria and has a positive HCV detection test (HCV NAT or HCV antigen).

OR

ii. A documented negative HCV antibody, HCV antigen, or NAT laboratory test result followed within 12 months by a positive result of any of these tests (test conversion).

G. Council of State and Territorial Epidemiologists (CSTE) Position Statements for *Chronic Hepatitis C:*

1. Clinical criteria for chronic HCV:

a. No available evidence of clinical and relevant laboratory information indicative of acute infection (as noted earlier).

b. Most HCV-infected persons are asymptomatic; however, many have CLD, which can range from mild to severe.

2. Laboratory criteria for diagnosis of chronic HCV:

a. A positive test for anti-HCV.

b. HCV detection test for acute HCV:

i. NAT for HCV RNA positive (including qualitative, quantitative, or genotype testing).

ii. A positive test indicating presence of HCV antigen(s).

3. Criteria to distinguish a new case from an existing case of chronic HCV:

a. A new chronic case is an incident chronic hepatitis C case that meets the case criteria for chronic hepatitis C and has not previously been reported.

b. A confirmed acute case may not be reported as a probable chronic case (i.e., HCV antibody positive, but with an unknown HCV RNA NAT or antigen status).

4. The Centers for Disease Control and Prevention (CDC) 2020 case classification for chronic HCV:

a. Probable:

i. A case that does not meet clinical criteria or has no report of clinical criteria.

AND

ii. Has presumptive laboratory evidence.

AND

iii. Has no documentation of anti-HCV or RNA test conversion within 12 months AND

iv. Does not have HCV RNA detection reported.

b. Confirmed:

i. A case that does not meet clinical criteria or has no report of clinical criteria.

AND

ii. Has confirmatory laboratory evidence.

AND

iii. Has no documentation of anti-HCV or HCV RNA test conversion within 12 months.

INCIDENCE

A. The World Health Organization (WHO) estimates that more than 117 million individuals worldwide with hepatitis C and 58 million are chronically infected with HCV. Annually, 1.5 million individuals are newly infected with hepatitis C.

B. Of every 100 people infected with HCV, approximately:

1. 75 to 85 will go on to develop chronic infection.

2. 10 to 20 will go on to develop cirrhosis over a period of 20 to 30 years.

3. Rates of progression to cirrhosis are increased in the presence of a variety of factors: males more than females, ▶

age greater than 50 years, alcohol, nonalcoholic fatty liver disease (NAFLD), HBV or HIV coinfection, immunosuppressive therapy.

C. Among clients with cirrhosis, there is:

1. 1% to 5% annual risk of HCC.

2. 36% annual risk of hepatitic decompensation, for which the risk of death in the following year is 15% to 20%.

D. Approximately 15% to 25% of people clear the HCV virus without treatment and do not develop chronic infection. **Although they are no longer infected, they will still test positive for antibodies to HCV (anti-HCV):**

1. Predictors of spontaneous clearance include jaundice, elevated ALT level, hepatitis B surface antigen (HBsAg) positivity, female sex, younger age, HCV genotype 1, and host genetic polymorphisms, most notably those near the IL28B gene.

E. HCV infection becomes chronic in approximately 75% to 85% of cases. A person infected with HCV mounts an immune response to the virus, but replication of the virus during infection can result in changes that evade the immune response. This may explain how the virus establishes and maintains chronic infection.

F. Seroprevalence rates among individuals vary according to their associated risk factors. The highest rates occur in persons with large or repeated direct percutaneous exposure to blood or blood products, such as intravenous (IV) drug users and clients with hemophilia who have received multiple blood transfusions prior to 1990.

G. Seroprevalence among pregnant clients in the United States in 2018 was estimated at 1% to 2.5%. Maternal–fetal (vertical) transmission only ranges from 3% to 10%. Risk for transmission is highest among individuals with a high viral load at delivery. Maternal coinfection with HIV has been associated with increased risk of perinatal transmission of HCV RNA.

H. Serum anti-HCV antibody and HCV RNA have been detected in colostrum. However, although only a limited number of clients have been studied, the rate of transmission among breastfed infants is the same as among bottle-fed infants.

PATHOGENESIS

A. HCV is a small, single-stranded RNA virus with a lipid envelope and is a member of the Flavivirus family. It is a bloodborne virus, and the most common modes of infection are through exposure to small quantities of blood injection drug use, unsafe injection practices, and transfusion of unscreened blood and blood products.

B. The WHO also notes that one of the most common transmissions is in the reuse or inadequate sterilization of medical equipment, especially syringes and needles in healthcare settings.

C. Sexual transmission of HCV is uncommon except with high-risk behavior.

D. The incubation period averages 6 to 7 weeks, with a range of 2 weeks to 6 months. The time from exposure to the development of viremia generally is 1 to 2 weeks.

PREDISPOSING FACTORS

A. All people with HCV-RNA in their blood are considered to be infectious. The following groups are at high risk for HCV infection and should be tested:

1. IV drug users who have shared needles.

2. Intranasal cocaine users, presumably resulting from epistaxis and shared equipment.

3. Hemophiliacs, hemodialysis clients, and those who received blood transfusions before 1992.

4. Recipients of solid organ transplants prior to 1992.

5. Healthcare workers with percutaneous exposures.

6. Individuals with multiple sexual partners.

7. Transmission among contacts living with infected persons may occur with percutaneous or mucosal exposure to blood.

8. Infants of infected mothers, by vertical transmission.

9. HCV is more common in males than females.

10. Tattooing, body piercing, and acupuncture with unsterile equipment.

11. HIV.

12. Incarceration.

COMMON COMPLAINTS

A. Chronic HCV: asymptomatic unless there is progressive inflammation and complications from cirrhosis.

B. Malaise.

C. Anorexia.

D. Nausea.

E. Myalgia.

F. Fever.

G. Abdominal pain.

OTHER SIGNS AND SYMPTOMS

A. Jaundice (occurs in fewer than 20% of clients).

B. Hepatomegaly is present in one-third of clients with an acute infection.

C. Ascites.

D. Spider nevi.

E. Dark urine.

F. Clay-colored stools.

SUBJECTIVE DATA

A. Review duration, onset, and severity of symptoms.

B. Ask the client about other family members and sexual contacts with similar symptoms.

C. Review family history of hepatocellular carcinoma (HCC).

D. Review the client's history of blood transfusions, tattoos, incarceration, IV drug use, and alcohol abuse.

E. Ask the client about occupational exposure to blood and bodily secretions.

F. Ask about high-risk sexual practices.

G. Inquire about recent international travel.

H. Establish the client's usual weight; note amount of any weight lost and over what length of time.

I. Review for a history of variceal bleeding.

J. Ask if the client has been treated for any type of hepatitis:

1. How long ago was the client treated?

2. Did the client complete therapy? If not, why?

3. What was the client's response to therapy (e.g., nonresponder, relapser)?

K. Has the client had any testing for cirrhosis/liver biopsy? What testing and when?

L. Review HIV status.

PHYSICAL EXAMINATION

A. Check temperature (acute infection), pulse, respirations, blood pressure (BP), and height and weight to calculate body mass index (BMI).

B. Inspect:

1. Observe general appearance, muscle wasting, edema, and demeanor. **Administer a depression self-assessment tool at each visit when on HCV therapy.** ▶

2. Inspect the skin for jaundice, rash, dehydration, palmar erythema, excoriations, spider nevi, and tattoos/piercings.
3. Inspect the eyes for yellow sclera.
4. Inspect mucous membranes and nail beds for clubbing and cyanosis.
5. Inspect for gynecomastia and small testes.

C. Auscultate:
1. Auscultate lung fields and heart.
2. Auscultate all quadrants of the abdomen and evaluate for abdominal bruit.

D. Percuss:
1. Abdomen.

E. Palpate:
1. Palpate all quadrants of the abdomen for masses, liver enlargement or tenderness, characteristics of cirrhosis, and hepatosplenomegaly, which occurs in about 10% of cases.
2. Palpate the lymph nodes for lymphadenopathy and enlarged parotid.

DIAGNOSTIC TESTS

A. Complete blood count (CBC) with platelets.
B. Complete liver panel:
1. Aspartate aminotransferase (AST)/ALT.
2. Total bilirubin.
3. Prothrombin time (PT)/international normalized ration (INR).
4. Albumin.
C. Alkaline phosphatase (ALP).
D. Other viral infection markers, especially hepatitis B virus (HBV) and HIV.
E. Alpha-fetoprotein (AFP; rule out other causes of liver disease).
F. Gamma-glutamyl transferase (GGT).
G. Thyroid profile.
H. A1C.
I. After HBV confirmation:
1. HBV genotype.
2. HBV DNA viral load.
J. Serum fibrosis panel:
1. AST-to-platelet ratio index (APRI): used for estimating hepatic fibrosis. Online calculator can be found at: www.hepatitisc.uw.edu/page/clinical-calculators/apri.
2. Fibrosis-4 (FIB-4) is an index for estimating hepatic fibrosis based on a calculation derived from AST, ALT, platelet concentrations, and age. Online calculator can be found at www.hepatitisc.uw.edu/page/clinical-calculators/fib-4.
3. FibroTest (FibroSure): commercial biomarker test that uses the results of six blood markers to estimate serum hepatic fibrosis markers.
K. Imaging:
1. Ultrasonography: FibroScan: Transient shear-wave elastography measures liver stiffness as a surrogate for fibrosis.
2. CT or MRI to help exclude biliary obstruction.
L. Liver biopsy to assess the severity of disease.
M. Pregnancy testing before and during antiviral therapy.
N. The in-depth initial and follow-up testing for HCV is located at the American Association for the Study of Liver Disease (AASLD) and Infectious Diseases Society of America (IDSA) website at www.hcvguidelines.org/evaluate. These guidelines are dynamic; therefore, the website should be accessed frequently.

DIFFERENTIAL DIAGNOSES

A. Hepatitis C.
B. Hepatitis A.
C. Hepatitis B.
D. Alcoholic liver disease (ALD).
E. Drug toxicities.
F. Opportunistic infections associated with HIV infection.
G. Infectious hepatitis (viral, bacterial, fungal, and parasitic).
H. Autoimmune diseases.

PLAN

A. General interventions:
1. Discuss the possibility of transmission to others, and tell the client to refrain from donating blood, organs, tissues, or semen and from sharing toothbrushes and razors.
2. All clients with chronic HCV should be immunized against hepatitis A and hepatitis B.
3. Any blood spills should be cleaned using a 1:10 dilution of bleach to water for disinfecting the area. Gloves should be worn when cleaning up any blood spills.
4. The WHO recommends that alcohol screening and behavioral interventions for people with moderate to high alcohol intake should be instituted.
5. The CDC recommends that anyone born between the years of 1945 and 1965 be tested for HCV one time as serum screening.

B. Client teaching: *See Client Teaching Guide for this chapter, "Jaundice and Hepatitis."*
1. Counsel the client to avoid hepatotoxic medications and alcohol:
a. Recommend abstinence or minimal alcohol consumption. More than seven drinks of alcohol per week for females and more than 14 drinks per week for males are associated with an increase of cirrhosis and HCC. Alcohol-free beers with limitations still contain some alcohol per beer.

C. Pharmaceutical therapy:
1. Treatment is aimed at inhibiting HCV replication and eradicating infection. Pharmaceutical regimens are quickly evolving as new medications come to the market. The AASLD and IDSA guidelines break down therapy and management in detail by genotype/subtypes, presence of cirrhosis, whether a client is treatment-naïve or treatment-experienced, and medications and length of therapy.
2. The guidelines are located at www.hcvguidelines.org/contents.
3. The guidelines also include the management of unique populations including clients with HIV/HCV coinfection, clients with decompensated cirrhosis, clients who develop recurrent HCV infection postliver transplant, clients with renal impairment, kidney transplant clients, management of acute HCV infection, HCV in pregnancy and children, management of chronic HCV infection, HCV in people who inject drugs, men who have sex with men (MSM), and testing and treatment in correctional facilities.

FOLLOW-UP

A. Refer to AASLD/IDSA current guidelines on testing, managing, and treating hepatitis C at www.hcvguidelines.org/references/aasldidsa-2020.

CONSULTATION/REFERRAL

A. Referrals include gastroenterologist, hepatologist, infectious disease, psychiatrist, endocrinologist, neurologist, hematologist, dietitian, and social workers.

B. Clients who are coinfected with HBV or HIV or have end-stage renal disease should be referred to and managed by a hepatologist, infectious disease provider, or gastroenterologist for treatment.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Routine serologic testing of pregnant clients for HCV infection is not recommended. Clients with significant risk factors for HCV should be offered antibody screening.

2. According to current guidelines of the U.S. Public Health Service and the American Academy of Pediatrics, maternal HCV infection is not a contraindication to breastfeeding. HCV-positive mothers should consider abstaining from breastfeeding if their nipples are cracked or bleeding.

3. The method of delivery has not been shown to increase the risk of vertical transmission of HCV. Cesarean delivery is reserved for obstetric indications.

B. Adults:

1. Infected persons with steady partners do not need to change their sexual practices. However, they should be informed of the possible risk of transmission and of what precautions to use to prevent transmission.

2. Persons with multiple partners should be advised to reduce the number of partners and to use condoms to prevent transmission.

C. Geriatrics:

1. As the population ages, the cases of HCV will increase more in older adults than in other age groups.

2. There is substantial decrease in liver mass, in the number of hepatocytes, and in the portal blood flow, which results in alteration of metabolism. There are significant reductions up to 30% to 40% in parenchymal volume, liver blood flow, and perfusion.

3. Older adults have a higher rate of mortality than younger clients, reflecting in part a higher prevalence of comorbid conditions such as diminished immune response, metabolic derangement, nutritional deficiencies, and greater cumulative exposure to environmental hepatotoxins.

4. Older age is an independent factor associated with a lower likelihood of being considered for antiviral therapies:

a. Adults over 60 have a higher prevalence of comorbidities, particularly cardiovascular, renal, pulmonary, and hematologic diseases.

b. Older adults are less likely to accept antiviral treatment.

c. Discontinuation of therapy and reductions are more frequently required in this age group.

d. Older adults have a faster progression to fibrosis.

5. Liver transplant programs may have relative contraindication inclusion criteria for liver transplant for ages more than 65 to 70 years.

RESOURCES

Immunization Action Coalition: www.immunize.org.

Project Inform Information, Inspiration, and Advocacy for People with HIV/AIDS and Hepatitis C: https://www.thebodypro.com/author/project-inform

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Hepatitis C Online. (n.d.). Fibrosis-4 (FIB-4) calculator. https://www.hepatitisc.uw.edu/page/clinical-calculators/fib-4

World Health Organization Guidelines for the screening, care, and treatment of persons with hepatitis C infection. https://www.who.int/news-room/fact-sheets/detail/hepatitis-c

HERNIAS, ABDOMINAL

DEFINITION

A. A hernia is the protrusion of a peritoneum-lined sac through some defect in the abdominal wall. Abdominal wall hernias are the most common surgical procedure. Hernias are a leading cause of disability and work loss. Types include the following:

1. Umbilical hernia: occurs when the intestinal muscles fail to close around the umbilicus, allowing the omentum and/or intestines to protrude into the weaker area.

2. Incisional hernia: caused by a defect in the abdominal musculature that develops after a surgical incision.

3. Epigastric hernia: protrusion of fat or omentum through the linea alba between the umbilicus and the xiphoid. Epigastric hernias are generally less than 2 cm in diameter.

4. Diastasis recti: acquired hernia most often due to pregnancy and obesity. The right and left rectus muscles separate, but there is no facial defect.

5. Obturator hernia: follows the path of the obturator nerves and muscles.

INCIDENCE

A. Umbilical hernias are common and often asymptomatic and may be present in up to 25% of the population when examined by ultrasound imaging. Umbilical hernias are more common in African American infants, females, and older adults. This type of hernia has a higher risk of incarceration and strangulation and therefore a greater mortality because the large bowel is frequently entrapped.

B. Epigastric hernias are most common in males 20 to 50 years old.

C. Incisional hernias typically are noted in the early postoperative period; however, there is an increase in incisional hernias during pregnancy. These iatrogenic hernias occur in 2% to 10% of abdominal operations. In addition to hernias, separation of the recti abdominis muscles (diastasis recti) is often caused by pregnancy or obesity.

D. Obturator hernias occur more commonly in females. Females have a larger canal diameter, which is noted predominantly in thin older adult females.

PATHOGENESIS

A. Incisional hernias are due to failure of fascial tissues to heal and close.

B. Epigastric hernias are defects in the abdominal midline between the umbilicus and the xiphoid process. They are usually related to a congenital weakness, increased intra-abdominal pressure, surrounding muscle weakness, or chronic abdominal wall strain.

C. An umbilical hernia is caused by failure of the umbilical ring to obliterate after birth. In the infant, the umbilical ring often closes spontaneously within the first 1 to 2 years of life. Increased abdominal pressure or congenital defects cause ▶

abdominal hernias that allow abdominal contents to protrude through the opening defect. In adults with an umbilical hernia, obesity increases the danger of incarceration.

PREDISPOSING FACTORS

A. Congenital predisposition.
B. Male.
C. Obesity.
D. Multiparity.
E. Cirrhosis and ascites.
F. Trauma or straining.
G. African American ancestry.
H. Chronic cough; can precipitate or worsen herniation.
I. Previous abdominal surgery.
J. Straining, coughing, and sneezing.
K. Straining with chronic constipation.
L. Incisional hernia factors:
  1. Smoking.
  2. Connective tissue disorder.
  3. Infection.
  4. Malnutrition.
  5. Immunosuppressive medications.
M. Age: Obturator hernias occur predominantly in older adults.
N. Maternal smoking is associated with an increased prevalence of omphalocele and gastroschisis.

COMMON COMPLAINTS

A. Asymptomatic: may only be detected on routine physical examination:
  1. Swelling or fullness at the hernia site.
  2. Varying degrees of discomfort.
  3. The only sign of a hernia may be increased irritability.
  4. Bulge of abdomen or of a previous scar.
B. Symptoms aggravated by cough and straining.

OTHER SIGNS AND SYMPTOMS

A. Incisional: bulge through incision wall (may be intermittent).
B. Epigastric: small, usually painless subcutaneous mass.
C. Umbilical:
  1. Adult: vague, intermittent pain; palpable mass.
D. Reducible or irreducible: Signs and symptoms are related to the degree of pressure of their contents rather than to size. Most clients are asymptomatic or complain of only mild pain.
E. Strangulated: colicky abdominal pain, nausea, vomiting, abdominal distension, hyperperistalsis.

SUBJECTIVE DATA

A. Review onset, duration, and course of symptoms.
B. Ask the client about previous abdominal surgeries, wound infection, and pregnancies.
C. Review history of straining, trauma, or physical labor.
D. Determine if the client has signs and symptoms of strangulation of entrapped bowel: pain, nausea, vomiting, distension, and fever.
E. Determine if the client can reduce hernia.
F. Ask the client whether the hernia is enlarging and uncomfortable.
G. Review the client's bowel history, specifically constipation.
H. Review the client's history for chronic obstructive pulmonary disease (COPD)/chronic cough.
I. Review the client's history for symptoms of obstructive uropathy where flow of urine is blocked causing injury to one or both kidneys.
J. Review how the hernia affects the client's activities of daily living (ADLs).

PHYSICAL EXAMINATION

A. Examination is the same for all types of abdominal hernias. Perform exam while the client is standing and supine. History and physical examination are the best means of diagnosing hernias.
B. Check temperature (if indicated), pulse, respirations, and blood pressure (BP).
C. Inspect:
  1. Inspect contour and symmetry of the abdomen for bulges or masses. The bulge may be asymmetric.
  2. Inspect irreducible hernias for discoloration, edema, and ascites.
  3. Assess:
    a. Have the client perform Valsalva maneuver while standing.
    b. Have the client lie supine, lift head from exam table, and then bear down to tense abdomen.
D. Auscultate:
  1. Abdomen in all quadrants for bowel sounds.
E. Percuss:
  1. Liver.
  2. Spleen.
  3. Abdomen.
F. Palpate:
  1. Palpate the entire abdomen for masses, hepatomegaly, and ascites. Umbilical hernias may be obscured by subcutaneous fat.
  2. Palpate the groin.
  3. Palpate the hernia to try to gently reduce it.

DIAGNOSTIC TESTS

A. None required if the hernia is easily reducible (depending on the type of hernia).
B. Complete blood count (CBC): white blood count (WBC) increased, hematocrit (Hct) increased.
C. Electrolytes: Na+ increased or decreased.
D. Abdominal radiography reveals abnormally high levels of gas in bowel.
E. Ultrasonography, if strangulation is suspected.
F. CT scan of the abdomen and pelvis may be indicated.

DIFFERENTIAL DIAGNOSES

A. Abdominal hernia.
B. Diastasis recti.
C. Ascites.
D. Abdominal wall tumor or cyst.
E. Bowel obstruction.

PLAN

A. General interventions:
  1. Assess type of hernia and further diagnostics that are recommended.
B. Client teaching:
  1. Discuss the hernia and available options for treatment.
  2. Teach the client signs and symptoms of strangulation.
  3. Instruct the client to refrain from heavy lifting.
  4. Advise the client to wear a support garment.
C. Medical and surgical management:

Reduction should not be attempted if there are signs of inflammation or obstruction. Reduction of a strangulated bowel leads to persistent ischemia or necrosis with no clinical improvement.

1. Try to reduce hernia unless strangulated. See Section II: Procedures, "Hernia Reduction (Inguinal/Groin)."

a. Easily reducible: Abdominal contents can be easily returned to their original compartment. Allows symptomatic relief.

b. Incarcerated: cannot be returned to its original compartment. The incarcerated tissue may be bowel, omentum, or other abdominal contents.

c. Strangulated: surgical emergency—blood supply to the herniated tissue is compromised.

2. Do not try to reduce strangulated hernias because reduction can cause gangrenous bowel to enter peritoneal cavity.

3. A truss fits snugly over a hernia to prevent abdominal contents from entering the hernial sac. It does not cure a hernia and is used only when the client is not a surgical candidate.

4. Surgery may be done laparoscopically or through an open procedure and by sutured or mesh repair, depending on the age of the person, type and size of the hernia, and the presence of strangulation.

FOLLOW-UP

A. Instruct the client to call the office if fever or severe pain occurs. Otherwise, no follow-up is required unless for postoperative repair. Postoperative follow-up is with the physician who performed the surgery.

EMERGENT ISSUES/INSTRUCTIONS

A. Consult a physician or send the client to the ED if the client has abdominal tenderness, discoloration, or edema at the site; fever; or signs of bowel obstruction.

CONSULTATION/REFERRAL

A. A surgical consultation may be warranted.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Incisional hernias are more common during pregnancy due to increased intra-abdominal pressure.

2. Bowel obstruction secondary to previous scarring may also be seen and is most common when the uterus emerges from the pelvis early in the second trimester, when the uterus is maximally distended at term, and in the immediate puerperium when the uterus promptly decreases in size.

3. Maternal smoking has been associated with an increased prevalence of omphalocele and gastroschisis.

B. Geriatrics:

1. Because of the anatomic position of the obturator hernia, the presentation is more common as a bowel obstruction than as a protrusion of bowel contents.

a. The geriatric population is more prone to develop electrolyte and acid–base imbalances from obstruction.

2. The geriatric population has a higher rate of ventral hernias attributed to the loss of muscle strength in the anterior abdominal wall and the prevalence of comorbidities that lead to an increased intra-abdominal pressure.

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HERNIAS, PELVIC

DEFINITION

A. Hernia is the protrusion of a peritoneum-lined sac through some defect from one anatomic space to another. As shown in Figure 14.3, there are three types of pelvic (inguinal) hernias distinguished by presentation:

1. Indirect: protrudes through internal inguinal ring; can remain in canal, exit external ring, or pass into scrotum; unilateral or bilateral.

2. Direct: protrudes through external inguinal ring; located in region of Hesselbach triangle; rarely enters scrotum.

3. Femoral: protrudes through femoral ring, femoral canal, and fossa ovalis.

INCIDENCE

A. The lifetime risk of development of a groin hernia is estimated at 25% for males and 2% for females.

B. Indirect inguinal hernias are the most common type of hernia. They affect both sexes. Indirect hernias occur at the extreme of ages, from 0.25% at 18 years of age to 4.2% at 75 to 80 years of age.

C. Direct inguinal hernias are less common than indirect inguinal hernias. They occur more often in males and are more common in those older than age 40. Primary inguinal hernias occur in 1% to 5% of infants and in 9% to 10% of those born prematurely.

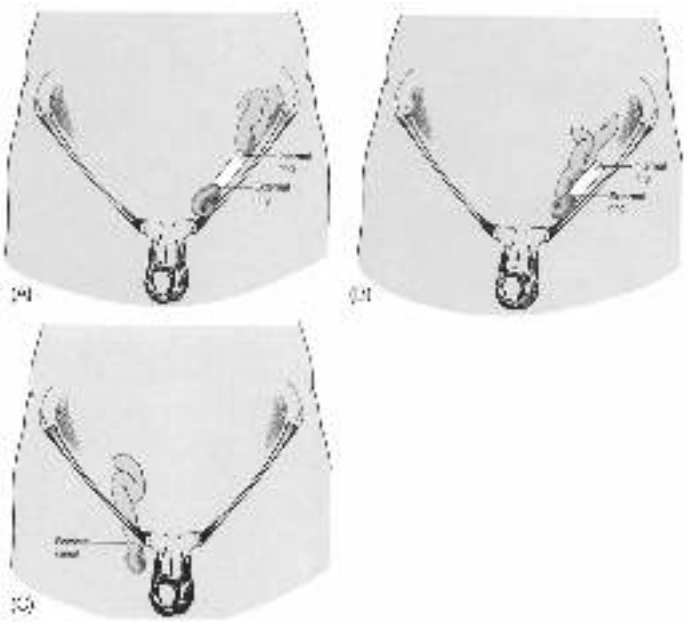


FIGURE 14.3 Pelvic hernias. (A) Indirect hernia comes down the canal and touches the fingertip on exam. (B) Direct hernia bulges anteriorly and pushes against the side of the finger on exam. (C) Femoral hernia protrudes through the femoral ring, femoral canal, and fossa ovalis, so the inguinal canal is empty on exam.

D. Femoral hernias are the least common type of hernia (<5% of groin hernias):

1. 35% to 40% of femoral hernias are not diagnosed until the client presents with strangulation or bowel obstruction.

2. They occur more often in females.

3. The incidence steadily increases with age and is higher among clients with recurrent hernias.

4. Right-sided presentation is more common than left.

E. Among inguinal hernias, a sliding component is found in 3%; they are overwhelmingly on the left side (left-to-right ratio, 4.5:1). Sliding hernias are much more common in males than in females, and the predominance increases with age.

F. Primary perineal hernias occur most often in older adult multiparous females.

PATHOGENESIS

A. Pelvic hernias occur because there is a potential space for protrusion—commonly of the bowel but occasionally of the omentum.

1. Indirect and direct hernias arise along the course that the testicle travels as it exits the abdomen and enters the scrotum during intrauterine life. Indirect hernias may be due to a congenital defect in which the processus vaginalis remains patent.

2. Femoral hernias occur at the fossa ovalis, where the femoral artery exits the abdomen.

PREDISPOSING FACTORS

A. Pregnancy.
B. Straining.
C. Age.
D. Obesity.
E. Sex.
F. Repetitive stress/hard physical labor.
G. Congenital defect.
H. Premature birth.
I. Chronic cough/chronic obstructive pulmonary disease (COPD).
J. Chronic constipation.
K. Family history of hernia.
L. History of an abdominal aortic aneurysm (AAA).
M. Straining to urinate.
N. Previous inguinal hernia or hernia repair.
O. Collagen vascular disease.
P. Peritoneal dialysis.

COMMON COMPLAINTS

A. Bulging or swelling localized in the groin or scrotum.
B. Dull ache in lower abdomen or groin.
C. Swelling of labia majora in females.
D. Bowel obstruction symptoms may be present with an incarcerated hernia.

OTHER SIGNS AND SYMPTOMS

A. Ability to reduce hernia.
B. Exacerbation on standing, straining, or coughing.
C. Strangulation.
D. Systemic toxicity secondary to ischemic bowel is possible. Signs of sepsis include fever, tachycardia, hypotension, vomiting, and confusion.

1. Colicky abdominal pain.

2. Extreme tender groin mass.

3. Hyperperistalsis.

4. Edema.

5. Discoloration.

SUBJECTIVE DATA

A. Review time of onset, duration, course of hernia, and swelling.
B. Review any symptoms and quality of pain. Blatant pain with hernias is unusual, and its presence should raise the possibility of incarceration or strangulation.
C. Ask the client about history of straining, trauma, physical labor, and pregnancy.
D. Inquire about symptoms of obstruction or strangulation of entrapped bowel: pain, nausea, and vomiting. Groin pain and tenderness are generally absent in strangulated femoral hernias.
E. Determine if the client has or can reduce the hernia.
F. Review irritating (e.g., exercise, straining, cough) and alleviating factors.
G. Review bowel habits and patterns, particularly constipation.
H. Evaluate history of COPD and cough.

PHYSICAL EXAMINATION

A. Physical examination is the same for all types of hernias and is directed at determining the type of hernia and whether it is reducible, incarcerated, or strangulated. Perform the exam while the client is standing and supine. Palpation is best done with the client standing:
B. Check temperature (if indicated), pulse, respirations, and blood pressure (BP).
C. Inspect:

1. Inspect for discoloration and edema of the herniated area.

2. Inspect for visible hernia. Instruct the client to perform Valsalva maneuver to increase intra-abdominal pressure.

3. Perform transillumination of the scrotum to evaluate any bowel contents.

4. Inspect for presence of ascites.

D. Auscultate:

1. Abdomen for bowel sounds.

E. Palpate:

1. Palpate the groin for lymphadenopathy, masses, and tenderness. *The right side is more commonly affected in both males and females*:

a. Males: Using the second or third finger, invaginate the scrotal skin, with and without cough and strain. There will be some degree of pressure with this maneuver, but a true hernia can typically be felt as a "silky" impulse tapping against the finger. Palpate the scrotum: Scrotal lump is either soft or unusually firm.

b. Females: Visually examine for a bulge, and then place two or three fingers across the inguinal canal and ask the client to bear down or cough to elicit the characteristic bulge or impulse. Palpate the labia for swelling: either soft or unusually firm.

DIAGNOSTIC TESTS

A. History and physical examination remain the best means of diagnosing hernias.
B. Perform ultrasonography for abdominal masses and strangulation.
C. MRI appears to be able to differentiate inguinal and femoral hernias with high sensitivity.
D. Sigmoidoscopy is not recommended as a screening test.
E. Plain abdominal x-rays are of limited value in evaluating an incarcerated hernia. ▶

F. Karyotyping should be considered when a testicle is palpable in the inguinal canal or found at herniorrhaphy in phenotypic females.
G. Routine laboratory work is not recommended.

DIFFERENTIAL DIAGNOSES

A. Inguinal or pelvic hernia.
B. Acute conditions:
1. Testicular torsion causes sudden, excruciating pain in or around the testicle, which may spread to the lower abdomen; the pain may get worse with standing. Other signs and symptoms include swelling, rising of the affected testicle, nausea, vomiting, fever, and fainting or light-headedness.
2. Epididymitis.
C. Nonacute conditions:
1. Testicular tumor.
2. Muscle strain.
3. Hip arthritis.
4. Undescended testicle.
5. Hydrocele.
6. Varicocele.
7. Spermatocele.
8. Lymphadenopathy.
D. Bowel obstruction.

PLAN

A. General interventions:
1. Evaluate hernia and determine diagnostic testing and referral options.
B. Client teaching:
1. Instruct the client to call the office right away if they find a lump or swelling in the scrotum, even if it is small or painless. Testicular tumors are usually painless.
2. Discuss condition and treatment options with the client:
a. Surgery is the only effective treatment.
b. Watchful waiting rather than surgical repair is an option if the client is asymptomatic as long as they are aware of the risk and understands the need for prompt attention should symptoms of complication occur.
c. Watchful waiting is not recommended in females, given their higher prevalence of femoral hernias, which are associated with a high risk of strangulation.
d. Nonsurgical therapy for groin hernias is the use of a truss. There are insufficient data to determine the efficacy of trusses in controlling symptoms. A truss has the potential risk of bowel constriction; prolonged use of a truss can lead to atrophy of the spermatic cord or fusion to the hernial sac.
3. Instruct the client on signs of strangulation.
4. Instruct the client to avoid heavy lifting and straining to have bowel movements (BMs) because they increase intra-abdominal pressure.
C. Medical and surgical management:
1. Gently reduce a groin hernia while the client lies supine with hips slightly flexed to relax the abdominal muscles. See Section II: Procedures, "Hernia Reduction (Inguinal/Groin)."

FOLLOW-UP

A. Preoperative:
1. Excessive waiting time for elective repair increases the risk of strangulation, bowel resection, and mortality, especially with older clients.
B. Postoperative evaluation for hernia recurrences as needed:
1. Immediately from repair.
2. Greater than 6 months and up to 5 years from repair.
3. Late recurrences beyond 5 years from the repair.

EMERGENT ISSUES/INSTRUCTIONS

A. Strangulated hernias are nonreducible, and blood supply to protruded tissue is compromised. Refer clients for immediate surgical intervention.
B. Instruct the client to return to the office if fever, severe pain, or strangulation occurs.

CONSULTATION/REFERRAL

A. Refer clients with femoral hernias to a physician. These hernias need to be repaired as soon as possible due to increased risk of incarceration and strangulation.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Preexisting groin hernias may become more symptomatic during the first trimester of pregnancy. The symptoms must be differentiated from round ligament pain. Umbilical hernia may become worse in pregnancy due to increased intra-abdominal pressure.
B. Geriatrics:
1. Groin hernias are one of the most frequently encountered pathologies occurring in old age, secondary to the presence of constipation, coughing, abdominal fat deposit, and loss of strength of the abdominal wall.
2. Signs and symptoms of prostatism are frequently present in males with hernias and may require relief before herniorrhaphy.
3. The higher rate of comorbidities and the usage of general anesthesia increase the risk of complications in the older client.
4. For the older adult with existing pelvic hernias, nurse practitioner (NPs) should practice special consideration of the following *geriatric syndrome*:
a. Pain: Poorly relieved pain is an important cause of functional impairment at any age group:
i. Older adults have a diminished sensorium, allowing pathology to advance to a dangerous point prior to symptom development.
ii. Older adults with abdominal pain may present with altered mental status. Cognitive impairment can make assessment and diagnosis of pain more difficult.
iii. For dementia or noncommunicative clients, the American Medical Directors Association has endorsed the Pain Assessment in Advanced Dementia (PAINAD) Scale, which scores the following items:
1) Breathing: Examples include normal, labored, hyperventilation.
2) Negative vocalization: Examples include moaning, negative language, crying.
3) Facial expression: Examples include smiling, frowning, grimacing.
4) Body language: Examples include relaxed, tense, rigid, or striking out.
5) Consolability: Examples include extent to which a caregiver is able to console, distract, or reassure.

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HIATAL HERNIA

DEFINITION

A. Hiatal hernia occurs when portions of the stomach or other abdominal cavity organs herniate through the esophageal hiatal of the diaphragm. The client may be asymptomatic, and the herniation may be found incidentally in the diagnostic evaluation of other problems. There are four types of hiatal hernia:

1. Type I: sliding hernia. A portion of the stomach, gastroesophageal (GE) junction, slides in and out of the hiatus. The fundus of the stomach remains below the GE junction.

2. Type II: paraesophageal hernia. The gastric fundus of the stomach slides through the hiatus and lies next to the esophagus.

3. Type III: combined. Combination of sliding and paraesophageal hiatus hernia.

4. Type IV: complex paraesophageal hernia. The stomach, small and large bowels, spleen, pancreas, or liver are pushed up into the chest through a large defect.

INCIDENCE

A. The frequency of hiatal hernias increases with age, from 10% in clients younger than 40 years to 70% in clients older than 70 years.

B. Sex: Hiatal hernias are more common in females.

C. The most common hiatal hernia is the type I sliding hernia (95%).

D. Type II through type IV hernias make up the remaining 5% and are referred to as paraesophageal hernias (PEH).

E. The least common herniation is type II.

PATHOGENESIS

A. The exact cause of a hiatal hernia is unknown. It is known for the widening of the GE junction and/or laxity of the phrenoesophageal (gastrosplenic and gastrocolic ligaments). A hiatal hernia may also be a congenital defect.

PREDISPOSING FACTORS

A. Obesity.
B. Pregnancy.
C. Cough (chronic).
D. Straining with lifting or straining to have bowel movement.

COMMON COMPLAINTS

A. Asymptomatic (found incidentally, on upper endoscopy or other radiographic imaging being performed for other problems).
B. Heartburn.
C. Upper abdominal pain.
D. Vague, intermittent chest discomfort or pain.
E. GE reflux or "acid brash."
F. Trouble swallowing.
G. Choking.

OTHER SIGNS AND SYMPTOMS

A. Burping.
B. Hiccups.
C. Coughing.
D. Vomiting blood.
E. Blood in stools.
F. Weight loss.

POTENTIAL COMPLICATIONS

A. Strangulation/incarceration.
B. Gastric volvulus.
C. Perforation.
D. Respiratory compromise secondary to the hernia.

SUBJECTIVE DATA

A. Ask client what activity brought about or preceded the episode.

B. What are alleviating and aggravating factors?

C. Review medical history for a diagnosis of gastroesophageal reflux disease (GERD):

1. If the client has a diagnosis of GERD, has the client been prescribed and are they taking proton pump inhibitors (PPIs)?

2. Has the client had an esophagogastroduodenoscopy (EGD)? Was a hiatal hernia noted at the time of the procedure?

D. Review medical history for cardiac diagnosis.

E. Ask the client to list all medications currently being taken, including herbals and over-the-counter (OTC) medications. Specifically review whether the client is taking OTC antacids, H2 blockers, or OTC PPIs.

PHYSICAL EXAMINATION

A. Check pulse, respirations, and blood pressure (BP). Measure height and weight to calculate body mass index (BMI).

B. Inspect:

1. Inspect general appearance and observation of respiration, noting any distress.

2. Assess swallowing ability.

C. Auscultate:

1. Auscultate abdomen in all four quadrants.

2. Conduct a complete heart and lung exam.

D. Palpate:

1. Palpate upper gastric region for tenderness or distension.

2. Palpate abdomen for organomegaly or masses.

E. Rectal examination (if indicated for hematemesis or blood in the stool).

DIAGNOSTIC TESTS

A. The 2013 Society of American Gastrointestinal and Endoscopic Surgeons (SAGES) guidelines recommend tests should be done to diagnose hiatal hernia only if they will change clinical outcome.

1. Barium swallow (identifies anatomy and size of hernia).

2. Esophagogastroduodenoscopy (EGD).

3. High-resolution manometry.

4. CT scan.

5. Large hiatal hernias may cause iron deficiency anemia:

a. Complete blood count (CBC).
b. Iron.
c. Ferritin.

DIFFERENTIAL DIAGNOSES

A. Hiatal hernia.
B. GERD.
C. Cholecystitis.
D. Esophageal spasm.
E. Angina pectoris.
F. Coronary artery disease.
G. Cancer (gastric or esophageal).

PLAN

A. General interventions:

1. Management depends on the cause and severity of symptoms. Asymptomatic sliding hernias do not require surgical repair. Management of sliding hernias with reflux includes managing symptoms of GERD.

B. Client teaching:

1. Educate client about modifying controllable risk factors, such as keeping diabetes and hypertension under control, diet and weight control, exercise, and stopping smoking.

C. Dietary management:

1. There is no specific diet for a hiatus hernia; however, dietary modifications for GERD are encouraged.
2. Food should be cut up into small bite-sized pieces and thoroughly chewed.

D. Pharmaceutical therapy:

1. There is no medication specific for a hiatal hernia; however, symptomatic acid reflux can be treated by neutralizing acid with antacids or by blocking acid secretion with H2 receptor blocking agents for more potent PPIs:
   a. PPIs increase the risk of *Clostridioides difficile* infection.
   b. PPIs are associated with bone loss and fractures.

E. Medical and surgical management:

1. Surgical repair is not indicated in the absence of reflux disease.
2. Age and client comorbidities should be considered in the routine elective repair of completely asymptomatic PEH.
3. The SAGES guidelines recommend that, during operations for Roux-en-Y gastric bypass (RYGB), sleeve gastrectomy, and placement of adjustable gastric bands, all hiatal hernias should be repaired.
4. All symptomatic types II to IV PEH should be repaired, especially in the presence of acute obstructive symptoms or volvulus:
   a. There are three major types of surgical repair: Nissen fundoplication, Belsey fundoplication, and Hill repair. These procedures offer relief in 80% to 90% of clients.

FOLLOW-UP

A. Follow-up is determined by client's needs, severity of symptoms, and whether complications are present.

EMERGENT ISSUES/INSTRUCTIONS

A. An emergent surgical repair of a hiatal hernia is required in clients with a gastric volvulus, uncontrolled bleeding, obstruction, strangulation, perforation, and respiratory compromise.

CONSULTATION/REFERRAL

A. Consider a surgical consultation for hiatus repair for:

1. Clients who have no relief from PPIs.
2. Pulmonary complications that are linked to GERD, including asthma and recurrent aspiration pneumonia.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. The incidence of a hiatal hernia increases due to the increase in intra-abdominal pressure from the uterus. In pregnancy, the most common type of hernia is hiatal hernia present in 15% to 20% of pregnancies, appearing during the second trimester.

B. Geriatrics:

1. For the older adult with existing GERD, nurse practitioner (NPs) should practice special consideration of the following *geriatric syndromes*:
   a. Falls:
      i. Consistently assess for falls. PPIs are associated with osteopenia/osteoporosis.
      ii. At the minimum, older adults should be asked how many times they have fallen since their last visit. A reported fall should trigger questions about whether the fall led to injury, a visit to urgent care or hospital ED, or a hospital admission.
      iii. Safety measures to reduce falls in the home include removing small area carpets, keeping night lights along the path to the bathroom, and installing a raised toilet seat or nearby grab bars. *See Client Teaching Guide for Chapter 3, "Safety Issues: Fall Prevention."*
   b. Weight loss/appetite suppression: Weight loss is a predictor of mortality. Clinically significant weight loss is considered to be 5% loss of usual body weight in 3 months or 10% in 6 months.
   c. Due to gastrointestinal (GI) distress, abdominal discomfort, and diarrhea, older adults are at increased risk of avoiding eating.
   d. Monitor weight patterns/BMI. (Caution: BMI is not used to identify loss of muscle mass.)
   e. Monitoring both serum albumin and anemia: Hypoalbuminemia and anemia are not necessarily associated with low bodyweight. They can be caused by decreases in liver protein synthesis:
      i. Serum albumin concentration is commonly recommended.
      ii. Hemoglobin (Hgb), prealbumin, and transferrin.
      iii. Cholesterol.
2. Beers Criteria cautions for PPIs:
   a. Due to polypharmacy, a full review of medications, herbals, and OTC drugs should be undertaken to identify medications that increase symptoms of GERD, medications that should be avoided in the geriatric population, duplicate medications, and medications that are able to be decreased or deprescribed. (Refer to section on deprescribing in Chapter 3, "Adult-Geriatric Assessments.")
   b. Prescribe the lowest dose for the shortest amount of time.
   c. PPIs are on the American Geriatrics Society 2021 Beers Criteria for potential inappropriate medications use in older adults. (Refer to Appendix C, Table C.1.) Avoid scheduled use of PPIs for greater than 8 weeks unless needed for high-risk clients. PPIs are associated with the following:
      i. Osteoporosis/osteopenia, prolonged exposure may increase of fractures.
      ii. Community-acquired pneumonia.
      iii. *C. difficile* infections and diarrhea.
      iv. Vitamin B_{12} deficiency and hypomagnesium.
   d. See Appendix D for the PPI deprescribing algorithm from deprescribing.org.

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IRRITABLE BOWEL SYNDROME

DEFINITION

A. Irritable bowel syndrome (IBS) is the most common of the gastrointestinal (GI) motility disorders. IBS is responsible for significant direct and indirect healthcare costs. It is a chronic relapsing functional disturbance of intestinal motility marked by a common symptom complex that includes bloating and abdominal pain or discomfort associated with defecation.

B. Bladder dysfunction has been identified in 50% of clients with IBS. Clients commonly transition between subgroups.

C. According to the Rome III criteria, IBS is the presence of recurrent abdominal pain or discomfort at least 3 days/mo that occurs in association with altered bowel habits over a period of at least 3 months.

D. IBS is defined by symptom-based diagnostic criteria, in the absence of detectable organic causes. Rome III criteria for IBS is related to stool characteristics:

1. IBS with diarrhea predominant (IBS-D): loose stools (small volume, pasty/mushy or watery) more than 25% of the time and hard stools less than 25% of the time.

2. IBS with constipation (small, hard, pellet like stools) predominant (IBS-C): hard stools more than 25% of the time and loose stools less than 25% of the time.

3. IBS with alternating bouts of constipation and diarrhea, mixed or cyclic pattern (IBS-M): both hard and soft stools more than 25% of the time.

E. On clinical grounds, other subclassifications are used:

1. Based on symptoms:

a. IBS with predominant bowel dysfunction.
b. IBS with predominant pain.
c. IBS with predominant bloating.

2. Based on precipitating factors:

a. Postinfectious (PI-IBS).
b. Food-induced (meal-induced).
c. Stress-related.

INCIDENCE

A. IBS is common, accounting for about 50% of GI complaints seen by healthcare professionals, and is a major cause of morbidity in the United States. Studies suggest nearly 20% of all adults suffer from some form of the condition; however, only a fraction seek medical help. IBS is recognized in children; symptoms consistent with IBS are reported in 16% of students age 11 to 17 years. IBS is not described in preschool-age children.

B. The condition affects between 5% and 10% of healthy people at any one point in time and, in most people, runs a relapsing and remitting course.

C. IBS with diarrhea is more common in males.

D. IBS with constipation is more common in females.

E. The prevalence is lower in people aged 50 years or older.

F. The American College of Gastroenterology (ACG) has recognized IBS as a key component of Gulf War syndrome.

PATHOGENESIS

A. IBS has an absence of detectable pathology, and laboratory tests are unrevealing. The understanding of IBS has evolved from a disturbance in bowel motor activity to a more integrated understanding of visceral hypersensitivity and brain–gut interaction. It is thought to be both a normal response to severe stress and a learned visceral response to stress:

1. Nonpropulsive colonic contractions lead to IBS-C predominant.

2. Increased contraction in the small bowel and proximal colon with diminished activity in the distal colon lead to IBS-D predominant.

3. Most clients with functional disorders appear to have inappropriate perception of physiologic events and altered reflex responses in different gut regions.

4. The brain–gut transmitters act at different sites in the brain and gut and lead to varied effects on GI motility, pain control, emotional behavior, and immunity. Serotonin plays a critical role in the regulation of GI motility, secretion, and sensation. Studies have shown that IBS may be related to an imbalance in mucosal serotonin and 5-hydroxytryptamine (5-HT) availability caused by defects in 5-HT production, serotonin receptors, or serotonin transport.

PREDISPOSING FACTORS

A. Age:

1. IBS mainly occurs between the ages of 15 and 65 years:

a. 50% of IBS occur prior to age 35.
b. 40% of IBS occur between ages 35 and 50.

B. Sex:

1. Females are two to three times more likely to have IBS.

C. Emotional factors and situational stress.

D. Prior GI infection-induced IBS.

E. Genetics is considered a factor.

F. Carbohydrate intolerance may produce significant symptoms.

COMMON COMPLAINTS

A. Chronic relapsing stool pattern:

1. Diarrhea, over three loose stools per day. OR

2. Alternation of diarrhea with constipation. Diarrhea is typically small in volume, has visible mucus, and may follow a hard movement by a few hours. OR

3. Constipation: less than three bowel movements (BMs) per week.

B. Feeling of incomplete evacuation.

C. Abdominal distension and bloating.

D. Straining with BMs.

E. Aching or cramps in periumbilical or lower abdominal region.

F. Pain relief with BM.

OTHER SIGNS AND SYMPTOMS

A. Change in bowel function.

B. Clear mucous stool.

C. Pain may be precipitated by meals.

D. Pain radiates to left chest or arm, from gas in splenic flexure. Nocturnal pain is unusual and is considered a warning sign.

E. Flatulence.

F. Nausea.

G. Anxiety.

H. Depression.

I. Preoccupation with bowel symptoms.

J. Extraintestinal symptoms:

1. Dysmenorrhea.

2. Urinary frequency, urgency, and incomplete bladder emptying.

3. Impaired sexual function and dyspareunia.

4. Fibromyalgia.

K. Menses may exacerbate IBS symptoms.

SUBJECTIVE DATA

A. Review pattern of main symptoms, including onset, duration, and usual course.
B. Ask the client what are the predominant symptoms: abdominal pain, diarrhea, or constipation.
C. Review the client's history for stress factors and ask if recurrent symptoms occur in relation to them.
D. Ask what other symptoms occur with the pain, diarrhea, or constipation, such as bloating, blood in stool, or nighttime BMs. **Bleeding, weight loss, and nocturnal diarrhea are not characteristic of IBS. IBS symptoms disappear during sleep.**
E. Establish client's normal weight history, and determine amount of weight loss, if any, over what time period.
F. Review the client's diet, including the following:
1. Response to milk or lactose products.
2. Artificial sweeteners.
3. Alcohol intake.
4. Irregular or inadequate meals.
5. Insufficient fluid intake.
6. Excessive fiber intake.
7. Obsession with dietary hygiene.
8. Response to gluten (wheat, barley, rye) ingestion.

G. Inquire about the client's prescription, herbal, and over-the-counter (OTC) medications. Ask specifically about the use of laxatives.
H. Review travel and food history for dominant history of diarrhea.
I. Ask the client if there is a family history of colon cancer, ulcerative colitis (UC), Crohn's disease (CD), or malabsorption.
J. Is there any fever accompanying lower abdominal pain?
K. What is the relation of symptoms to menstruation?

PHYSICAL EXAMINATION

A. Check temperature (if indicated), pulse, respirations, and blood pressure (BP). Measure height and weight to calculate a body mass index (BMI).
B. Inspect:
1. Observe general appearance. Does the client appear anxious or depressed?
2. Inspect abdominal contour for masses and bulges.

C. Auscultate:
1. Abdomen for bowel sounds; note whether they are normal or mildly hyperactive.

D. Percuss:
1. Abdomen for tympany or dullness.

E. Palpate:
1. Abdomen:
   a. Evaluate the abdomen for mild tenderness, rigidity, guarding, and masses.
   b. Evaluate for hepatosplenomegaly.
   c. Evaluate for lymphadenopathy.

F. Rectal exam:
1. Check for masses and tenderness.
2. Obtain stool for diagnostic tests.
3. Rectal exam is normal with IBS.

DIAGNOSTIC TESTS

A. The ACG Task Force on IBS recommends that individuals younger than 50 years of age who do not have alarm features need not undergo routine colonic imaging. Individuals with IBS symptoms who have alarm features, such as anemia or weight loss, or those who are older than 60 years of age should undergo colonic imaging to exclude organic disease.

TABLE 14.19 ROME III DIAGNOSTIC CRITERIA FOR IBS

Onset of symptoms at least 6 months before diagnosis. Recurrent abdominal pain or discomfort for more than 3 days per month during the past 3 months.

At least two of the following features:
A. Improvement with defecation.
B. Association with a change in frequency of stool.
C. Association with a change in stool form.

B. Diagnosis of IBS is usually suspected on the basis of the client's history and physical examination without additional tests (Table 14.19).
C. Diagnostic tests are performed to exclude organic disease that may masquerade as IBS:
1. Complete blood count (CBC).
2. Sedimentation rate or C-reactive protein.
3. In clients presenting with chronic diarrhea, the American Gastroenterology Association (AGA) suggests against the use of erythrocyte sedimentation rate or C-reactive protein to screen for IBD.
4. Serum potassium, if the client is on diuretics; hypokalemia may reduce bowel contractility and produce an ileus.
5. Blood glucose, if diarrhea predominates; rule out diabetes mellitus, which may present as diarrhea due to diabetic gastroenteropathy.
6. Thyroid function study.
7. Stool specimen: Culture for leukocytes and fat, ova and parasites, and occult blood. **Leukocyte-free mucus is a hallmark of IBS.**
8. Stool cultures for *Clostridioides difficile* toxin assay, if clinically indicated.
9. Barium enema (BE) and/or proctosigmoidoscopy for severe signs and symptoms, after consultation or referral.
10. Celiac serology, if appropriate.
11. Mucosal biopsy is appropriate if a colonoscopy or sigmoidoscopy is performed.
12. Esophagogastroduodenoscopy (EGD) and distal duodenal biopsy in clients with diarrhea should be considered to rule out celiac disease, tropical sprue, giardiasis, and for clients in whom abdominal pain and discomfort are located more in the upper abdomen.
13. Hydrogen breath test to evaluate lactose intolerance and small intestinal bowel overgrowth (SIBO).

DIFFERENTIAL DIAGNOSES

A. IBS.
B. Refer to Table 14.19 for common and red flag differential diagnoses for IBS:
1. Inflammatory bowel disease (IBD):
   a. CD.
   b. UC.
2. Viral or bacterial gastroenteritis or acute diarrhea due to protozoa or bacteria.
3. Diverticulitis.
4. GI neoplasm.
5. Medications:
   a. Laxative abuse.
   b. Drug(s) side effects.

C. Lactose insufficiency/deficiency.
D. Diabetes.
E. Celiac spruce/gluten etiology.

▶

F. Endometriosis/pelvic inflammatory disease (PID).
G. Zollinger–Ellison syndrome.

PLAN

A. General interventions:
1. Advise the client to keep a diary of events of BMs and precipitating factors. A BM record is available at www.nhsggc.org.uk/media/241191/nhsggc-bristol-stool-chart.pdf.
2. Encourage the client to quit smoking because nicotine may aggravate symptoms.
3. Recommend daily exercise to reduce stress.
4. Stress management should be encouraged, including counseling, tapes, meditation, and yoga.
B. Client teaching: *See Client Teaching Guide for this chapter, "Irritable Bowel Syndrome."*
C. Dietary management:
1. Encourage lifestyle changes and dietary modification first. Prescribe a high-fiber diet.
2. Tell the client to avoid foods that aggravate the bowel. Avoid gas-producing foods, such as broccoli, beans, onions, garlic, and so forth. Advise to follow a low FODMAP (fermentable oligosaccharides, disaccharides, monosaccharides, and polyols) diet. See Appendix B, "Dietary Recommendations," for the FODMAP diet. When diarrhea predominates, dietary review is essential for clues of intolerance to lactose or sorbitol.
3. Referral to registered dietary nutritionist (RDN) to collaborate with making dietary changes.
4. Reassess frequently if dietary changes are improving symptoms and/or client is compliant with diet.
D. Pharmaceutical therapy:
1. Advise the client to stop all nonessential medications that may affect bowel function, especially irritant laxatives. Avoid narcotics, depressants, and other long-term drug use if possible.
2. Recommend eliminating sorbitol-containing candy and restricting lactose-containing milk products.
3. Drug of choice: psyllium hydrophilic mucilloid (Metamucil) one tablespoon per day in 8 oz of juice or water, followed with another 8 oz of liquid. This treats both diarrhea and constipation.
4. Pain relief:
a. Opiates should be avoided due to the risk of dependence and addiction in chronic conditions.
b. Nonsteroidal anti-inflammatory drugs (NSAIDs) have an undesirable side effect on the GI tract.
c. Antispasmodics and anticholinergic agents (IBS-D predominant):
i. Hyoscyamine (Levsin, Levbid):
1) Levsin 0.125 to 0.25 mg (one to two tablets) orally or sublingual Q4H; not to exceed 12 tablets a day.
2) Levbid 0.375 to 0.75 mg orally BID.
3) Hyoscyamine is available in immediate-release, extended-release, time capsules, biphasic tablets, elixir, and drop formulations.
ii. Dicyclomine (Bentyl) 20 to 40 mg QID; discontinue if not effective within 2 weeks or if 80 mg daily is associated with adverse effects. Also available in intramuscular formulation.
d. Tricyclic antidepressants for diarrhea predominant IBS. *Tricyclics should be avoided for constipated clients*:
i. Amitriptyline (Elavil) 10 mg every bedtime initially, titrate up slowly to 75 mg/d at bedtime as needed.
ii. Desipramine (Norpramin) 10 mg every bedtime initially, titrate up slowly up to 75 mg/d.
iii. Imipramine (Tofranil) 10 to 50 mg orally every bedtime.
5. Selective serotonin reuptake inhibitors (SSRIs):
a. Paroxetine (Paxil) 10 to 60 mg/d.
b. Citalopram (Celexa) 5 to 60 mg/d.
6. Antidiarrheals:
a. Opiate-derived antidiarrheals are reserved for very severe cases secondary to the potential for abuse.
b. Loperamide (Imodium):
i. Adults: 2 to 12 mg orally divided into BID/TID doses; doses differ greatly among individuals.
c. Alosetron (Lotronex), a 5-HT3 receptor antagonist, is indicated for females with severe IBS-D predominant. Starting dose is 0.5 mg BID; may increase to 1 mg BID after 4 weeks of starting dose. Discontinue if no relief after 4 weeks of 1-mg dosing.
7. Laxatives and stool softeners (IBS-C predominant) along with dietary measures and fiber supplements:
a. Mineral oil 15 to 45 mL orally daily or divided into TID dosing.
b. Stimulant laxatives may be necessary intermittently for short periods, but prolonged use of stimulant laxatives should be avoided.
c. Lactulose is a colonic acidifier that promotes laxation. Transit time through the colon may be slow; 24 to 48 hours may be required to produce a BM. The usual dose is 1 to 2 tablespoons daily. Lactulose contains galactose and lactose and should be used with caution for clients with diabetes.
8. Lubiprostone (Amitiza), a selective C-2 chloride-channel activator, is indicated for females 18 years and over with IBS-C predominant. 8-µL doses BID with food and water:
a. Lubiprostone should not be used in clients with mechanical bowel obstruction or preexisting diarrhea.
b. Clients should have a documented negative pregnancy test before starting therapy and should be advised to use contraception when taking lubiprostone.
9. Probiotics.
10. Linaclotide (LINZESS), a guanylate cyclase-C (GC-C) agonist, was recently released to market for both IBS-C and chronic idiopathic constipation (CIC).
11. Eluxadoline (Viberzi), a mu-opioid receptor agonist, is indicated for IBS-D predominant. There are two recommended dosings:
a. Dosing is 100 mg tablet BID taken with food.
b. The alternative dosing is 75 mg BID taken with food for clients with the following conditions:
i. Do not have a gallbladder.
ii. Unable to tolerate 100 mg BID.
iii. Are receiving concomitant OATP1B1 inhibitor, such as the lipid-lowering drug gemfibrozil and its metabolite gemfibrozil 1-O-beta.
iv. Have mild or moderate hepatic impairment.
c. If a dose is missed, clients should not compensate by taking two doses at once.

FOLLOW-UP

A. Reevaluate the effectiveness of treatment in 2 weeks. Treatment may be challenging for symptom management and numerous tests may be inconclusive, but rule out pathology.

B. If symptoms persist without relief, have the client return as needed.

C. Return if diarrhea lasts more than 2 weeks on medication therapy.

CONSULTATION/REFERRAL

A. Refer to a gastroenterologist for red flag symptoms:

1. Red flag features that require referral to a specialist for endoscopic evaluation include:

a. Increased rectal bleeding.
b. Weight loss.
c. Unexplained iron-deficiency anemia.
d. Abdominal symptoms during the night.
e. Strong family history of bowel disorders (e.g., colon cancer, celiac sprue, IBD).

B. Alosetron (Lotronex) is prescribed under a restricted distribution program through a gastroenterologist.

C. Consult a physician if all treatment options fail.

D. Consider a psychiatric consultation, if indicated, for anxiety, depression, somatization, and symptom-related fears.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. Annual rectal examination and sigmoidoscopy are recommended after age 50.

2. When constipation predominates, rule out malignancy, particularly in clients older than age 40 who have weight loss or a family history of colon cancer.

B. Geriatrics:

1. For the older adult with existing IBS, nurse practitioners (NPs) should practice special consideration of the following *geriatric syndromes*:

a. Depression: Anxiety and depression are common in IBS and are factors that lead to decreased health-related quality of life. A depression screen should be done at each office visit:

i. An online Patient Health Questionnaire-9 (PHQ-9) is available at www.mdcalc.com/phq-9-patient-health-questionnaire-9.

ii. The Geriatric Depression Scale, Short Form, a 15-item questionnaire that can be quickly applied in the clinical setting, is available at web.stanford.edu/yesavage/GDS.english.short.html.

b. Pain: Poorly relieved pain is an important cause of functional impairment at any age group:

i. Older adults have a diminished sensorium, allowing pathology to advance to a dangerous point prior to symptom development.

ii. Older adults with abdominal pain may present with altered mental status. Cognitive impairment can make assessment and diagnosis of pain more difficult.

iii. For dementia or noncommunicative clients, the American Medical Directors Association has endorsed the Pain Assessment in Advanced Dementia (PAINAD) Scale, which scores the following items:

1) Breathing: Examples include normal, labored, hyperventilation.

2) Negative vocalization: Examples include moaning, negative language, crying.

3) Facial expression: Examples include smiling, frowning, grimacing.

4) Body language: Examples include relaxed, tense, rigid, or striking out.

5) Consolability: Examples include extent to which a caregiver is able to console, distract, or reassure.

2. Beers Criteria caution:

a. A full review of medications, herbals, and OTC drugs should be undertaken to identify medications that should be avoided in the geriatric population, duplicate medications, and medications that are able to be decreased or deprescribed. (Refer to section on deprescribing in Chapter 3, "Adult-Geriatric Assessments.")

b. Refer to Appendix C, Table C.1 to identify drugs that have highly anticholinergic side effects and are potentially inappropriate medications for use in older adults, including the following:

i. First-generation antihistamines.
ii. Antispasmodics.
iii. Antidepressants.
iv. Opioids.
v. Calcium channel blockers.
vi. Anticonvulsants.
vii. Muscle relaxants.

RESOURCES

Rome Foundation: https://romeonline.org

American Gastroenterological Association https://gastro.org

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JAUNDICE

DEFINITION

A. Jaundice is a yellow tinge of the skin or mucous membranes. It is a symptom, not a disease. The diagnostic approach begins with gathering a comprehensive history, physical examination, and screening labs. The differential diagnosis is formulated, and further testing may be warranted. The onset of jaundice usually prompts the client or family to seek medical attention. Jaundice can reflect a medical emergency secondary to massive hemolysis, ascending cholangitis, unconjugated hyperbilirubinemia in the neonatal period, and fulminant liver failure.

INCIDENCE

A. Incidence is variable according to pathogenesis, age, and population.

PATHOGENESIS

A. The mechanism responsible for jaundice includes excess bilirubin production, decreased hepatic uptake, impaired conjugation, intrahepatic cholestasis, extrahepatic obstruction, and hepatocellular injury (Table 14.20). However, it ▶

TABLE 14.20 CLASSIFICATION OF JAUNDICE ACCORDING TO BILE PIGMENT AND BY MECHANISM

Unconjugated Hyperbilirubinemia	Conjugated hyperbilirubinemia
Increased/overproduction of bilirubin	Hepatocellular injury/disease
Impaired/decreased hepatic uptake of bilirubin	Intrahepatic cholestasis
Impaired/decreased conjugation	Extrahepatic cholestasis (biliary obstruction)

is important to recognize that more than one mechanism can be operating in a given case (i.e., sickle cell anemia and HIV).

1. Excess bilirubin production results from accelerated red cell destruction. The excessive amounts of hemoglobin (Hgb) and resultant bilirubin released into the bloodstream overwhelm the liver's normal capacity for uptake, and an unconjugated hyperbilirubinemia ensues.

2. With decreased uptake and conjugation, there is often a concurrent, acquired illness such as infection, cardiac disease, or cancer. Hereditary conditions, such as Gilbert and Crigler–Najjar syndromes, are responsible.

3. Intrahepatic cholestasis may occur at a number of levels: intracellularly (e.g., hepatitis), at the canalicular level (when estrogen-induced), at the ductule (phenothiazine exposure), at the septal ducts (primary biliary cirrhosis), and at the intralobular ducts (cholangiocarcinoma).

4. Extrahepatic obstruction occurs when stone, stricture, or tumor blocks the flow of bile within the extrahepatic biliary tree. A history of gallstones, biliary tract surgery, or malignancy may be elicited.

PREDISPOSING FACTORS

A. Previous blood transfusion.
B. Travel to an area endemic for hepatitis.
C. Raw shellfish consumption.
D. Intravenous (IV) drug abuse.
E. High-risk sexual practices.
F. Family history of episodic jaundice.
G. History of gallstones.
H. Biliary obstruction/previous biliary tract surgery.
I. Alcoholism.
J. Chemical exposure.
K. Sickle cell disease.
L. Pregnancy (intrahepatic cholestasis).
M. Cancer.

COMMON COMPLAINTS

A. Pruritus.
B. Dark, tea-colored urine, from conjugated bilirubinuria.
C. Light, clay-colored stools, from absence of bile.
D. Fatigue.
E. Right upper quadrant (RUQ) pain.

OTHER SIGNS AND SYMPTOMS

A. Enlarged liver.
B. Splenomegaly.
C. Fever.
D. Chills.
E. Gastrointestinal (GI): appetite loss, weight loss, abdominal pain, nausea, or vomiting.
F. Ascites.
G. Shortness of breath.
H. Palpitations.
I. Ecchymosis.
J. Steatorrhea, severe.
K. Asterixis (tremor).
L. Myalgias.
M. Malaise.

SUBJECTIVE DATA

A. Review onset, duration, and course of symptoms.
B. Review medication history for drugs/herbals that may induce jaundice (Table 14.21).
C. Inquire about recent blood transfusions. Are there any known blood disorders in the client's family history?
D. Ask about contact with a person who has an infection, such as infectious hepatitis.
E. Ask about unprotected sexual activity/HIV status.
F. Review ingestion of potentially contaminated food or water, including *Amanita* mushrooms, milk, or shellfish.
G. Ask about the client's exposure to any toxic chemicals, such as carbon tetrachloride, chloroform, phosphorus, arsenic, ethanol, or halothane (Fluothane).
H. Review the client's history of nonsterile needle punctures.
I. Review the client's medical/surgical history for gallstones, hepatitis, tumor, pancreatitis, Wilson disease, Budd–Chiari syndrome, liver surgery, or transplantation. Is there a family history of gallstones?
J. Ask how much alcohol the client has ingested over the years, as well as daily intake of alcohol.
K. Ask about dark urine or white or clay-colored stool.
L. Inquire about dyspepsia, anorexia, nausea, vomiting, RUQ or epigastric pain, or pain radiating to the back or shoulder blade. Ask what the relationship of pain is to eating. ▶

TABLE 14.21 DRUGS AND HERBALS ASSOCIATED WITH JAUNDICE

ACE inhibitors
Acetaminophen (Tylenol)
Alkylated steroids
Aminobenzoic acid
Antibiotics
Antidiabetic drugs
Arsenic
Barbiturates
Chlorpromazine
Ethinyl estradiol/OCPs/hormone replacement
Herbal medications (e.g., Jamaican bush tea)
INH
Mercaptopurine (Purinethol)
Methyldopa (Aldomet)
Monoamine oxidase inhibitors
Paracetamol (acetaminophen)
Perphenazine (Trilafon)
Phenothiazine derivatives
PTU
Probenecid
Rifampin
Sulfonamides
Tamoxifen
TPN

ACE, angiotensin-converting enzyme; INH, isoniazid; OCPs, oral contraceptives; PTU, propylthiouracil; TPN, total parenteral nutrition.

M. Inquire about fever, fatigue, malaise, loss of vigor and strength, easy bruising, and weight loss.
N. Review travel history.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure (BP), and weight. Marked weight loss accompanied by jaundice suggests carcinoma of the head of the pancreas or metastatic disease obstructing the common duct. Note breath odor for fetor hepaticus (breath of the dead).
B. Inspect:
1. Inspect skin, mouth, palms, and sclera for yellow tinge. Severe jaundice may cause greenish tinge from oxidation of bilirubin to biliverdin:
a. In fair-skinned people, discoloration is most evident on the face, trunk, and sclera (sclera icterus).
b. In dark-skinned people, discoloration is most evident in sclera and roof of mouth.
c. Jaundice is most noticeable in natural sunlight. In artificial or poor light, it may be hard to detect.
2. Inspect the skin for spider angiomata, rashes, or scratches from severe itching due to pruritus, and for bruising or petechiae.
3. Inspect the palms for erythema or overt bleeding.
4. Inspect the chest for gynecomastia.
C. Auscultate:
1. Heart.
2. Lungs.
3. Abdomen.
D. Palpate:
1. Abdomen for tenderness, masses, liver enlargement in RUQ, and ascites:
a. Extrahepatic obstruction and intrahepatic cholestasis may be identical in presentation.
b. Tenderness is minimal unless cholangitis or rapid distension occurs.
c. Splenomegaly is unlikely except in primary biliary cirrhosis.
d. Gallbladder may be palpable (Courvoisier sign) when there is gradual development of the biliary tree. Sudden onset of pain results from passage of stone that becomes wedged into common duct; fever and sepsis shortly thereafter indicate cholangitis.
e. Malignancy usually presents as a rock-hard mass.
f. Absence of abdominal pain does not rule out obstruction, especially when it develops slowly from tumor growth or primary biliary cirrhosis.
g. Advanced hepatocellular disease is indicated by a small liver, signs of portal hypertension (ascites, splenomegaly, prominent abdominal venous pattern), asterixis, peripheral edema (from hypoalbuminemia), spider angiomata, gynecomastia, palmar erythema, and testicular atrophy.
E. Percuss:
1. Abdomen.
F. Neurologic exam:
1. Note level of consciousness.
2. Note asterixis, or flapping tremor elicited when arms are extended and wrists are dorsiflexed.
G. Rectal exam:
1. Check for masses and occult blood.

DIAGNOSTIC TESTS

A. The diagnostic approach begins with a careful history and physician examination, and screening laboratory studies. A differential is formulated, and appropriate further testing is performed to narrow the diagnosis.
B. Initial laboratory tests:
1. Serum bilirubin: direct, indirect (unconjugated), and total; clinically, jaundice becomes noticeable when levels reach 2.0 to 2.5 mg/dL:
a. Intrahepatic cholestasis and extrahepatic obstruction: elevated conjugated bilirubin, elevated serum alkaline phosphatase (ALP); mild to moderate rise in transaminase.
b. Gilbert syndrome is the most common cause of decreased uptake and unconjugated hyperbilirubinemia. It is a benign disorder that produces recurrent self-limited episodes of mild jaundice. Typically, the unconjugated fraction rises to no more than 1.5 to 3.0 mg/100 mL. In Gilbert syndrome, fasting and minor illness can precipitate jaundice.
2. ALP: Biliary obstruction or intrahepatic cholestasis is commonly exhibited by increased ALP out of proportion to aminotransferases.
3. Aspartate transaminase (AST)/alanine transaminase (ALT):
a. Elevation of ALT and AST may indicate liver cell damage or may be caused by opiates and aspirin.
b. Aspirin may also decrease AST and ALT.
4. Prothrombin time (PT):
a. PT may be prolonged due to malabsorption, but it is reversible by vitamin K injection.
b. Prolonged PT unresponsive to parenteral vitamin K strongly suggests hepatocellular failure.
c. Cholestasis and obstruction may also produce prolongation of PT but can be reversed by vitamin K.
d. Phenazopyridine (Pyridium) may cause a false-positive bilirubin result.
C. Other laboratory testing as needed:
1. Total serum protein.
2. Serum albumin and globulin:
a. Albumin decreases with cirrhosis and chronic hepatitis.
b. Globulin increases with cirrhosis, chronic obstructive jaundice, and viral hepatitis.
c. To interpret albumin level, consider dietary intake and sources of possible protein loss.
3. Cholesterol.
4. Lactate dehydrogenase (LDH).
5. Gamma-glutamyl transpeptidase (GGT).
6. Serum ammonia.
7. Bile acids radioimmunoassay: Elevated levels indicate hepatic disease.
8. Serologic tests for viral hepatitis.
9. Serum iron, transferrin, and ferritin to evaluate hemochromatosis.
10. Serum ceruloplasmin levels to evaluate Wilson disease.
11. Alpha-1 antitrypsin activity to evaluate alpha-1 antitrypsin deficiency.
12. Urine for bilirubin: Bilirubinuria may be an early sign of liver disease:
a. Collect specimens over a 2-hour period after lunch.
b. Place specimen in a dark brown container and send to lab immediately to prevent decomposition.
D. Abdominal ultrasound is considered the screening procedure of choice. More selective imaging procedures are ordered based on clinical evaluation.
E. Other imaging procedures:

1. Endoscopic ultrasound.
2. Hepatobiliary iminodiacetic acid (HIDA) scan: uses radioactive isotopes.
3. Oral cholecystography.
4. Endoscopic retrograde cholangiopancreatography (ERCP).
5. Percutaneous transhepatic cholangiography (PTC).
6. Helical CT.
7. Magnetic resonance cholangiopancreatography (MRCP) is an alternative to ERCP.
8. Liver biopsy may be indicated for definitive diagnosis.

DIFFERENTIAL DIAGNOSES

A. Jaundice is a symptom, not a diagnosis.
B. Cancer of pancreas.
C. Obstruction of biliary tract.
D. Icteric phase of hepatitis.
E. Cirrhosis.
F. Right-sided heart failure (HF).
G. Chronic hemolysis from prosthetic heart valve.

PLAN

A. General interventions:
1. Management depends on primary diagnosis. Most clients can be managed on an ambulatory basis, unless they are unable to maintain hydration or begin to show evidence of severe hepatocellular failure.
B. Pharmaceutical therapy:
1. Cholestyramine 2 to 8 g orally BID may be used to help relieve itching. Cholestyramine is ineffective with complete biliary obstruction.

FOLLOW-UP

A. See the client 2 to 4 weeks after initial diagnosis and referral.
B. Subsequent routine evaluations are related to primary diagnosis.

EMERGENT ISSUES/INSTRUCTIONS

A. Hospital admission is mandatory when jaundice is complicated by a fever and peritoneal signs are indicative of cholangitis. IV antibiotics and prompt surgical consultation are required.

CONSULTATION/REFERRAL

A. Consult with a gastroenterologist familiar with liver disease and needle biopsy techniques when hepatocellular disease is suspected; when there is evidence of hepatic failure, portal hypertension, or encephalopathy; or when jaundice persists longer than 3 months.
B. Consultation with a gastroenterologist, surgeon, or radiologist experienced in evaluation of jaundice can be very useful when there is clinical suspicion of extrahepatic obstruction.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Viral hepatitis is the most frequent cause of jaundice in pregnancy.
2. Intrahepatic cholestasis in pregnancy is associated with modest maternal risks, including increased risk of peripartum bleeding and increased likelihood of subsequent cholelithiasis.
3. Cholestyramine resin, generally 4 g every 4 to 6 hours, has been reported to afford some relief of pruritus in 50% of affected females, presumably by removing a portion of the bile acids by irreversible binding in the gut.
4. Pregnant clients who take cholestyramine are at increased risk for depletion of vitamin K-dependent coagulation factors and should be followed with serial PT times. Such clients should also receive prophylactic vitamin K supplementation. If PT becomes prolonged, medication should be discontinued.
B. Geriatrics:
1. The most frequent cause of jaundice in clients older than 65 years is *cholelithiasis*.
2. The second most frequent cause of jaundice for clients 65 years and older is cancer, especially of the gallbladder.
3. Painless jaundice in older adults with weight loss and a mass, but with minimal pruritus, suggests biliary obstruction caused by cancer.
4. Drugs that are highly metabolized by cytochrome P450 or similar liver enzymes may result in competitive inhibition of each other, resulting in slow clearance, drug accumulation, and toxicity. Due to polypharmacy, a full review of medications, herbals, and over-the-counter (OTC) should be done for medications (monotherapy or in combination) that are hepatoxic and should be avoided in the geriatric population.

RESOURCES

Drugs.com Drug Interactions Checker: www.drugs.com/drug_interactions.html.
Medscape Drug Interaction Checker: reference.medscape.com/drug-interactionchecker.
RxList Drug Interactions Checker: www.rxlist.com/drug-interaction-checker.htm.

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MALABSORPTION

DEFINITION

A. Malabsorption syndrome is a group of signs and symptoms occurring as a result of digestive problems and absorption of nutrients. There may be a resultant decrease in absorption of fat-soluble vitamins A, D, E, and K. Poor absorption of carbohydrates, minerals, and proteins may also occur.
B. The presentation of malabsorption varies from severe overt symptoms with weight loss to discrete oligosymptomatic changes in hematologic/laboratory tests that are found incidentally. Malabsorption can result from congenital defects or from acquired defects, such as bariatric surgery, and may be either global or partial. The degree of nutrient malabsorption from bariatric surgery is dependent on the type of bariatric procedure.

INCIDENCE

A. Affects millions worldwide, but given the many etiologies it is difficult to determine the exact incidence and prevalence.

PATHOGENESIS

A. Deficiency of intestinal enzymes: lactase deficiency.
B. Inadequate digestion caused by diseases of the pancreas (such as cystic fibrosis [CF]), gallbladder, liver, and biliary systems.
C. Change in bacteria that normally live in the intestinal tract. ▶

D. Disease of intestinal walls, such as helminths (worms) or parasites, tropical sprue, and celiac disease.
E. Surgery that reduces the intestinal tract, decreasing the area for absorption, such as bariatric surgery.
F. Intolerance to gluten, or celiac sprue.
G. Atrophic gastritis results in hypochlorhydria and achlorhydria. The body does not produce enough pepsin and hydrochloric acid to release the food-bound vitamin B_{12} from protein.

PREDISPOSING FACTORS

A. Lactose deficiency:
  1. Incidence is increased in African Americans, Indians, and Asians.
  2. People of Jewish descent typically have onset in adulthood.
B. Family history of malabsorption or CF.
C. Use of drugs, such as mineral oil or other laxatives.
D. Excess alcohol consumption.
E. Travel to foreign countries.
F. Intestinal surgery:
  1. Partial or total gastrectomy.
  2. Small bowel resections (jejunum, ileum, ileocecal).
  3. Partial or total resection of the pancreas.
G. Chronic pancreatitis:
H. Advanced age (increased risk for malabsorption and malnutrition).
I. Long-term adherence to a strict vegetarian or vegan diet (exclusion of all forms of animal protein, including eggs and dairy products).
J. HIV infection.

COMMON COMPLAINTS

A. Diarrhea, stools pale, greasy, and copious.
B. Foul-smelling stools, frequently with mucus.
C. Weight loss despite adequate food intake.
D. Gas or vague abdominal discomfort.
E. Anorexia.

OTHER SIGNS AND SYMPTOMS

A. Early malabsorption:
  1. Minimal weight loss.
  2. Softer, more frequent stools.
  3. Steatorrhea; stools "float" due to increased trapped gas.
  4. Abdominal discomfort.
  5. Bloating.
B. Later signs: symptoms mentioned plus the following:
  1. Marked weight loss.
  2. Foul-smelling, bulky, greasy stools.
C. Malabsorption of fats and carbohydrates: previous symptoms plus the following:
  1. Foul-smelling, bulky, greasy, "sticky" stools that may be difficult to flush down the toilet.
  2. Ecchymosis.
  3. Bone pain.
  4. Glossitis.
  5. Muscle tenderness.
  6. Cramping in lower abdomen after bowel movement (BM).
D. Malabsorption of lactose:
  1. Nausea and bloating.
  2. Cramps.
  3. Diarrhea after ingesting more than customary intake of milk products.
  4. Absent or mild weight loss and steatorrhea.
  5. Good appetite.
E. Edema: with severe protein depletion/calorie malnutrition.
F. Ecchymosis and bleeding disorders secondary to vitamin K malabsorption.
G. Vitamin malabsorption:
  1. Generalized motor weakness (pantothenic acid, vitamin D).
  2. Peripheral neuropathy (thiamine).
  3. Sense of loss for vibration and position (cobalamin).
  4. Night blindness (vitamin A).
  5. Seizures (biotin).

SUBJECTIVE DATA

A. Review onset, duration, and course of symptoms. Are there any other family members with the same history/symptoms?
B. Ask the client about dietary intake history.
C. Review any changes in BMs and stool characteristics.
D. Inquire about recent travel to areas known for giardiasis or other parasites.
E. Document weight loss, how much, and over what period of time.
F. Review previous gastrointestinal (GI) surgery, including bariatric procedures and reversals, small bowel resections, and partial or total resection of the pancreas.
G. Ask the client about signs and symptoms of inflammatory bowel disease (IBD), such as easy bruising, paresthesia, and sore tongue.
H. Ask about any irradiation treatment.

PHYSICAL EXAMINATION

A. Check pulse, respirations, and blood pressure (BP; may have orthostatic hypotension). Check height and weight to calculate body mass index (BMI) in order to follow serial weight loss.
B. Inspect:
  1. Observe general appearance, noting wasting and apathetic appearance.
  2. If the client experiences a BM after the examination, observe the stool for volume, appearance, presence of blood, mucus, and the presence of gross worms/parasites.
  3. Inspect the skin for ecchymosis, jaundice, pallor, surgical scars, stigmata of hyperthyroidism or hepatocellular failure, or signs of Kaposi sarcoma. Alopecia or seborrheic dermatitis may be present.
  4. Inspect the ears, nose, and throat for glossitis, stomatitis, aphthous ulcers, poor dentition, and goiter.
C. Auscultate:
  1. Auscultate the heart for tachycardia.
  2. Auscultate the abdomen in all four quadrants for bowel sounds, noting borborygmi.
D. Percuss:
  1. Percuss abdomen.
E. Palpate:
  1. Palpate all lymph nodes; look for lymphadenopathy.
  2. Palpate the abdomen for organomegaly, focal tenderness, masses, distension, and ascites.
F. Neurologic exam:
  1. Assess for signs of vitamin B_{12} deficiency, including motor weakness, peripheral neuropathy, or ataxia.
G. Rectal exam:
  1. Note tenderness, discharge, blood, and stool.

DIAGNOSTIC TESTS

A. Assessment of stool fat: qualitative assessment on a single specimen.

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B. Quantitative assessment of a 72-hour stool collection while the client is following a 100 g of fat diet.
C. Sudan III stain: performed on a spot stool sample and is sensitive for malabsorption.
D. Increased fecal fat: test for celiac disease.
E. Abdominal ultrasound.
F. Colonoscopy to evaluate and obtain biopsies.
G. Endoscopy to evaluate and obtain biopsies.
H. Barium studies.
I. Consider CT of the abdomen.
J. Endoscopic retrograde cholangiopancreatography (ERCP) helps to document malabsorption due to pancreatic or biliary-related disorders.
K. Breath tests for carbohydrate malabsorption.
L. Complete blood count (CBC) and electrolytes.
M. Serum iron, vitamin B_{12}, and folate concentrations.
N. Prothrombin time (PT): may be prolonged due to vitamin K deficiency.
O. Vitamin levels: Vitamins A, D, E, and K may be decreased.
P. Total protein: may be decreased.
Q. Albumin: may be decreased.
R. Serum amylase.
S. Stool for ova and parasites; obtain on alternate days for three or more specimens because parasites are passed intermittently.
T. Serum immunoglobulin A (IgA): used to rule out IgA deficiency.
U. Three-stage Schilling test (vitamin B_{12} deficiency) identifies the cause of B_{12} deficiency. However, it is rarely used now due to B_{12} and methylmalonic acid availability. The administration of oral or injectable B_{12} is commonly ordered.

DIFFERENTIAL DIAGNOSES

A. Malabsorption.
B. AIDS.
C. Alcoholism.
D. Worms or parasites.
E. CF.
F. Celiac disease.
G. Failure to thrive (FTT).
H. Crohn's disease (CD).
I. Tropical sprue.
J. Side effect from bariatric surgery.
K. Disaccharidase deficiencies (lactase).
L. Fructose intolerance.
M. Lactose intolerance.
N. Milk or protein allergy.
O. Whipple disease.
P. Zollinger–Ellison syndrome.
Q. Chronic pancreatitis.
R. Cirrhosis.

PLAN

A. General interventions:
1. Treatment depends on underlying cause.
B. Client teaching: *See Client Teaching Guide for this chapter, "Lactose Intolerance and Malabsorption."*
C. Dietary management:
1. In most clients, diet modification or dietary supplements restore health.
D. Pharmaceutical therapy:
1. Vitamin supplements: Fat-soluble vitamins A, D, and K are most likely to be depleted. Supplements help prevent malnutrition, even though caloric intake may be replenished:
a. Vitamin A: 25,000 to 50,000 U/d orally.
b. Vitamin D: 30,000 U/d orally.
c. Vitamin K: 4 to 12 mg/d orally.
d. Vitamin B_{12}: 1,000 g/mo by intramuscular (IM) injection.
2. Enzyme replacements: These replace endogenous exocrine pancreatic enzymes and aid in digestion of starches, fat, and proteins. Pancreatic supplements are typically expressed in U.S. Pharmacopeia Convention (USP) units. One international unit (IU) is equivalent to approximately two to three USP units:
a. Pancreatin: Doses vary with the condition being treated. Oral doses are given before or with meals or each snack. They may also be given in divided doses at 1- to 2-hour intervals throughout the day**. Use with extreme caution in clients with a known hypersensitivity to pork products:**
i. Malabsorption syndrome: 8,000 to 24,000 USP units of lipase activity occasionally up to 36,000 units of lipase activity may be required. Considerable dosage variation exists, partly due to the susceptibility of pancreatin to acid–peptic inactivity in the upper GI tract.
ii. Functional indigestion: 1,200 to 2,400 USP of lipase activity.
b. Pancrelipase (Creon, Pancrease, Ultrase, Viokase): This must be given with each meal or snack:
i. Adults: one to three capsules or tablets orally with meals; titrate dose to desired clinical response.
3. Antispasmodics:
a. Anticholinergic agents are used to reduce the cholinergic stimulation of colonic activity that occurs in response to a meal.
b. Drug of choice: dicyclomine hydrochloride (Antispas), 10 to 20 mg orally before meals.
4. Iron and folic acid supplementation are usually required for celiac disease.
5. Calcium and magnesium supplementation are required after extensive small intestinal resection.
6. Antibiotic therapy for bacterial overgrowth.
7. Corticosteroids and anti-inflammatory agents to treat regional enteritis.

FOLLOW-UP

A. If exam is normal, observe the client for 1 month and have the client keep a diary of food intake and weight.
B. For persistent malabsorption, monitor for osteoporosis/bone mass with a dual-energy x-ray absorptiometry (DEXA) scan.

EMERGENT ISSUES/INSTRUCTIONS

A. Any client with weight loss of 15 kg is likely to have a life-threatening condition and requires prompt consultation and hospitalization.

CONSULTATION/REFERRAL

A. Consult a gastroenterologist when malabsorption is documented by 72-hour stool fat assessment.
B. Consider a dietary consultation.
C. Refer back to the bariatric surgeon/center, depending on surgical complications from bariatric surgery.
D. Consider a referral for allergy testing for milk and proteins.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Weight loss/appetite suppression: Weight loss is a predictor of mortality. Clinically significant weight loss is considered to be 5% loss of usual body weight in 3 months or 10% in 6 months

2. Individuals 65 years of age and older are more likely to develop a vitamin B_{12} deficiency because they are at risk for both malabsorption and malnutrition.

3. The frail older adults may have dietary insufficiency due to:

a. Cognitive disabilities.
b. Social isolation, depression, bereavement.
c. Physical impairment/mobility limitations.
d. Poor dentition:
   i. Poor-fitting dentures.
   ii. Edentulous.
e. Poverty.
f. Loss of the sense of taste.
g. Inability to prepare meals.
h. Lack of assistance feeding.
i. Too little time to eat/be fed.

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NAUSEA AND VOMITING

DEFINITION

A. Nausea and vomiting are common symptoms for many conditions and diseases; several terms describe the symptoms (Table 14.22).

1. Hyperemesis gravidarum is a condition of persistent, uncontrollable vomiting that begins in the first weeks of pregnancy and may continue throughout pregnancy.

2. Chronic nausea and vomiting is defined as at least 1 month in duration.

3. Complications of nausea and vomiting include fluid depletion, hypokalemia, and metabolic alkalosis.

4. Vomiting is also considered a protective mechanism to remove harmful ingested substances.

INCIDENCE

A. Nausea and vomiting are very common, and the etiology is dependent on the disease/condition.

B. During pregnancy, 50% to 95% of females experience nausea and/or vomiting; 50% have both nausea and vomiting. Onset after the initial 9 weeks of pregnancy should direct especially careful evaluation for another cause within the differential diagnoses of nausea and vomiting in nonpregnant clients. Hyperemesis gravidarum occurs in 1 in every 0.5% to 2% of pregnancies. Severe hyperemesis requires hospitalization in 0.3% to 2% of pregnancies.

C. Approximately one-third of surgical clients have nausea and/or vomiting after general anesthesia.

D. The incidence of nausea and/or vomiting subsequent to cancer treatment is high.

PATHOGENESIS

A. Protective mechanisms are activated by numerous gastrointestinal (GI) and non-GI causes. Normal function of the upper GI tract involves an interaction between the gut and the central nervous system (CNS).

B. Nausea and vomiting in pregnancy are related to increased hormones, including human chorionic gonadotropin (HCG), estrogen, and progesterone, as well as decreased gastric motility and relative hypoglycemia that result from a night-long fast.

PREDISPOSING FACTORS

A. Acute nausea and vomiting.

1. Medications: digitalis toxicity, opiate use, anesthetic, chemotherapy agents, drug withdrawal, nicotine/nicotine patches, antibiotics, hormones, and antivirals.

2. Ketoacidosis.

3. Pregnancy or hormones.

4. Binge drinking.

5. Hepatitis.

B. Recurrent or chronic nausea and vomiting.

1. Psychogenic vomiting.

2. Metabolic disturbances.

3. Gastric retention.

4. Bile reflux.

5. Pregnancy.

6. Radiation.

7. Gastroparesis.

TABLE 14.22 DEFINITIONS OF TERMINOLOGY USED TO DESCRIBE NAUSEA AND VOMITING

Anorexia	Loss of desire to eat; a true loss of appetite
Early satiety	Fear of being full after eating an unusually small quantity of food
Nausea	The unpleasant sensation of the imminent need to vomit, usually referred to the throat or epigastrium; a sensation that may or may not ultimately lead to vomiting
Regurgitation	The act by which food is brought back into the mouth without the abdominal and diaphragmatic muscular activity that characterizes vomiting
Retching	Spasmodic respiratory movements against a closed glottis with contraction of the abdominal musculature without expulsion of any gastric contents, referred to as "dry heaves"
Rumination	Chewing and swallowing of regurgitated food that has come back into the mouth through voluntary increase in abdominal pressure within minutes of eating or during eating; rumination may be accompanied by weight loss and bulimia
Sitophobia	Fear of eating because due to subsequent or associated discomfort
Vomiting	Forceful oral expulsion of gastric contents associated with contraction of the abdominal and chest wall musculature

C. Nausea and vomiting with abdominal pain.
- **1.** Viral gastroenteritis.
- **2.** Acute gastritis.
- **3.** Food poisoning.
- **4.** Peptic ulcer disease (PUD).
- **5.** Acute pancreatitis.
- **6.** Small bowel obstruction.
- **7.** Acute appendicitis.
- **8.** Acute cholecystitis.
- **9.** Acute cholangitis.
- **10.** Acute pyelonephritis.
- **11.** Inferior myocardial infarction (MI).

D. Nausea and vomiting with neurologic symptoms.
- **1.** Increased intracranial pressure (ICP).
- **2.** Midline cerebellar hemorrhage.
- **3.** Vestibular disturbances.
- **4.** Migraine headaches.
- **5.** Autonomic dysfunction.
- **6.** Head trauma.
- **7.** Multiple sclerosis.

COMMON COMPLAINTS

A. "Queasy" sensation.
B. Food aversion.
C. Inability to retain food or liquids.

OTHER SIGNS AND SYMPTOMS

A. Increased salivation.
B. Bitter taste, "acid brash": indicates ulcer or small bowel obstruction.
C. Weight loss.
D. Dehydration.
E. Sweating.
F. Fast pulse.
G. Pale skin.
H. Rapid breathing.
I. Light-headedness.

SUBJECTIVE DATA

A. Review onset, duration (acute or chronic problem?), and course of symptoms, including the quality (projectile?) and quantity of emesis. What were the color, taste, and consistency of the emesis? Was blood present?
- **1.** Vomiting bright red blood indicates a hemorrhage—*peptic ulcer.*
- **2.** Dark red blood indicates a hemorrhage—*esophageal or gastric varices.*
- **3.** Coffee grounds material is indicative of digested blood from a slowly bleeding gastric ulcer or duodenal ulcer (DU).
- **4.** Vomiting fecal material is a sign of distal *small bowel obstruction* and blind-loop syndrome.
- **5.** Explosive projectile vomiting is associated with increased ICP such as with meningitis.

B. Ask the client about other symptoms, including pain, fever, diarrhea, and headache.
C. Inquire if other family members are also ill and what their symptoms are.
D. Review the timing of vomiting in relation to meals, time of day, odors, and activity. Does vomiting occur before or after food intake?
E. Ask the client about medication intake such as antibiotics, chemotherapy, herbals, digitalis, opiates, and birth control pills.
F. Ask about self-image, binge eating, and self-induced emesis.
G. Review any exposure to hepatitis or travel to places with poor sanitation and outbreaks of cholera.
H. Review the client's medical history for vertigo, head injury, jaundice, diabetes, hypertension, and pregnancy.
I. Inquire about first day of last period and birth control method used.
J. Establish usual weight. Has there been any recent weight change, how many pounds, and over what period of time?
K. Ask about the client's history of diabetes, gallbladder disease, ulcer disease, or cancer.

PHYSICAL EXAMINATION

A. Check temperature (if indicated), pulse (which should be checked both standing and lying down), respirations, blood pressure (BP), and weight.
B. Inspect:
- **1.** Observe general overall appearance of skin for pallor and signs of dehydration. Tenting of skin when it is rolled between your thumb and index finger may indicate dehydration.
- **2.** Inspect for signs of autonomic insufficiency. Postural hypotension, lack of sweat, and blunted pulse and BP responses to Valsalva maneuver suggest autonomic dysfunction and a bowel motility problem as the underlying etiology of nausea and vomiting. Postural hypotension indicates marked volume depletion or circulatory collapse.
- **3.** Oral exam: Inspect mouth, teeth, and gums. Pay particular attention for dental enamel erosion as a sign of bulimia.

C. Palpate:
- **1.** Palpate the abdomen for masses, distension, tenderness, signs of peritonitis, and organomegaly.
- **2.** Palpate the back; note costovertebral angle (CVA) tenderness.

D. Percuss:
- **1.** Percuss the abdomen.

E. Auscultate:
- **1.** Auscultate the abdomen for bowel sounds in all quadrants.
- **2.** Auscultate the heart and lungs.

F. Rectal exam (if indicated).
G. Neurologic examination (if indicated).

DIAGNOSTIC TESTS

A. The cause of an acute episode of nausea and vomiting is typically determined through a detailed history and physical examination. Only if the cause is unclear should further diagnostic tests be performed.
B. Urine: ketones, specific gravity, pregnancy test, culture and sensitivity, if indicated.
C. Serum labs: multiple chemistry profile, including amylase, electrolytes, blood urea nitrogen (BUN), creatinine, glucose, and transaminase.
D. Drug screen.
E. Hepatitis panel.
F. Upper GI.
G. Ultrasonography:
- **1.** Obstetric ultrasound to rule out multiple gestation and/or molar pregnancy if persistent nausea and vomiting, especially after 16 weeks' gestation.
- **2.** Upper abdominal ultrasound, if clinically indicated, to evaluate the pancreas and/or biliary tree.

H. Stool for occult blood.

▶

I. Endoscopy.
J. CT scan.
1. Head CT scan, if indicated.
2. Abdominal CT if appendicitis is suspected.
K. Gastric scintigraphy, to rule out gastroparesis if indicated.
L. EKG if chest pain/MI is suspected.

DIFFERENTIAL DIAGNOSES

A. See "Predisposing Factors."

PLAN

A. General interventions:
1. Assess hydration status. Proceed to intravenous (IV) hydration and antiemetics until ketones clear.
B. Dietary management:
1. See Appendix B, "Dietary Recommendations."
C. Pharmaceutical therapy (not an exhaustive list):
1. Antiemetics:
a. Dextrose/fructose/phosphoric acid solution (Emetrol); available over the counter:
i. Adults: 15 to 30 mL PO at 15-minute intervals (not to exceed five doses)
ii. Tell the client not to dilute the drug or take fluids 15 minutes before or after taking the medication.
iii. Diabetics should be instructed not to use without consulting their provider.
iv. If nausea continues despite medication, contact healthcare provider.
b. Promethazine (Phenergan):
i. Drug of choice for gastroenteritis.
ii. Adults: 25 to 50 mg PO or per rectum every 4 to 6 hours.
c. Trimethobenzamide (Tigan):
i. Used for gastroenteritis and motion sickness.
ii. Adults: 300 mg orally three or four times daily.
iii. Adults: 200 mg IM three or four times daily.
d. Prochlorperazine (Compazine):
i. Adults: 5 to 10 mg three or four times daily; spansules 15 mg Q12H; suppositories 25 mg BID.
2. Outpatient IV hydration in pregnancy: 1 L lactated Ringer solution to correct dehydration and restore electrolyte balance. Check urine ketones after first liter to determine if additional liter is necessary to clear ketones.
3. 5-hydroxytryptamine (5-HT3, or serotonin) antagonists are utilized for nausea related to chemotherapy and postoperative nausea and vomiting. 5-HT3 drugs are available in oral, IV, and transdermal formulations. Consult a prescribing reference for current dosing/formulations:
a. Ondansetron (Zofran).
b. Granisetron (Sancuso and Kytril).
c. Palonosetron (Aloxi).
d. Dolasetron mesylate (Anzemet).
4. Dronabinol (Marinol), cannabinoid 2.5, 5, and 10 mg capsules are indicated for refractory nausea and vomiting associated with chemotherapy. Therapy is individualized before and after chemotherapy.

FOLLOW-UP

A. Tell the client to call or visit the office in 24 hours to check response and nutritional intake.

EMERGENT ISSUES/INSTRUCTIONS

A. First priority is to rule out acute surgical etiology such as bowel obstruction and peritonitis.
B. ***If overdose is suspected, contact 1-800-222-1222, the American Association of Poison Control Centers, the local poison control center, or the ED immediately.***

CONSULTATION/REFERRAL

A. Consult a physician and hospitalize the client promptly if there is evidence of bowel obstruction; increased ICP; or another GI, neurologic, or metabolic emergency.
B. Hyperemesis gravidarum is the most severe form of nausea and vomiting in pregnancy. Hyperemesis gravidarum may require hospitalization to correct fluid and electrolyte imbalance, and nutritional deficiencies.
C. Call for a psychiatric consultation for suspected psychogenic nausea and vomiting.
D. If postural hypotension occurs, especially in older adults, hospital admission for parenteral fluid and electrolyte replacement is indicated.
E. Endocrinologist, gastroenterologist, and surgical consultations for treatment, consideration, and placement of a gastric stimulator for diabetic gastroparesis.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Determine whether the client is ingesting nonfood substances, such as starch, clay, or toothpaste, which would indicate pica.
2. Intractable nausea and vomiting require ultrasonography to rule out hydatidiform mole.
3. Emetrol, promethazine (Phenergan), and prochlorperazine (Compazine) may be used during pregnancy.
4. The only Food and Drug Administration (FDA)-approved drug for treating nausea and vomiting in pregnancy is doxylamine/pyridoxine. Antihistamines, promotility agents (e.g., metoclopramide), Emetrol, promethazine (Phenergan), and prochlorperazine (Compazine) may be used during pregnancy. In cases of refractory to standard therapy, ondansetron and steroids may be considered.
5. Uncorrected hyperemesis gravidarum can result in severe electrolyte imbalance and possible hepatic and renal damage. The major concern for fetal well-being is uncorrected ketosis, which may result in fetal abnormalities or death in early pregnancy.
6. Tell the client to eat toast or a dry cracker; to eat small, frequent meals and snacks; to drink fluids separately from meals; and to avoid fatty, fried, greasy, or spicy foods and foods with strong odors.
B. Geriatrics:
1. There should be a low threshold for hospitalization of older adults to treat dehydration and correction of fluid and electrolyte imbalance.
2. Use of the lower dose ranges of an antiemetic is sufficient for most older adults due to their susceptibility to hypotension and neuromuscular reactions. Dosages should be increased more gradually in older adults.
3. For the older adult with existing nausea and vomiting, nurse practitioners (NPs) should practice special consideration of the following *geriatric syndromes*:
a. Delirium is a common early sign of acute illness or infection in older adults. It is more common in clients with existing cognitive impairment. Dehydration is more common in older adults. Signs of dehydration include confusion, muscle weakness, fever, dizziness, poor skin turgor, hypotension, and tachycardia. ▶

Diminished thirst mechanism and decreased body water exacerbate dehydration.

i. At a minimum, clients and their family should be asked if the client has experienced episodes of altered mental status.

ii. The Brief Confusion Assessment Method (bCAM) is a well-documented assessment for delirium. The bCAM is available at www.mnhospitals.org/Portals/0/Documents/ptsafety/LEAPT%20Delirium/HELP%20Program%20CAM%20Flowsheet.pdf.

b. Weight loss/appetite suppression: Weight loss is a predictor of mortality. Clinically significant weight loss is considered to be 5% loss of usual body weight in 3 months or 10% in 6 months:

i. Due to GI distress, abdominal discomfort, diarrhea, older adults are at increased risk of avoiding eating.

ii. Monitor weight patterns/body mass index (BMI). (Caution: BMI is not used to identify loss of muscle mass.)

iii. Monitoring both serum albumin and anemia: Hhypoalbuminemia and anemia are not necessarily associated with low bodyweight. They can be caused by decreases in liver protein synthesis.

1) Serum albumin concentration is commonly recommended.
2) Hemoglobin, prealbumin, and transferrin.
3) Cholesterol.

c. Falls: Consistently assess for falls. Discuss the risk of dizziness or light-headedness when standing.

i. At the minimum, older adults should be asked how many times they have fallen since their last visit. A reported fall should trigger questions about whether the fall led to injury, a visit to urgent care or hospital ED, or a hospital admission. Safety measures to reduce falls in the home include removing small area carpets, keeping night lights along the path to the bathroom, installing a toilet seat or nearby grab bars. *See Client Teaching Guide for Chapter 3, "Safety Issues: Fall Prevention."*

RESOURCES

American Cancer Society: www.cancer.org
Rome Foundation: http://theromefoundation.org

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PANCREATITIS

DEFINITION

A. Acute pancreatitis is defined as an inflammatory pancreatic condition characterized by local injury, systemic inflammatory response, and possibly organ failure. The most common causes of acute pancreatitis are gallstones and alcohol use.

B. Pancreatitis is subdivided into several categories based on the 2012 Atlanta Classification of acute pancreatitis including:

1. Mild acute pancreatitis:
   a. No organ failure, local or systemic complications.
2. Moderately severe acute pancreatitis:
   a. Organ failure that resolves within 48 hours. AND/OR
   b. Local or systemic complications without persistent organ failure:
      i. Local complications of acute pancreatitis include:
         1) Acute peripancreatic fluid collections.
         2) Pancreatic pseudocysts.
         3) Acute necrotic collections.
         4) Walled-off pancreatic necrosis.
3. Severe acute pancreatitis: persistent organ failure greater than 48 hours.
4. Interstitial edematous acute pancreatitis.
5. Necrotizing acute pancreatitis.
6. Organ failure and systemic complications of acute pancreatitis.

INCIDENCE

A. Pancreatitis is the most common gastroenterologic condition resulting in hospitalization.

B. In the United States, the incidence is 600 to 700 per 100,000 people, with an approximate 1% mortality rate. The peak age of acute pancreatitis is in the fifth and sixth decades; however, mortality increases with age, pancreatitis, organ failure, or pancreatic necrosis.

C. The most common causes are alcohol use and gallstones. However, the incidence is on the rise in part due to hypertriglyceridemia and metabolic syndrome.

PATHOGENESIS

A. Pancreatitis involves localized destruction in the pancreas and systemic inflammatory response. There is also a genetic predisposition for pancreatitis.

B. The inciting event is the premature activation of trypsinogen to trypsin. Normal feedback mechanisms that prevent over accumulation or early release of digestive enzymes are impaired. Accumulation of digestive enzymes creates injury to the pancreas.

C. Impairment can occur at any point along the pathway of digestive enzymes, from injury of the acinar cells that produce digestive enzymes, to early release of enzymes from their transport mechanism, to problems involving the release along the brush border of the duodenum.

D. Once injury occurs, the inflammatory response ensues and causes increased vascular permeability of pancreas, leading to hemorrhage and edema. Necrosis of the tissue can then occur.

E. Mediators of this event are released into the bloodstream, leading to bacteremia, acute respiratory distress syndrome, pleural effusion, gastrointestinal (GI) hemorrhage, and renal failure.

F. Immune-mediated toxicity is thought to be the cause of amino salicylates and sulfonamides, direct toxicity with diuretics, and accumulation of toxic metabolites with valproate and pentamidine.

G. Drug-induced; common drugs include azathioprine, erythromycin, simvastatin, itraconazole, lamivudine, olanzapine, and sulfasalazine.

PREDISPOSING FACTORS

A. Alcohol use.
B. Gallstones.

C. The mnemonic **IT HURTS BADLE** can be used to identify other predisposing factors:
- **1. I**nfectious (bacterial, viral, and parasitic).
- **2. T**umor.
- **3. H**ypercalcemia and hypertriglyceridemia.
- **4. U**lcer (penetrating).
- **5. R**enal failure.
- **6. T**rauma.
- **7. S**tricture.
- **8. B**iliary tract obstruction.
- **9. A**lcohol or anatomic abnormality.
- **10. D**rugs.
- **11. L**ipids: hypertriglyceridemia.
- **12. E**ndoscopic retrograde cholangiopancreatography (ERCP; postprocedure).

COMMON COMPLAINTS

A. Cardinal symptoms:
- **1.** Characteristic abdominal pain located in the epigastrium or left upper quadrant (LUQ), acute onset and steadily progressive; described as steady, boring and dull; radiating into the back or flank, occurring with nausea and anorexia.

OTHER SIGNS AND SYMPTOMS

A. Fever.
B. Dehydration.
C. Tachycardia/tachypnea.
D. Abdominal bloating and distention.
E. Diarrhea.
F. Fatigue.
G. Jaundice.

SUBJECTIVE DATA

A. Elicit information regarding onset, duration, location and characteristics of pain, aggravating and alleviating symptoms, radiation of the pain, and severity of symptoms.
B. Obtain the client's medical history with thorough review of systems.
C. Review all of the client's prescription and over-the-counter (OTC) medications and herbals.
D. Inquire regarding the client's social habits:
- **1.** Alcohol use (how much, how often, over what length of time).
- **2.** Smoking (how much, how often, what length of time).
- **3.** Assess hobbies and occupation to assess risk of toxin exposure.
- **4.** Inquire regarding surgeries and recent procedures, especially an ERCP.

E. Inquire about familial pancreatitis.
F. Review bowel habits and note changes: constipation, diarrhea, anorexia, food intolerance, nausea, vomiting, or bloating.

PHYSICAL EXAMINATION

Evaluate for a "surgical abdomen," defined as a rapidly worsening prognosis in the absence of surgical intervention. Once a surgical abdomen has been excluded, the remainder of the evaluation will be guided by the cause, chronicity of symptoms, along with the location of pain.

A. Check temperature, blood pressure (BP; hypotension in severe cases), pulse (tachycardia), respirations (tachypnea), and height and weight to calculate body mass index (BMI). Notice any weight loss.
B. Inspect:
- **1.** Observe overall appearance: facial expressions, demeanor with walking, refusal to move/writhing; note grimace during exam.
- **2.** Assess for signs of dehydration, including confusion, muscle weakness, fever, dizziness, poor skin turgor, dry mucous membranes, hypotension, and tachycardia.
- **3.** Observe for jaundice and skin changes:
  - a. Sclera icterus.
  - b. Skin:
    - i. Ecchymosis due to hemorrhage:
      - 1) Grey–Turner sign: back/flank.
      - 2) Cullen sign: abdominal/periumbilical.
    - ii. Xanthomas: hypertriglyceridemia.

C. Auscultate:
- **1.** Heart.
- **2.** Lungs:
  - a. Rales may be present if the client has atelectasis due to shallow respirations or pneumonia.
  - b. Decreased breath sounds due to pleural effusions.
- **3.** Abdomen: Assess for bowel sounds in all four quadrants. Diminished bowel sounds; possibly tinkling bowel sounds due to distention.

D. Palpate:
- **1.** Neck: parotid mass if pancreatitis is secondary to mumps infection.
- **2.** Abdomen examination:
  - a. Classic: tenderness in the epigastrium.
  - b. Palpate for hepatomegaly and splenomegaly, rebound tenderness, and peritoneal signs.
  - c. Older adults may lack classic peritoneal signs of rebound and guarding.

E. Percuss:
- **1.** Abdomen for hepatomegaly or splenomegaly. Tympany may be present with a distended abdomen.

F. Neurologic exam
- **1.** Assess for mental status changes which could be indicative of encephalopathy, impending coma, or shock.

DIAGNOSTIC TESTS

A. Laboratory:
- **1.** Serum amylase and lipase: in all clients suspected of acute pancreatitis. A threefold elevation of serum lipase or amylase is identified by the American College of Gastroenterology as one of the three clinical features necessary for diagnosis, along with classic pain consistent with acute pancreatitis and abdominal imaging consistent with acute pancreatitis (ultrasound, CT, or MRI):
  - a. Lipase has a higher diagnostic accuracy compared with amylase as the serum lipase levels are elevated for a longer period of time.
  - b. Amylase for clients with hypertriglyceridemia may be falsely low.
- **2.** C-reactive protein (CRP) on admission and daily for the first 72 hours after admission.
- **3.** Complete blood count (CBC).
- **4.** Comprehensive metabolic profile (CMP).
- **5.** Liver function tests: alanine transaminase (ALT), aspartate transaminase (AST), lactate dehydrogenase (LDH), triglycerides.
- **6.** Blood urea nitrogen (BUN) and creatinine.
- **7.** Arterial blood gases.

B. EKG: Assess for acute coronary syndrome.
C. Imaging:
- **1.** X-ray:

a. Chest: for pneumonia or pleural effusion.
b. Abdominal plain films: to assess for free air.

2. Ultrasound should be performed in all clients at baseline to assess the biliary tract to determine if there is presence of gallstones and/or a stone in the common bile duct (CBT).

3. CT abdomen with contrast:
a. Selective use when there is a broad differential diagnosis that includes acute pancreatitis or suspected local complications suggested on ultrasound.
b. Assessment of local complications 48 to 72 hours after the onset of symptoms.
c. Contraindications to CT with contrast include renal dysfunction and allergy to the contrast medium.

4. Magnetic resonance cholangiopancreatography (MRCP): reserved for severe cases with an elevation of liver enzymes and CBD is not visualized adequately or is found to be normal on ultrasound.

5. ERCP: within 24 to 72 hours in clients with acute gallstone pancreatitis associated with bile duct obstruction or cholangitis.

DIFFERENTIAL DIAGNOSES

A. Pancreatitis.
B. Causes of acute abdomen:
1. Cholecystitis.
2. Appendicitis.
3. Ectopic pregnancy.
4. Ischemic bowel.
5. Small or large bowel obstruction.
6. Perforated viscus.
C. Infectious origin:
1. Abscess.
2. Pyelonephritis.
3. Viral: HIV, cytomegalovirus (CMV), Epstein–Barr, mumps, Coxsackie.
4. Bacterial: *Campylobacter jejuni, Legionella, Mycobacterium tuberculosis, Mycoplasma, Shigella.*
5. Parasitic: *Ascaris lumbricoides*, Cryptosporidium, Microsporidia.
D. GI pathology:
1. Esophagitis.
2. Esophageal rupture.
3. Peptic ulcer disease (PUD).
4. Bowel perforation.
5. Bowel obstruction.
E. Cardiovascular pathology:
1. Acute coronary syndrome.
2. Aortic dissection.
F. Hepatic pathology: hepatitis (all types).
G. Renal pathology: nephrolithiasis.
H. Pancreatic cancer (increases at age 40 years or older).

PLAN

A. General interventions:
1. Acute pancreatitis management is dependent not only on the etiology of the illness but more importantly on the severity. Acute pancreatitis can range from mild and self-limited to life-threatening.
2. Clients diagnosed with acute pancreatitis are managed inpatient. For severe cases, defined by organ failure, ICU admission is necessary.
3. Once the diagnosis is confirmed, rapid assessment of hydration status is warranted. Fluid resuscitation is critical within the first 24 hours to prevent complications.
4. Risk assessment should be performed to determine the severity of pancreatitis; results guide further management. There are several risk assessment tools available:
a. Calculation of an Acute Physiologic Assessment and Chronic Health Evaluation (APACHE) II score on admission and daily for the first 72 hours after admission. An APACHE II calculator is located at www.globalrph.com/apacheii.htm.
b. Ranson's Criteria in Acute Pancreatitis-Prognostic Implication located at www.globalrph.com/ransons.htm.
5. For mild pancreatitis, fluid resuscitation, pain management, and supportive care are recommended. Generally, mild pancreatitis is self-limited.
6. Although lipase and amylase measurement can assist in diagnosing pancreatitis, following the levels during treatment is not recommended.
B. Pharmaceutical therapy:
1. Antibiotics are not indicated unless an extrapancreatic infection exists.
C. Diet:
1. Enforcing a strict NPO diet is unnecessary for a client with mild acute pancreatitis. Once abdominal pain, nausea, and vomiting resolve, they may begin a low-fat diet as tolerated.

FOLLOW-UP

A. Reevaluate in the office posthospitalization: For mild cases, following with the primary care provider is recommended. However, for moderately severe or severe cases, follow-up with the specialist is recommended.
B. Monitor for increasing pain, anorexia, recurrence of nausea and vomiting, dyspnea or tachycardia, fever, weight loss, or jaundice.
C. Lab follow-up: It is not recommended to repeat amylase and lipase. Lab follow-up is dependent on the etiology of pancreatitis. For instance, following serum transaminases, alkaline phosphatase, bilirubin if biliary obstruction is the cause, or following a lipid profile if due to hypertriglyceridemia.
D. Follow up with specialist (e.g., surgeon, hepatologist, gastroenterologist, oncologist, infectious disease specialist) as directed.
E. For clients who developed pancreatitis from alcohol abuse, continued counseling and referral into an alcohol treatment program are indicated. Clients should be counseled at follow-up visits regarding the importance of continual abstinence from alcohol to prevent the development of chronic pancreatitis.
F. Clients who smoke should be counseled on cessation because smoking is an independent risk factor for development of pancreatitis.
G. Long-term management of acute pancreatitis involves eliminating the causative factor to prevent recurrence.
H. Recurrence rate of biliary pancreatitis is 60% and usually occurs within 6 weeks. Therefore, cholecystectomy at time of hospitalization or within 4 weeks of discharge is recommended.

CONSULTATION/REFERRAL

A. Dependent on etiology.
B. Endocrinology: hypercalcemia, hypertriglyceridemia, or resulting pancreatic insufficiency.
C. Rheumatology: autoimmune pancreatitis.
D. Gastroenterology/surgery: abscess, hemorrhage, pseudocyst, cholecystectomy.
E. Infectious disease: underlying infection.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Delirium is a common early sign of acute illness or infection in older adults. It is more common in clients with existing cognitive impairment. At a minimum, clients and their family should be asked if the client has experienced episodes of altered mental status:

a. The Brief Confusion Assessment Method (bCAM) is a well-documented assessment for delirium. The bCAM is available at www.mnhospitals.org/Portals/0/Documents/ptsafety/LEAPT%20Delirium/HELP%20Program%20CAM%20Flowsheet.pdf.

2. Older adults have a diminished sensorium, allowing pathology to advance to a dangerous point prior to symptom development:

a. The level of pain is much less severe at presentation and continues to be at a lower level of pain.

b. Older adults may present with altered mental status. Cognitive impairment can make assessment and diagnosis of pain more difficult. For dementia or noncommunicative clients, the American Medical Directors Association has endorsed the Pain Assessment in Advanced Dementia (PAINAD) Scale, which scores the following items:

i. Breathing: Examples include normal, labored, hyperventilation.

ii. Negative vocalization: Examples include moaning, negative language, crying.

iii. Facial expression: Examples include smiling, frowning, grimacing.

iv. Body language: Examples include relaxed, tense, rigid, or striking out.

v. Consolability: Examples include extent to which a caregiver is able to console, distract, or reassure.

RESOURCES

American Gastroenterological Association https://gastro.org

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PEPTIC ULCER DISEASE

DEFINITION

A. Peptic ulcer disease (PUD) is circumscribed ulceration of the gastrointestinal (GI) mucosa occurring in areas exposed to acid and pepsin. The client's ulcer history tends to predict future behavior and risk of future complications. Complications include bleeding ulcer, perforation, and obstruction.

B. The stomach is divided on the basis of its physiologic functions into two main portions. The proximal two-thirds, the fundic gland area, acts as a receptacle for ingested food and secrete acid and pepsin. The distal third, the pyloric gland area, mixes and propels food into the duodenum and produces the hormone gastrin. "Peptic" lesions may occur in the esophagus (esophagitis), stomach (gastritis), or duodenum (duodenitis).

C. There is often no correlation between the presence of an active ulcer, noted by endoscopy, and symptoms. The disappearance of symptoms does not guarantee ulcer healing.

INCIDENCE

A. The annual incidence of peptic ulcer is estimated to range from 0.1% to 1.8%. The ulcer incidence in *Helicobacter pylori*-infected individuals is about 1% per year. The recurrence rate is 50% to 80% during the 6 to 12 months following the initial ulcer healing, although relapses are not always symptomatic. Some peptic ulcers heal spontaneously, and 2% to 20% of clients have multiple simultaneous ulcers.

B. From 16% to 31% of ulcers are caused by nonsteroidal anti-inflammatory drugs (NSAIDs). Epidemiologic studies show that risks of peptic ulcer and death are three to six times higher among people who take NSAIDs.

C. Cigarette smokers are twice as likely to develop ulcers as nonsmokers.

D. Increased prevalence in males and people with chronic conditions.

PATHOGENESIS

A. Although the precise mechanisms of ulcer formation remain incompletely understood, the process appears to involve the interplay of acid production, pepsin secretion, bacterial infection, and mucosal defense mechanisms. Excess acid production is the hallmark of duodenal ulcer (DU) disease. Pepsin secretion is also elevated in DU disease.

B. The relation of aspirin and other NSAIDs to ulcer disease is due largely to the drugs' potent inhibition of gastric mucosal prostaglandin synthesis. In addition to prostaglandin inhibition, many NSAID preparations produce acute diffuse mucosal injury by means of a direct erosive effect.

PREDISPOSING FACTORS

A. *H. pylori* infection is the most common cause of ulceration.

B. Use of NSAIDs, especially aspirin, ibuprofen, and naproxen, is associated with acute erosive gastritis.

C. Smoking.

D. Genetic factors: family history of ulcer disease.

E. Age:

1. DU occurs between ages 25 and 75.

2. Gastric ulcer (GU) occurs between ages 55 and 65.

F. Sex:

1. The male-to-female ratio for gastritis is 1:1.

2. The male-to-female ratio for peptic ulcers is 2:1.

G. Excessive alcohol consumption, which stimulates acid secretion.

H. Medications:

1. Corticosteroids potentiate ulcer risk in clients who use NSAIDs concurrently.

2. Anticoagulants, such as warfarin.

3. Bisphosphonates.

4. Spironolactone.

5. Selective serotonin reuptake inhibitors (SSRIs).

I. Improper diet, irregular meals, and skipped meals.

J. Severe physiologic stress:

1. Burns.

2. Central nervous system (CNS) trauma.

3. Surgery.

4. Severe medical illness:
   a. Cirrhosis.
   b. Chronic obstructive pulmonary disease (COPD).
   c. Renal failure.
   d. Crohn's disease.

K. Other causes:
1. Radiation-induced ulcer.
2. Chemotherapy-induced ulcer.
3. Vascular insufficiency.
4. Duodenal obstruction.

L. Zollinger–Ellison syndrome.
M. Bile reflux.
N. Illicit drugs: crack cocaine.

COMMON COMPLAINTS

A. Perforated peptic ulcer presents with a sudden severe onset of sharp abdominal pain.
B. Pain described as aching, boring, gnawing, or burning feeling.
C. Epigastric pain in right upper quadrant (RUQ) and left upper quadrant (LUQ) of the abdomen or occasionally below breast.
D. Pain that awakens the client at night or in early morning.

OTHER SIGNS AND SYMPTOMS

A. Asymptomatic.
B. GI distress 1 to 3 hours after a meal, on an empty stomach.
C. Pain relieved by food, antacids, or vomiting.
D. Nausea and vomiting.
E. Hematemesis.
F. Chest discomfort.
G. Blood in stools, "grape jelly" or maroon-colored stools.
H. Loss of appetite or weight.
I. Weight gain; those with DU may eat more to ease pain.
J. Anemia.

OTHER SIGNS AND SYMPTOMS

A. Massive bleeding:
1. Acute, bright red hematemesis or large amount of melena with clots in the stool, or "grape jelly" stool.
2. Rapid pulse, drop in blood pressure, hypovolemia, and shock.

B. Subacute bleeding:
1. Intermittent melena or coffee-ground emesis.
2. Hypotension.
3. Weakness and dizziness.

C. Chronic bleeding:
1. Intermittent appearance of blood.
2. Increased weakness, paleness, or shortness of breath.
3. Occult blood.

SUBJECTIVE DATA

A. Ask the client to describe onset, duration, type, and location of pain. Does it occur at any special time, for example, before meals, after meals, or during the night?
B. Has the client had a previous ulcer? What was the treatment; if oral treatment was prescribed, did the client complete the therapy?
C. Have the client describe what alleviates pain, such as taking antacids, and what worsens pain, such as use of aspirin, oral steroids, or NSAIDs.
D. Review associated symptoms, such as nausea, vomiting, and heartburn.
E. Ask the client if any first-degree relatives have ulcers.
F. Inquire whether the client is a smoker. If so, how much and for how long?
G. Ask the client about alcohol consumption: How much and for how long?
H. Inquire whether any blood has ever been vomited or passed in stool. If so, have the client describe it.
I. Take the client's dietary history, including time of meals, frequency of skipped meals, weight loss, and so forth.
J. Review all medications, including a review of over-the-counter (OTC) and herbal products such as ginkgo biloba; special attention should be taken to inquire about aspirin and NSAIDs.
K. Obtain medical history of associated diseases, such as cirrhosis, pancreatitis, arthritis, COPD, and hyperparathyroidism.
L. If the client suspects blood in stool, ask if there has been a change in bowel pattern, presence of abdominal pain or tenderness, and what kind of food, such as red beets, the client recently ingested.

PHYSICAL EXAMINATION

A. Check temperature (if indicated), pulse, respirations, blood pressure, and weight.
B. Inspect:
1. Overall appearance of client.

C. Auscultate:
1. Abdomen for bowel sounds.

D. Percuss:
1. Abdomen for hepatosplenomegaly.

E. Palpate:
1. Abdomen for tenderness, rigidity, masses, and liver or spleen enlargement.

F. Rectal exam:
1. Check for tenderness and masses.
2. Take stool specimen.

DIAGNOSTIC TESTS

A. Complete blood count (CBC).
B. Stool for occult blood.
C. Coagulation studies.
D. Testing for *H. pylori:*
1. **Endoscopy with biopsy is the most accurate test.**
2. Rapid urease test is the diagnostic test of choice.
3. Urea breath test (UBT).
4. Serum test for *H. pylori* antibodies.
5. Stool *H. pylori* antigen testing: more accurate than antibody testing and less expensive.

E. Radiography with barium meal.
F. Mucosal biopsy, after GI consultation, to rule out cancer.
G. Fasting gastrin level (screening for Zollinger–Ellison syndrome).
H. Proton pump inhibitors (PPIs) are associated with osteopenia/osteoporosis; depending on the length of therapy, consider a dual-energy x-ray absorptiometry (DEXA) scan.

DIFFERENTIAL DIAGNOSES

A. PUD.
B. Gastroesophageal reflux disease (GERD).
C. Zollinger–Ellison syndrome: Fasting serum gastrin level 500 pg/mL in the presence of acid hypersecretion is diagnostic.
D. Cancer:
1. Gastric lymphoma.
2. Gastric cancer.
3. Pancreatic cancer.

E. Pancreatitis (acute or chronic).

▶

F. Myocardial ischemia.
G. Abdominal aneurysm.
H. Diverticulitis.
I. Drug-induced dyspepsia:
1. Theophylline.
2. Digitalis.
J. Crohn's disease (CD) involving the stomach or duodenum.
K. Gastric infections.
L. Cholelithiasis.

PLAN

A. General interventions:
1. Goals are to alleviate pain, promote healing, limit complications, and prevent recurrences while minimizing the costs and side effects of treatment:
B. Client teaching: *See Client Teaching Guide for this chapter, "Management of Ulcers."*
1. Encourage the client to stop taking NSAIDs, unless medically indicated:
a. If NSAID use is unavoidable, the lowest possible dose and duration and cotherapy with a PPI based on triple therapy are recommended.
2. Smoking cessation should be highly encouraged at each visit.
C. Dietary management:
1. Advise the client to avoid alcohol; coffee, including decaffeinated; and other caffeine-containing beverages because they stimulate acid secretion (see section "Bland Diet" in Appendix B).
D. Medical and surgical management:
1. Diagnostic evaluation of the ulcer is by means of endoscopy.
2. Test for *H. pylori* (carbon isotope-UBT, blood test for antibodies to *H. pylori*, stool test, or gastric biopsy at the time of esophagogastroduodenoscopy [EGD]):
a. A single negative *H. pylori* test should be interpreted cautiously, especially in the face of active bleeding. Blood in the stomach can alter the pH indicator in the rapid urease test. False-negatives are likely, and additional testing for *H. pylori* is essential.
b. Concurrent use of a PPI, antibiotics, or bismuth will cause a false-negative test.
3. Surgery remains an option for treatment of refractory disease and complications. The most serious indications for surgery include brisk bleeding of 6 to 8 units of blood in 24 hours, recurrent bleeding episodes, perforation, gastric outlet obstruction refractory to medical therapy, and failure of a benign GU to heal after 15 weeks. Emergency intervention may be required, such as withholding food and oral fluids, starting an intravenous (IV), placing a nasogastric (NG) tube, providing oxygen therapy, or performing a blood transfusion. If life-threatening bleeding occurs, treat shock.
E. Pharmaceutical therapy:
1. The treatment of a peptic ulcer begins with the eradication of *H. pylori* in infected individuals. Empiric therapy for the infection is reasonable for uncomplicated cases in the absence of NSAID use. Documenting infection, even in clients with known ulcers, is an essential step prior to initiating antimicrobial therapy:
a. Triple therapy anti-*H. pylori* regimen PPI + amoxicillin + clarithromycin:
i. PPI-based regimen (choose one):
1) Rabeprazole (Aciphex) 20 mg BID; total treatment duration 7 days.
2) Esomeprazole (Nexium) 40 mg once a day; total treatment duration 10 days.
3) Lansoprazole (Prevacid) 30 mg BID; total treatment duration 14 days.
ii. Amoxicillin 1,000 mg BID.
iii. Clarithromycin 500 mg BID.
b. Quadruple therapy–Helidac therapy (sold in a combination pack), oral doses QID for 14 days:
i. Choose standard-dose PPI, or ranitidine 150 mg BID x 14 days.
ii. Metronidazole (Flagyl) 250 mg QID x 14 days.
iii. Tetracycline 500 mg QID x 14 days.
iv. Bismuth subsalicylate 525 mg (two chewable tablets) QID x 14 days.
2. Antisecretory therapy is the mainstay of therapy in uninfected clients and is used for maintenance therapy in selected cases. Full doses of H2 receptor antagonist provide effective initial therapy; however, PPIs are more effective.
3. Clients with uncomplicated, small (<1 cm) DU or GU who have received adequate treatment for *H. pylori* probably are asymptomatic and do not need any further therapy directed at ulcer healing. Maintenance acid suppression with a PPI for 1 year following *H. pyloric* eradication is recommended for clients with a complicated DU. If the ulcer is giant (>2 cm), showing densely fibrosed ulcer beds, or if the client is a high-risk client with a protracted history, then the client should be kept on a PPI until a follow-up endoscopy is performed:
a. H2 receptor antagonist-based therapy: Split dose, evening, and nighttime therapy are all effective. In the United States, cimetidine, ranitidine, and famotidine are approved for GU healing. H2 receptor antagonist-based regimens are not U.S. Food and Drug Administration (FDA)-approved treatment regimens. Choose one antibiotic combination (amoxicillin + clarithromycin OR metronidazole + tetracycline as noted above) for 2 weeks and add one of the following:
i. Cimetidine (Tagamet) 400 mg orally BID or 800 mg at bedtime for 6 to 8 weeks.
ii. Ranitidine (Zantac) 150 mg BID or 300 mg at bedtime for 6 to 8 weeks.
iii. Famotidine (Pepcid) 20 mg orally BID or 40 mg at bedtime for 6 to 8 weeks.
iv. Nizatidine 150 mg orally BID or 300 mg at bedtime for 6 to 8 weeks.
b. PPIs are effective in inducing ulcer healing. The PPIs are the most potent inhibitors of gastric acid secretion. Once-daily dosing is generally sufficient for acid inhibition; however, a second dose may be necessary and should be given before the evening meal. Once-daily PPI dosing inhibits acid output by 66% after 5 days. Optimal dosing is immediately before breakfast:
i. Omeprazole (Prilosec/Zegerid) 20 to 40 mg every morning for 4 weeks.
ii. Esomeprazole (Nexium) 20 to 40 mg every morning for 4 to 8 weeks.
iii. Lansoprazole (Prevacid) 15 to 30 mg every morning for 4 weeks.
iv. Dexlansoprazole (Kapidex) 30 to 60 mg every morning for 4 to 8 weeks.

v. Pantoprazole (Protonix) 20 to 40 mg every morning for 4 weeks.
vi. Rabeprazole (Aciphex) 20 mg every morning for 4 weeks.
vii. Dexlansoprazole (Dexilant) 60 mg every morning for 8 weeks.

c. PPIs should not be given concomitantly with prostaglandin, or other antisecretory agents due to the marked reduction in their effects. An H2 antagonist can be used with a PPI if given after a sufficient interval between their administrations; the minimal times have not been established. The H2 antagonist could be given at bedtime for breakthrough symptoms after a morning dose of a PPI.

d. Combination products currently on the market:
i. Omeclamox-Pak: omeprazole 20 mg (two caps), amoxicillin 500 mg (four caps), and clarithromycin 500 mg (two tabs) per Pak. Take omeprazole 20 mg, amoxicillin 100 mg, and clarithromycin 500 mg BID. Treatment is 10 days.
ii. Prevpac: lansoprazole 30 mg (two caps), amoxicillin 500 mg (four caps), and clarithromycin 500 mg (two caps) per Pak. Take lansoprazole 30 mg, amoxicillin 1,000 mg, and clarithromycin 500 mg BID Treatment is 10 or 14 days.

4. Sucralfate (Carafate) 1 g orally QID, 1 hour before meals and at bedtime; maintenance therapy is 1 g BID. Sucralfate is not recommended for *H. pylori* or NSAID ulcers.

5. Misoprostol (Cytotec, a prostaglandin analogue) is effective for peptic ulcers due to NSAID use; peptic ulcers respond well to misoprostol 100 to 200 mcg orally QID.

FOLLOW-UP

A. Therapeutic trial of lifestyle changes combined with an H2 receptor antagonist, sucralfate, or omeprazole for 1 to 2 weeks should provide relief. Reevaluate the client after 2 weeks, and if symptoms are improved prescribe a full course of 6 to 8 weeks.

B. Reevaluate the client again at 8 weeks, after the full course of therapy is completed.

C. Consider repeating breath test to confirm eradication of *H. pylori.*

D. Some authorities advocate routine endoscopic or radiologic documentation of healing. However, no studies show this to be cost-effective in uncomplicated cases in which symptoms resolve within 4 to 6 weeks and do not recur.

E. For refractory GU—that is, persistent pain after 8 weeks despite a full medical regimen or unresponsive to treatment for *H. pylori*—endoscopic examination and biopsy are needed, especially in clients older than age 40 who are at increased risk of gastric cancer. Barium study is not sufficient because even malignant ulcers may shrink in size in response to therapy.

F. Evaluate refractory cases for Zollinger–Ellison syndrome, especially when there are multiple ulcers, occurrences in unusual places, marked abdominal pain, or a secretory diarrhea.

G. There is an increased risk of osteoporotic fracture from the long-term use of PPIs.

H. Clients beginning long-term NSAID therapy should first be tested for *H. pylori*.

EMERGENT ISSUES/INSTRUCTIONS

A. Admit the client to a hospital when symptoms of hemorrhage, penetration, perforation, or gastric outlet obstruction are present.

CONSULTATION/REFERRAL

A. Consult both a surgeon and a gastroenterologist.

B. Refer clients with recurrences or refractory disease to a physician for evaluation for *H. pylori* infection by endoscopy or breath test. If present, eradicate with a 2-week course of triple therapy.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. The drugs misoprostol and ranitidine are abortifacients. They cross the placental barrier and are excreted in breast milk and are contraindicated in pregnancy or suspected pregnancy. Use in sexually active clients of childbearing age should be done with proper warning and detailed client education.

B. Adults:

1. DUs are more common in people between ages 45 and 54; GUs, between 55 and 64 years.

2. Clients older than age 40 are at greater risk of gastric cancer and should undergo either an upper GI series or endoscopy to document the nature and location of lesions when there is strong clinical suspicion of ulcer disease.

3. Gastritis not associated with reflux is present in 75% of people older than age 50.

C. Geriatrics:

1. Silent disease is particularly common among older adults and those using NSAIDs.

2. Lethargy, confusion, slurred speech, agitation, and visual hallucinations have been reported with cimetidine, particularly in older adults.

3. In older adults, DU symptoms remain classic with early morning awakening to pain, then quick relief by food or antacids. GU symptoms are less obvious, with burning or gnawing pain experienced in <50% of older adults.

4. Anemia may be the only symptom of gastric cancer. Cancer must always be considered and confirmed by endoscopy with biopsy.

5. For the older adult with existing PUD, nurse practitioner (NPs) should practice special consideration of the following *geriatric syndromes*:

a. Delirium is a common early sign of acute illness or infection in older adults. It is more common in clients with existing cognitive impairment. Dehydration is more common in older adults. Signs of dehydration include confusion, muscle weakness, fever, dizziness, poor skin turgor, hypotension, and tachycardia. Diminished thirst mechanism and decreased body water exacerbate dehydration:
i. At a minimum, clients and their family should be asked if the client has experienced episodes of altered mental status:
1) The Brief Confusion Assessment Method (bCAM) is a well-documented assessment for delirium. The bCAM is available at www.mnhospitals.org/Portals/0/Documents/ptsafety/LEAPT%20Delirium/HELP%20Program%20CAM%20Flowsheet.pdf.
ii. Pain: Poorly relieved pain is an important cause of functional impairment at any age group: ▶

1) Older adults have a diminished sensorium, allowing pathology to advance to a dangerous point prior to symptom development.
2) Older adults with abdominal pain may present with altered mental status. Cognitive impairment can make assessment and diagnosis of pain more difficult.
3) For dementia or noncommunicative clients, the American Medical Directors Association has endorsed the Pain Assessment in Advanced Dementia (PAINAD) Scale, which scores the following items:
a) Breathing: Examples include normal, labored, hyperventilation.
b) Negative vocalization: Examples include moaning, negative language, crying.
c) Facial expression: Examples include smiling, frowning, grimacing.
d) Consolability: Examples include extent to which a caregiver is able to console, distract, or reassure.
e) Body language: Examples include relaxed, tense, rigid, or striking out.

b. Falls: Consistently assess for falls. PPIs are associated with osteopenia and osteoporosis and place the client at an increased risk of fractures.
i. At the minimum, older adults should be asked how many times they have fallen since their last visit. A reported fall should trigger questions about whether the fall led to injury, a visit to urgent care or hospital ED, or a hospital admission.
ii. Safety measures to reduce falls in the home include removing small area carpets, keeping night lights along the path to the bathroom, and installing a toilet seat or nearby grab bars.
iii. *See Client Teaching Guide for Chapter 3, "Safety Issues: Fall Prevention."*

6. Beers Criteria caution:
a. Due to polypharmacy, a full review of medications, herbals, and OTC drugs should be undertaken to identify medications that should be avoided in the geriatric population, duplicate medications, and medications that are able to be decreased or deprescribed (refer to deprescribing section in Chapter 3, "Adult-Geriatric Assessments.").
b. When prescribing to older adults, use the lowest dose, for the shortest period of time.
i. Avoid prescribing a long-acting NSAID, such as indomethacin (Indocin) and piroxicam (Feldene).
c. PPI:
i. PPIs are on the American Geriatrics Society 2015 Beers Criteria for potential inappropriate medications use in older adults. (Refer to Appendix C, Table C.1). Avoid scheduled use of PPIs for >8 weeks unless needed for high-risk clients. PPIs are associated with the following:
1) Osteoporosis/osteopenia, prolonged exposure may increase of fractures.
2) Community-acquired pneumonia.
3) *Clostridioides difficile* infections and diarrhea.
4) Vitamin B_{12} deficiency and hypomagnesium.
d. See Appendix D for the PPI deprescribing algorithm from deprescribing.org.
e. Due to other medications they take for comorbid health conditions, older adults are especially at risk with the combination of NSAIDs and the following:
i. Aspirin.
ii. Clopidogrel (Plavix).
iii. Dabigatran (Pradaxa).
iv. Dipyridamole (Persantine).
v. Prasugrel (Effient).
vi. Ticlopidine (Ticlid).
vii. Warfarin (Coumadin).
viii. Corticosteroids.

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POSTBARIATRIC SURGERY LONG-TERM FOLLOW-UP

DEFINITION

A. The number of obese people in the United States has more than doubled in the past 50 years. The traditional management of obesity, a combination of diet, exercise, and behavioral modification, often results in moderate success with limited sustainability. Increasing prevalence of obesity combined with improvements in surgical weight-loss procedures and improved insurance coverage has resulted in exponential growth of the number of people opting for surgical management. Individuals who undergo surgical weight-loss procedures have unique healthcare needs and require lifelong follow-up. Although the bariatric treatment team is the ideal source of follow-up and monitoring, primary care providers can play a critical role and need to be cognizant of the unique needs of this population.

B. The primary mechanism of action for surgical weight-loss procedures is either restriction or a combination of restriction and malabsorption (Table 14.23). All procedures have some form of restriction that reduces the volume of food that can be ingested. Restriction can occur by means of a physical barrier such as a laparoscopic adjustable gastric band (LAGB; Figure 14.4), or an intragastric balloon (Figure 14.7), or by removing a portion of the digestive tract such as with the gastric sleeve (GS; Figure 14.5). Malabsorptive procedures cause weight loss by changing the way nutrients are absorbed, which is accomplished by removing portions of the stomach and/or small intestine and sometimes by rerouting the digestive tract (Roux-en-Y; Figure 14.6). The success of surgical weight loss is defined by initial weight loss, maintenance of weight loss, and prevention of complications. Success is directly related to the aftercare a person receives.

INCIDENCE

A. According to the 2015 to 2016 National Health and Nutrition Examination Survey (NHANES), the prevalence of obesity is 39.8%; among adults aged 40 to 59 years, the prevalence is higher (42.8%). The American Society for Metabolic and Bariatric Surgery (ASMBS) reports an estimated 228,000 weight-loss operations were performed in 2017. About 90% of surgical weight-loss procedures are performed laparoscopically now. The most common procedures performed today include the gastric bypass, GS, adjustable gastric band, and biliopancreatic diversion with duodenal switch. The intragastric balloons were U.S. Food and Drug Administration (FDA) ▶

TABLE 14.23 SURGICAL WEIGHT-LOSS PROCEDURES AND MECHANISM OF ACTION

Procedure	Mechanism of Action	Advantages	Disadvantages
LAGB; Figure 14.4	Restriction	No cutting or rerouting of digestive tract Reversible and adjustable Lowest risk of nutritional deficiencies	Slower (more gradual) weight loss Greater chance of failure to lose 50% of excess weight Foreign device in body; slippage and erosion possible Highest rate of reoperation
GS; Figure 14.5	Restriction and some malabsorption	No rerouting Hormonal hunger suppression, appetite reduction, and increased satiety	Not reversible Potential for nutrient deficiencies Higher complication rate than LAGB
RYGB; Figure 14.6	Restriction and malabsorption	Reduces appetite, enhances satiety Better initial and long-term weight loss compared with LAGB	Long-term nutritional-deficiency risk Lifelong adherence to diet and supplementation required Higher complication rate compared with LAGB and GS
BPD/DS	Malabsorption and some restriction	Greatest weight loss overall Reduces appetite, improves satiety Most effective for treatment of diabetes mellitus	Greatest risk of nutritional deficiencies Strict lifetime adherence to diet and supplements Highest complication rate
Intragastric balloons; Figure 14.7	Restriction	Approved for BMI 30–40 Placed endoscopically performed under conscious sedation or general sedation Can be adjunctive therapy to achieve weight loss reduction prior to bariatric surgery	Designed to remain implanted for 6 months and allow adjustment in fluid volume to address client symptoms and weight loss plateaus

BPD/DS, biliopancreatic diversion with duodenal switch; GS, gastric sleeve; LAGB, laparoscopic adjustable gastric band; RYGB, Roux-en-Y gastric bypass.



FIGURE 14.4 Laparoscopic adjustable gastric band.

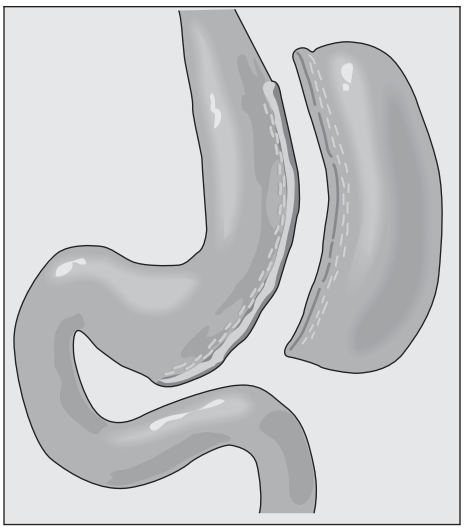


FIGURE 14.5 Sleeve bypass procedure with stomach resection.

approved in 2015; only about 2.75% were done in 2017. The intragastric balloon is a temporary weight loss procedure with placement only for 6 months.

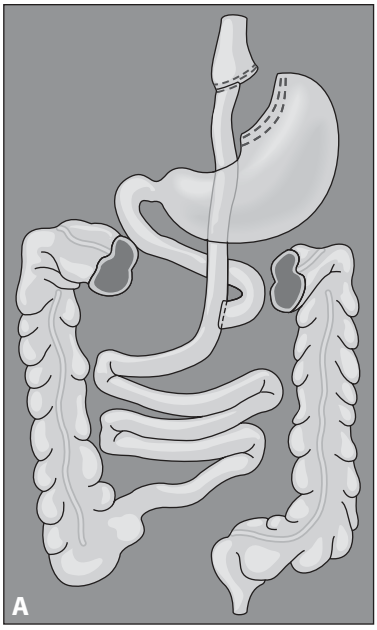
PATHOGENESIS

A. The pathogenesis of obesity is reviewed under "Obesity" (Chapter 24). Significant improvements in the safety of surgical weight-loss procedures in recent years result from improved surgical techniques, accreditation, and the use of laparoscopy. The overall mortality rate is about 0.5%. The incidences of complications vary by surgical procedure. Postoperative complications may occur immediately or may occur many years after surgery. Nutritional deficiencies are by far the most common long-term complication.

PREDISPOSING FACTORS

A. Higher body mass index (BMI).
B. Noncompliance with bariatric diet and exercise.
C. Lack of follow-up with healthcare professionals.
D. Obesity-related health problems:
  1. Sleep apnea.
  2. Diabetes.
  3. Arthritis.

▶



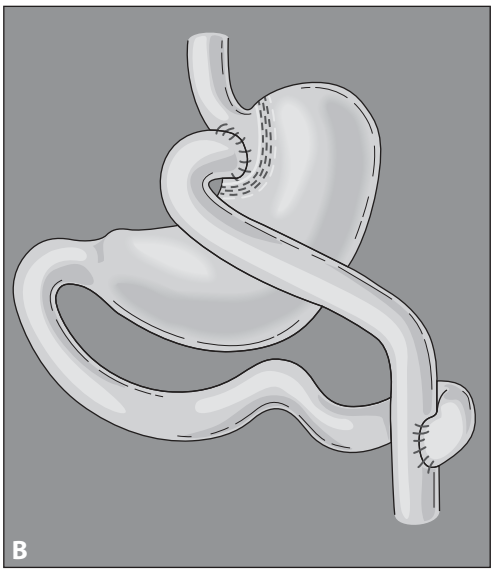


FIGURE 14.6 Roux-en-Y gastric bypass. (**A**) Malabsorption procedure to assist in weight loss: Example of portions of the stomach and/or small intestine removed.(**B**) Malabsorption procedure: Rerouting of the digestive tract.

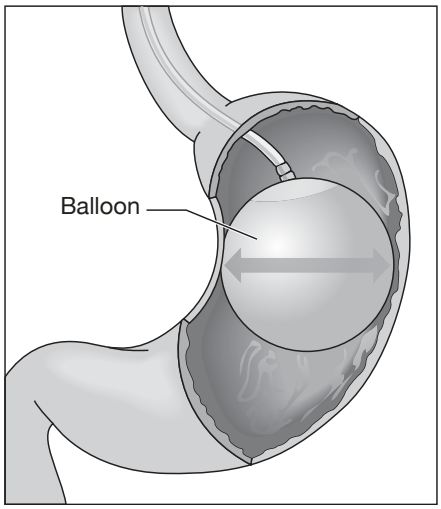


FIGURE 14.7 Intragastric balloon.

4. Hypertension.
5. Gastroesophageal reflux disease (GERD).

E. Complexity and type of surgery.

COMMON COMPLAINTS

A. Functional and nutritional:

1. Dumping syndrome usually occurs within 30 minutes of eating high-fat/high-sugar foods and involves flushing, sweating, light-headedness, tachycardia, palpitations, nausea, diarrhea, and cramping.

2. Hypoglycemia occurs 1 to 3 hours after eating high-carb meals and involves shakiness, anxiety, sweating, chills, clamminess, confusion, rapid heart rate, dizziness, hunger, and nausea.

3. New or exacerbated reflux is more common with a GS.

4. Vitamin deficiencies or toxicities (Table 14.24):

a. Most common:

i. Iron deficiency: fatigue, lethargy, pica, food cravings.

ii. Iron toxicity: gastrointestinal (GI) irritation, nausea, vomiting, indigestion, constipation, diarrhea.

iii. Protein deficiency: weakness, decreased muscle mass, brittle hair, generalized edema.

iv. Folate deficiency: fatigue, palpitations, sore tongue, diarrhea, restless legs.

v. Calcium deficiency: usually silent, hyperparathyroidism.

vi. Calcium toxicity: constipation, nausea, vomiting, dry mouth, loss of appetite.

vii. Vitamin D deficiency: spasms/twitching of eyes, burning in mouth, sweating, weakness.

viii. Vitamin B_{12} deficiency: fatigue, burning lips/mouth, rapid heart rate, palpitations, sore tongue, weakness, mood changes, neurologic changes.

b. Less common:

i. Thiamine (B1) deficiency: usually in first 3 months postop, often a result of vomiting, blurred or double vision, difficulty swallowing, rapid heart rate, fatigue, confusion, memory loss, burning feet, leg weakness, amnesia.

ii. Zinc deficiency: loss of smell, diminished sense of taste, poor wound healing, skin rashes or roughness, hair loss, poor appetite, lethargy, grooved or deformed nails, canker sores.

iii. Magnesium deficiency: hyperexcitability, cramps, tremors, fasciculation, spasms, fatigue, loss of appetite, apathy, confusion, insomnia, irritability, poor memory.

iv. Selenium deficiency (very rare): signs of hypothyroidism (selenium is necessary for conversion of thyroxine into its active form, triiodothyronine).

v. Selenium toxicity (rare): hair loss, abnormal nails, dermatitis, peripheral neuropathy, nausea, diarrhea, fatigue, irritability, garlic odor of breath.

5. Surgical complications:

a. Short term:

i. Anastomotic leak: <1% to 3% with Roux-en-Y gastric bypass (RYGB).

ii. GI hemorrhage, bleeding: 0.5% to 2%.

iii. Wound infection: more common with open approach.

iv. Thromboembolism: deep vein thrombosis (DVT), pulmonary embolism (PE), <1%.

v. Anastomotic strictures: typically within first 3 months, nausea and/or vomiting.

vi. Acute distention of the distal stomach.

vii. Respiratory insufficiency, pneumonia.

▶

TABLE 14.24 MOST COMMON NUTRIENT DEFICIENCIES IN POSTBARIATRIC SURGERY

Nutrient	Protein	Iron	Folate	Calcium	Vitamin B_{12}	Vitamin D
Assay	Serum albumin <3.5 mg/dL May need prealbumin and serum creatinine levels also	Iron saturation <10% Serum ferritin <10 ng/mL or iron saturation <7% regardless of ferritin value	Serum folic acid	Total and ionized calcium Phosphorus 24-hr urinary calcium excretion Intact serum parathyroid hormone Bone density	Serum B_{12} level May need methylmalonic acid level also	25 (OH)D3 <30 ng/mL
Incidence	18%–25% after malabsorptive procedures	6%–50%	1%–10%	10%–25%	5%–25%	Up to 63%
Complications and symptoms	Anemia Edema Alopecia Asthenia (weakness, decreased muscle mass, brittle hair, generalized edema)	Microcytic, hypochromic anemia (pallor, fatigue, poor capillary refill, palpitations, pica, brittle hair)	Megaloblastic or macrocytic anemia (palpitations; fatigue; diarrhea; smooth, sore tongue) Neural tube defects May aggravate B_{12} deficiency	Secondary hyperparathyroidism Enhanced bone loss Metabolic bone disease (tetany, tingling, cramping)	Megaloblastic anemia with macrocytosis Neuropathy Cognitive dysfunction (glossitis, constipation, diarrhea, neurologic changes, depression, dementia)	Myopathy Secondary hypocalcemia
Recommended daily amount for prevention	1.1–1.5 g/kg ideal body weight/d 10%–35% of total energy intake should be from protein	45–60 mg/d from multivitamins and iron supplements for malabsorptive procedures Menstruating females may need more	400 mcg/d	1,200–1,500 mg/d of calcium citrate from food and supplements	500 mcg/d oral for band 1,000 mcg/d oral for malabsorptive procedures; may need 500–1,000 mcg/mo intramuscular	At least 3,000 IU of oral D3 daily, better to titrate to therapeutic level
Treatment dosing	If previous options are not effective, may need parenteral nutrition	150–200 mg of elemental iron BID, preferably with vitamin C	1,000 mcg/d for 3 months	2,000 mg/d with adequate vitamin D supplementation	Depends on the level of deficiency Recheck levels after 3–6 months of repletion	6,000–10,000 IU/d or 50,000 IU/wk up to 50,000 IU/d Recheck level every 3 months

Source: Adapted from Becker, D. A., Balcer, L. J., & Galetta, S. L. (2012). The neurological complications of nutritional deficiency following bariatric surgery. *Journal of Obesity, 2012*. https://doi.org/10.1155/2012/608534; Handzlik-Orlik, G., Holecki, M., Orlik, B., Wylezol, M., & Dulawa, J. (2015). Nutrition management of the post-bariatric surgery patient. *Nutrition in Clinical Practice: Official Publication of the American Society for Parenteral and Enteral Nutrition, 30*(3), 383–392. https://doi.org/10.1177/0884533614564995; and Kerner, J. (2014). Nutrition support after bariatric surgery. *Support Line, 36*(3), 9–20.

b. Long term:
   i. Band slippage: abdominal pain, acid reflux, regurgitation, or dysphagia.
   ii. Band erosion: may be asymptomatic, abdominal pain, GI bleeding, weight loss, abdominal sepsis.
   iii. Intestinal obstruction: caused by internal hernias, adhesions, and anastomotic stenosis—colicky central abdominal pain, nausea, vomiting, abdominal distention, absolute constipation.
   iv. Hepatobiliary complications: rapid weight loss associated with gallstone formation.
   v. GI bleeding: rare and usually caused by ulceration.
   vi. Marginal ulcers: abdominal pain, vomiting, bleeding, or anemia.
   vii. Micronutrient deficiencies.

OTHER SIGNS AND SYMPTOMS

A. Expected weight loss:
   1. LAGB: initial loss of 40% to 50% of excess body weight in 3 to 5 years. Expected maintenance <50%.
   2. GS: initial loss of >50% of excess body weight in 3 to 5 years. Expected maintenance >50%.
   3. RYGB: initial loss of 60% to 80% of excess body weight in 1 year. Expected maintenance >50%.
   4. Biliopancreatic diversion with duodenal switch (BPD/DS): initial loss of 60% to 70% of excess body weight in 1 year. Expected maintenance of 60% to 70%.
   5. Intragastric balloons: loss of about 10% to 15% of body weight typical during the 6 months following intragastric ▶

balloon placement. Expected maintenance: The gastric balloon is only placed for 6 months then removed.

B. Weight regain of up to 20 lbs is common after 2 years.

C. Constipation and/or diarrhea may occur, depending on the procedure.

D. Weight loss of <25% of excess body weight is considered a surgical failure and may be revised.

E. Even if weight loss is adequate, clients may express disappointment and/or depression related to rate or amount of weight lost.

F. Nausea and vomiting after LAGB may indicate need for band adjustment.

SUBJECTIVE DATA

A. Review onset and duration of symptoms.

B. Elicit the date of surgery, type of surgery, and any reoperations or complications.

C. Review previous highest weight and amount of excess weight lost since surgery.

D. Evaluate the location and level of pain/discomfort.

E. Evaluate the overall psychosocial changes since surgery.

F. Review a 24-hour food recall, choices of healthy foods, skipping meals, food aversion (e.g., red meat), and intolerance.

G. Review medications and supplement use.

PHYSICAL EXAMINATION

A. Check height, weight, waist, and hip circumference. Calculate BMI, waist-to-hip ratio, pulse, respirations, and blood pressure. Check temperature if infection is suspected.

B. Inspect:

1. Examine the skin, evaluate surgical site(s), evaluate redness, and tenderness.

2. Oral/dental examination.

3. Evaluate for dehydration.

4. Eye examination: Evaluate eye movement (thiamine deficiency).

5. Evaluate gait.

6. General overview of personal presence and affect.

C. Auscultate:

1. Auscultate the heart and lungs.

2. Auscultate the abdomen for bowel sounds.

D. Palpate:

1. Abdomen:

a. Evaluate for the presence of tenderness.

b. Evaluate for masses.

E. Neurologic examination:

1. Perform neurologic examination, including checking deep tendon reflexes (DTRs), sense of smell, and Babinski reflex (vitamin B_{12} deficiency).

DIAGNOSTIC TESTS

A. Annual recommended laboratory testing for all procedures:

1. Complete blood count (CBC) with differential.

2. Liver function tests.

3. Glucose.

4. Creatinine.

5. Electrolytes.

B. Annual laboratory testing suggested for LAGB, recommended for all other procedures:

1. Iron/ferritin.

2. Vitamin B_{12}.

3. Folate.

4. Calcium.

5. Intact parathyroid hormone (PTH).

6. 25-hydroxy vitamin D.

7. Albumin/prealbumin.

C. Optional labs that may be required based on symptoms:

1. Zinc.

2. Copper.

3. Vitamin B_1.

4. Vitamin B_6.

5. Vitamin A every 24 months and beyond.

D. Other labs as appropriate for condition and prevention:

1. Monitor HgA1c and blood glucose closely in clients with diabetes. Diabetes has resolved after bariatric surgery in some clients.

2. Lipid profile.

E. Abdominal ultrasound.

F. CT scan abdomen.

G. Doppler ultrasound of limb for suspected DVT.

H. Pulmonary ventilation/perfusion scan for suspected pulmonary embolus.

I. Bone mineral density (DEXA) scan: recommended annually until stable after malabsorptive procedures.

J. Endoscopy as needed for abdominal complaints.

DIFFERENTIAL DIAGNOSES

A. Postoperative surgical complication(s):

1. Infection.

2. Abdominal pain.

3. Fascial dehiscence.

4. DVT.

5. Bowel obstruction.

6. Band slippage.

7. Anastomosis leakage.

8. Stomal stenosis/stricture.

B. Cholecystitis.

C. Dumping syndrome.

D. Food intolerance.

E. Gastric ulcer (GU).

F. Gastroenteritis.

G. Vitamin deficiency.

H. Malnutrition/protein deficiency.

I. Incisional hernia.

J. Osteoporosis.

PLAN

A. General interventions:

1. Lifelong follow-up is required after bariatric surgery. Ideally, clients should follow up with their surgical group, but many do not. Primary care providers are well-positioned to capture those lost to follow-up. Continuous reinforcement of good nutritional habits is important.

B. Client teaching:

1. Many complications can be prevented by adhering to diet and lifestyle recommendations:

2. Supplements are required lifelong; strict adherence will prevent deficiencies.

3. Dumping syndrome can be prevented by avoiding foods that trigger it, such as high-sugar and/or high-fat foods. Clients can keep food diaries to identify triggers.

4. Hypoglycemia can usually be prevented by careful monitoring of carbohydrate intake.

5. Surgery does not replace the need for a balanced diet or exercise. Approximately 150 minutes of moderate activity each week is recommended, though safety and tolerance differ so exercise recommendations should be individualized. Any activity is better than none. ▶

6. Exercise is important in counteracting adverse effects on muscle mass and bone density.

7. Food should be chewed thoroughly and consumed slowly. Liquids should be avoided 30 minutes before and after meals. Avoid eating and drinking liquids simultaneously.

8. Protein is important for maintaining muscle mass during rapid weight loss and avoiding hunger during maintenance. Between 60 and 100 g of protein daily is recommended. Limit carbohydrate intake to 50 g/d or less. Limiting carbohydrate intake reduces risk of weight regain by preventing rebound hunger.

9. To prevent dehydration, as well as reduce the risk of kidney stones and constipation, encourage 64 oz of fluids daily.

10. Stress, boredom, and emotions often affect eating habits. Identifying problems and seeking help early are important.

11. Support groups can be a vital source of education and social support, both of which are key to weight loss and maintenance.

12. Weight plateaus are common and normal. This is the body's way of trying to establish a new set point. Do not be discouraged by plateaus. Consistency is key to overcoming them.

13. Adequate sleep and successful stress management are also key to successful weight loss and maintenance.

C. Pharmaceutical therapy:

1. Medication absorption can be altered after bariatric surgery. Evaluate need for adjustments of medication dosing, especially diabetic, psychiatric, and antihypertensive medications, as well as any medication with a narrow therapeutic window.

2. Clients may require alternate formulations of medications, including crushed, chewable, liquid, patches, intramuscular, or subcutaneous. Long-acting, extended-release, or enteric coated medications may not be absorbed as well and may need to be switched to immediate release.

Drugs with a small therapeutic window such as digoxin, carbamazepine, theophylline, and phenytoin should not be crushed due to the increase in toxicity.

3. Medications designed to release slowly should not be crushed or chewed because the full dose will be released faster than expected. A resource guide for crushing medications is available at www.ismp.org/recommendations/do-not-crush.

4. Universal postoperative supplementation (Table 14.25):

a. Multivitamin and mineral supplementation:
   i. LAGB: one daily.
   ii. All other procedures: two daily (take each dose at a separate time).
   iii. Tablets, chewable, or liquid.

b. Calcium:
   i. LAGB, GS, RYGB1, 200 to 1,500 mg daily from food and supplements.
   ii. BPD/DS: 1,800 to 2,400 mg daily from food and supplements.
   iii. Calcium citrate should be taken in divided doses (23 doses of 500 mg).
   iv. Chewable or liquid.

c. Vitamin D_3 (all procedures):
   i. 3,000 IU daily, titrate to therapeutic level.
   ii. Vitamin D should be taken with the calcium supplement.

d. Iron:
   i. 18 mg/d elemental iron.
   ii. RYGB, SG, BPD/DS 45 to 60 mg daily from multivitamin plus additional supplementation.
   iii. Menstruating females: 45 to 60 mg/d.
   iv. Iron should be taking separately at least 2 hours apart from calcium supplements.
   v. Iron should not be taken with milk products.

e. Vitamin B_{12} (cobalamin):
   i. Oral or sublingual: 350 to 500 mcg/d.
   ii. Intranasal: 1,000 mcg/wk.
   iii. Intramuscular 1,000 mcg/mo.

f. Folate (folic acid):
   i. 400 to 800 mcg/d.
   ii. Females of childbearing age: 800 to 1,000 mcg/d.

g. Vitamin A:
   i. LAGB: 5000 IU/d.
   ii. RYGB or SG: 5,000 to 10,000 IU/d.
   iii. BPD/DS: 10,000 IU/d.

h. Vitamin E: (all procedures) 15 mg/d.

i. Vitamin K:
   i. LAGB, SG or RYGB: 90 to 120 mcg/d.
   ii. BPD/DS: 300 mcg/d.

j. Zinc:
   i. SG or LAGB: 8 to 11 mg/d.
   ii. RYGB: 8 to 22 mg/d.
   iii. BPD/DS: 16 to 22 mg/d.

k. Copper:
   i. SG or LAGB: 1 mg/d.
   ii. RYGB or BPD/DS: 2 mg/d.

5. Long-term anticoagulation may be needed for DVT/PE prophylaxis.

6. Nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroids should be avoided to reduce the risk of marginal ulcers.

D. Psychosocial changes:

1. Bariatric surgery often results in dramatic lifestyle and body changes and may require significant psychosocial adjustments for the client as well as their friends and family:

a. Monitor for depression/anxiety, body image concerns, and social support.

b. Alcoholism can be a concern after bariatric surgery. Less alcohol is needed to elevate blood alcohol levels and blood alcohol levels are sustained longer after bariatric surgery.

FOLLOW-UP

A. Bariatric surgery requires lifelong follow-up, ideally by a multidisciplinary team of healthcare providers. Initial follow-up schedules are set by the surgeon. Long-term follow-up (after the first 2 years) is usually provided for by the bariatric surgery program.

B. Medications with narrow therapeutic windows and medication are dosed based on weight should be evaluated frequently.

C. Annual laboratory and diagnostic testing is required (refer to "Diagnostic Tests"). Early recognition of nutritional deficiencies can prevent permanent damage and even death.

D. Chronic disease management (particularly diabetes, lipids, and bone disease) can be simpler or more complex after bariatric surgery, depending on the client's adherence to lifestyle and nutritional recommendations. ▶

TABLE 14.25 ROUTINE SUPPLEMENTATION BASED ON TYPE OF GASTRIC BYPASS SURGERY

Supplement	Gastric Band	Gastric Sleeve	Gastric Bypass	BPD/DS
Multivitamin plus mineral	One daily	Two daily	Two daily	Two daily
Calcium	1,200–1,500 mg daily from food and calcium citrate divided doses	1,200–1,500 mg daily from food and calcium citrate divided doses	1,200–1,500 mg daily from food and calcium citrate divided doses	1,200–1,500 mg daily from food and calcium citrate divided doses
Vitamin D	3,000 IU daily; titrate to therapeutic level	3,000 IU daily; titrate to therapeutic level	3,000 IU daily; titrate to therapeutic level	3,000 IU daily; titrate to therapeutic level
Iron	Not usually necessary	45–60 mg/d from multivitamin plus additional supplement	45–60 mg/d from multivitamin plus additional supplement	45–60 mg/d from multivitamin plus additional supplement
Vitamin B_{12}	Not usually necessary	As needed to maintain levels in the form best tolerated	As needed to maintain levels in the form best tolerated	As needed to maintain levels in the form best tolerated

BPD/DS, biliopancreatic diversion with duodenal switch.

Source: Adapted from Kerner, J. (2014). Nutrition support after bariatric surgery. *Support Line, 36*(3), 9–20; Shannon, C., Gervasoni, A., & Williams, T. (2013). The bariatric surgery patient–nutrition considerations. *Australian Family Physician, 42*(8), 547–552.

CONSULTATION/REFERRAL

A. Bariatric surgeon for surgical complications, revisions.
B. Surgeon for cholecystectomy (preferably bariatric surgeon or general/GI surgeon experienced in the care of bariatric clients).
C. Gastroenterology consult.
D. Nutrition consultation and/or counseling.
E. Psychologist consultation.
F. Physical therapy/exercise specialist.
G. Support group.

INDIVIDUAL CONSIDERATIONS

A. Females:
  1. All females of childbearing age should receive adequate folate supplementation and be given contraception and preconception counseling.
  2. Oral contraceptives (OCPs) may not be as effective after bariatric surgery due to changes in absorption.
B. Pregnancy:
  1. Pregnancy should be delayed for 12 to 18 months after bariatric surgery or however long it takes for weight loss to stabilize.
  2. Bariatric surgeon consult may be required.
  3. Increased folic acid may be needed preconception to reduce risk of neural tube defects.
  4. Increased vitamin supplementation may be necessary during pregnancy. Vitamin A should be limited to 5,000 IU daily.
  5. Gastric band may need to be adjusted during pregnancy.
  6. Serial ultrasounds may need to be done to follow fetal growth.
C. Geriatrics:
  1. Discussion of bariatric surgery in older adults (over 60 years of age) is controversial. However, the client should not be denied bariatric surgery due to age. Mortality rate was 0.01%, complication rate ranged from 9.7% to 14.7%. At 1-year follow-up, excess weight loss was approximately 53.77%, with improvement of diabetes by 54.5%, hypertension improvement 42.5%, and cholesterol improvement 41.2%.

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ULCERATIVE COLITIS

DEFINITION

A. Ulcerative colitis (UC) is one of the two inflammatory bowel diseases (IBDs), along with Crohn's disease (CD). UC is limited to the colon; it extends proximally from the anal verge in an uninterrupted pattern to a part of or the entire colon. Extracolonic manifestations also include uveitis, pyoderma gangrenosum, pleuritis erythema nodosum, ankylosing spondylitis, and spondyloarthropathies. Refer to Table 14.26 for the definition of severity of UC.
B. Neither UC nor CD should be confused with irritable bowel disease (IBD), which affects the motility of the colon. Smoking is negatively associated with UC; the relationship is reversed in CD. The general course of UC is intermittent exacerbations and remissions. In severe cases, surgery may be required. The choice of treatment depends on disease activity and extent of pathology, client acceptability, and mode of drug delivery.
C. Histologically, most of the pathology of UC is limited to the mucosa and submucosa. The severity of UC is defined by the following:
  1. Pan-ulcerative (total colitis): extensive disease with evidence of UC proximal to the splenic flexure. Massive ▶

TABLE 14.26 DEFINITION OF SEVERITY OF ULCERATIVE COLITIS

Mild UC	• ≤4 bloody stools/d with or without blood • No systemic toxicity • Normal ESR • Mild abdominal pain or cramping
Moderate UC	• >4 bloody stools/d • No signs of systemic toxicity • Pulse <90 beats/min • Temperature <37.5°C (99.5°F) • Hemoglobin >10.5 g/dL • ESR <30 mm/hr • Moderate abdominal pain
Acute severe UC[a]	• ≥6 bloody stools/d or observable massive and significant blood BM *AND* • 1 or more symptoms of systemic toxicity • Tachycardia >90 beats/min • Temperature >37.8°C (100.4°F) • Hemoglobin <10.5 g/dL • Increased ESR (>30 mm/hr)
Fulminant UC	• >10 stools/d • Continuous rectal bleeding • Systemic toxicity • Tachycardia >90 beats/min • Fever >37.8°C (100.4°F) • Anemia requiring blood transfusions • Abdominal tenderness and distension • Colonic dilation on radiography • May lead to toxic megacolon or colonic perforation

[a]Acute severe colitis is defined by Truelove and Witts Criteria (1955).

BM, bowel movement; ESR, erythrocytic sedimentation rate; UC, ulcerative colitis.

Source: Adapted from Truelove, S. C., and Witts, L. J. (1955). Cortisone in ulcerative colitis; final report on a therapeutic trial. *British Medical Journal 2*(4947), 1041–1048.

dilation of the colon (toxic megacolon) may lead to bowel perforation.

2. Left-sided disease: continuous UC that is present from the rectum, the descending colon up to, but not proximal to, the splenic flexure.

3. Proctosigmoiditis: disease limited to the rectum and sigmoid colon involvement.

4. Ulcerative proctitis: disease limited to the rectum, usually less than the full rectum.

D. In 2015, the Toronto Consensus Clinical Practice Guidelines for the Management of Nonhospitalized UC published 34 recommendations, including statements on 5-aminosalicylates (5-ASA) corticosteroids, immunosuppressants, anti-tumor necrosis factor (TNF) therapy, and other agents, including probiotics. The guidelines are available at www.gastrojournal.org/article/S0016-5085(15)00303-0/abstract.

E. The 2015 Toronto Consensus guideline defined remission and response with UC as:

1. Complete remission: both symptomatic remission and endoscopic healing defined as follows:

a. Endoscopic healing: normal mucosa, vascular blurring, or chronic changes (e.g., inflammatory polyps, scarring) without friability.

b. **Symptomatic remission: normal stool frequency (≤3/d) and no blood in the stool.**

2. Symptomatic response: meaningful improvement in symptoms as judged by both the client and physician in the absence of remission; response should not be considered a desirable final outcome but is useful to assess early response to treatment.

INCIDENCE

A. The annual incidence of UC is 10.4 to 12 per 100,000, depending on the country.

B. UC is three times more common than CD.

C. UC can occur at any age, but predominantly occurs between ages 15 and 30.

D. The most common cause of death in clients with UC is toxic megacolon.

E. Adenocarcinoma of the colon develops in 3% to 5% of clients with UC; the risk increases with the duration of the disease.

F. Approximately 6.2% of clients with IBD have a major extraintestinal manifestation.

1. Uveitis is the most common: 3.8%.

2. Primary sclerosing cholangitis: 3%.

3. Ankylosing spondylitis: 2.7%.

4. Erythema nodosum: 1.9%.

5. Pyoderma gangrenosum: 1.2%.

PATHOGENESIS

A. Exact etiology is unknown.

B. UC may be considered an autoimmune disease. Persons with UC often have p-antineutrophil cytoplasmic antibodies (p-ANCAs). Abnormalities of humoral and cell-mediated immunity and/or generalized enhanced reactivity against intestinal bacterial antigen may also be causes of UC.

PREDISPOSING FACTORS

A. Caucasian.

B. Jewish descent.

C. 30% more females than males.

D. Genetic susceptibility (chromosomes 12 and 16).

COMMON COMPLAINTS

A. Frequent small-volume diarrhea.

B. Bloody diarrhea with or without mucus.

C. Severe bowel urgency.

D. Abdominal cramps and pain with bowel movement (BM).

E. Constipation.

F. Anorexia.

G. Anemia.

H. Nocturnal BMs.

OTHER SIGNS AND SYMPTOMS

A. Tenesmus (rectal urgency/constant feeling of need to pass stool).

B. Abdominal tenderness.

C. Arthralgias.

D. Fatigue secondary to anemia.

E. Severe UC:

1. Fever.

2. Tachycardia.

3. Significant abdominal tenderness.

4. Signs of volume depletion.

SUBJECTIVE DATA

A. Review the onset, duration, signs, and symptoms (number of stools, presence/absence of blood in the stool, fever, and abdominal pain).

B. Review the client's recent travel history or camping for presence of intestinal infection. ▶

C. Review the client's medication history, including antibiotics and nonsteroidal anti-inflammatory drugs (NSAIDs). One-third of clients with exacerbation of UC report recent NSAID use.
D. Review family history for IBD, celiac disease, and colorectal cancer.
E. Review the client's smoking status.
F. Review the client's history or contact related to tuberculosis (TB; testing required prior to biologic therapy).
G. Evaluate if the client has symptoms of uveitis, including light sensitivity, floaters, blurry vision, or pain/tenderness to touch.

PHYSICAL EXAMINATION

A. Check temperature (if indicated), pulse, and blood pressure. Measure height and weight to calculate body mass index (BMI). Follow serial weights.
B. Inspection:
1. Observe the general overall appearance for nutritional status, cachexia, and pallor.
2. Observe the perianal region for the presence of tags, fissures, fistulas, and abscess.
3. Observe the abdomen for distension and presence of surgical scars.
4. Eye examination for redness, irritation, and ocular complications (episcleritis, scleritis, and uveitis).
5. Evaluate for presence of dermatologic findings, including erythema nodosum and pyoderma gangrenosum.
C. Auscultate:
1. Auscultate the heart and lungs.
2. Auscultate all four quadrants of the abdomen.
D. Palpate:
1. Palpate all four quadrants of the abdomen, observing for tenderness, rebound, and guarding.
2. Evaluate the presence of hepatomegaly.
3. Evaluate the joints for tenderness, warmth, swelling, and effusion.
E. Rectal examination:
1. Perform digital rectal examination to assess for anal strictures and rectal masses.

DIAGNOSTIC TESTS

A. Diagnosis is best made with endoscopy and biopsy.
B. Laboratory tests:
1. Complete blood count (CBC) with electrolytes.
2. Platelet count.
3. Erythrocyte sedimentation rate (ESR).
4. C-reactive protein (CRP).
5. Cytomegalovirus (CMV): chronic immunosuppressive steroids.
6. HIV.
C. Stool testing:
1. Evaluate bacterial, viral, or parasitic causes of diarrhea.
a. Based on the American College of Gastroenterology (ACG) guidelines, rule out *Clostridioides difficile* in clients suspected of having.
2. Occult blood.
3. Fecal leukocytes.
D. Plain abdominal radiograph.
E. CT scan.
F. MRI.
G. Ultrasound.
H. Double-contrast barium enema.
I. Celiac antibody testing should be considered.
J. Intestinal TB testing should be considered.

DIFFERENTIAL DIAGNOSES

A. UC.
B. Ischemic colitis (especially in older adults).
C. Toxic megacolon.
D. Colon cancer.
E. Adenocarcinoma.
F. Rectal cancer.
G. Radiation colitis.
H. Intestinal infections.
I. Intestinal lymphoma.
J. Chronic diverticulitis.
K. Amebiasis.

PLAN

A. General interventions:
1. Severe UC should be managed jointly by a gastroenterologist in conjunction with a colorectal surgeon.
2. Stress reduction and stress management may improve symptoms.
3. Pretreatment screening for TB, using Mantoux (a purified protein derivative) skin testing, is needed before initiation of immunomodulators and thiopurines.
4. Immunization status:
a. Immunizations with inactivated vaccine should be brought up to date and rigorously maintained during treatment, including influenza, meningococcus, and pneumococcus.
b. Check varicella titers prior to treatment with immunomodulators and reimmunize if titers are low.
c. The risk of administering live vaccines (polio, rubella, and yellow fever) to clients on immunomodulators has not been established; however, most experts avoid live vaccines during treatment.
B. Client teaching: *See Client Teaching Guide for this chapter, "Crohn's Disease and Ulcerative Colitis."*
1. Clients with CD who smoke should be encouraged and offered help to stop smoking at each office visit. *See Client Teaching Guide for Chapter 25, "Nicotine Dependence."*
C. Dietary management:
1. Adequate nutrition is critical to promotion of healing. Sufficient protein and calories limit the stress on an inflamed bowel.
2. Many clients with UC have concurrent lactose intolerance (see Appendix B, "Dietary Recommendations," for lactose-intolerance dietary recommendations).
3. Decrease dietary fiber during increased disease activity.
4. Low-residue diet may decrease the frequency of BMs.
5. High-residue diet may be helpful in ulcerative proctitis when constipation is the dominant symptom.
6. Vitamin and nutritional supplements may be recommended.
D. Pharmaceutical therapy:
1. Refer to the ACG Practice Guidelines for full treatment algorithms at http://s3.gi.org/physicians/guidelines/UlcerativeColitis.pdf or the 2015 Toronto Consensus Guidelines for the Management of UC algorithms:
a. The choice of topical agents is guided by the proximal distribution of UC into the bowel, as well as client preference (Table 14.27).
b. Refer to Table 14.6 for a stepwise medication approach for both of the inflammatory bowel diseases (IBDs).
c. The aminosalicylates (5-ASA) class of anti-inflammatory drugs is the most common treatment for clients with mild (<4 bloody stools per day) or ▶

TABLE 14.27 MANAGEMENT OF MILD TO MODERATE DISTAL ULCERATIVE COLITIS WITH TOPICAL AGENTS

Topical agent	Proximal extent/distribution of agent
Suppository	10 cm
Hydrocortisone foam	15–20 cm
Enema	As far as the splenic flexure

Source: Adapted from the AGA Ulcerative Colitis in Adults Practice Guidelines (2010).

moderate active disease (>4 bloody stools a day without systemic toxicity).

i. Sulfasalazine (Azulfidine) for mild to moderate UC and remission maintenance:

1) Pediatrics: 50 to 75 mg/kg each 24 hours, divided into three to four doses.

2) **Reduced absorption of folic acid and digoxin has been reported when administered with sulfasalazine.**

ii. Balsalazide (Colazal) for mild to moderate UC and remission maintenance:

1) Induction: 6.75 g/d, given TID dosing.

2) Maintenance: 2 to 6.75 g/d, given BID dosing.

iii. Mesalamine for mild to moderate UC and remission maintenance.

d. Asacol:

i. **Two Asacol 400 mg tablets are not interchangeable or substitutable with one mesalamine delayed-release 800 mg tablet.**

e. Apriso: 1.5 mg/d orally in the morning.

f. Salofalk: 1 to 4 g/d in one or two divided doses:

i. Also available as a suppository, rectal suspension, and enema.

g. Pentasa: 1 g/d orally QID dosing for up to 8 weeks:

i. Also available in a suppository.

h. Multi-Matrix System (MMX) marketed as Lialda: active to moderate UC:

i. Induction: 2.4 to 4.8 g/d for up to 8 weeks.

ii. Maintenance: 2.4 g/d.

i. Rowasa enema: 4 g/60 mL rectally at bedtime; retain for approximately 8 hours.

i. Mesalamine (Canasa) suppository 1 g/d at bedtime; retain in the rectum for at least 1 to 3 hours. Treat active proctitis for 3 to 6 weeks.

j. Corticosteroids suppress the immune system and are used for moderate to severe UC:

i. Prednisone (Deltasone, Orasone):

1) Induction: 40 to 60 mg/d PO for 7 to 14 days followed by gradual taper by 5 mg/wk.

2) Maintenance: 2.5 to 5 mg/wk.

k. Immune modifiers are used to decrease corticosteroid dosage:

i. Azathioprine (AZA; Imuran) 2 to 3 mg/kg/d orally:

1) Monitoring includes CBC, including platelet counts weekly during the first month, twice monthly for the second and third months of therapy, then monthly or more frequently if dosage alteration is necessary.

2) Thiopurine methyltransferase (TPMT) testing: Testing is recommended for consideration to be given to either genotype or phenotype clients for TPMT.

ii. 6-Mercaptopurine (6-MP; Purinethol) 1 to 1.5 mg/kg/d orally.

1) Clients with little or no inherited TPMT activity are at increased risk for severe Purinethol toxicity and generally require substantial dose reduction.

l. Antibiotics.

i. Cyclosporine (Neoral, Sandimmune).

1) Intravenous (IV) infusion: 2 to 4 mg/kg/d.

2) Can be switched to a doubled oral dose for outpatient therapy.

ii. Ciprofloxacin (Cipro) 500 mg orally BID.

iii. Metronidazole (Flagyl).

iv. Biologic therapy (anti-tumor necrosis factor [TNF] agents) for moderate to severe UC: **A tuberculin skin test is recommended prior to therapy. Clients started on infliximab should also be screened for hepatitis B before initiating therapy:**

1) Infliximab (Remicade) infusion therapy has a half-life of approximately 10 days.

2) Adalimumab (Humira) for treatment of moderate to severe UC.

3) Golimumab (Simponi):

a) Initial dosage is 200 mg subcutaneously at week 0.

b) Second dose (week 2): 100 mg.

c) Maintenance dose: 100 mg every 4 weeks.

4) Vedolizumab (ENTYVIO):

a) Initial dose: 300 mg IV at weeks 0, 2, and 6.

b) Maintenance dose: 300 mg IV every 8 weeks of therapy.

c) **Discontinue if no evidence of therapeutic benefit by week 14.**

E. Surgical management:

1. Indications for consideration of a total colectomy:

a. Failed medical therapy: refractory UC.

b. Severe hemorrhage.

c. Fulminate colitis not responsive to treatment.

d. Toxic megacolon.

e. Obstruction or stricture.

2. Indications for consideration for elective surgery:

a. Long-term steroid dependence.

b. Dysplasia or adenocarcinoma found on screening biopsy.

c. Disease present 7 to 10 years.

FOLLOW-UP

A. Screening colonoscopy is recommended for all clients with UC for 8 to 10 years after the onset of symptoms due to the increase in colonic neoplasia.

B. Clients with extensive UC or left-sided colitis with negative findings on the screening colonoscopy should begin surveillance colonoscopy in 1 to 2 years.

C. Steroids should not be used as maintenance therapy. Clients who require long-term steroids are at increased risk of osteoporosis. ▶

D. Subsequent laboratory monitoring tests are dependent on the prescribed therapy.

E. Anxiety and depression are higher in clients with IBD. Screen for depression at each visit.

EMERGENT ISSUES/INSTRUCTIONS

A. Clients with ocular complications require an urgent consultation.

CONSULTATION/REFERRAL

A. Gastroenterologist for confirmatory diagnosis with a colonoscopy.

B. Consultation with a surgeon for severe or fulminant colitis. Toxic megacolon is a life-threatening complication and requires urgent surgical intervention.

C. Clients who have had UC for 8 to 10 years are at risk for colon cancer; therefore, colonoscopy for surveillance is recommended.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. Live vaccinations should not be administered to immunocompromised clients. If required, vaccines should be administered at the time of UC diagnosis:

a. The flu and pneumonia should be routinely administered.

b. Consider administering the human papillomavirus vaccine.

2. Females with IBD have been reported to have a high incidence of abnormal Pap smears. Adherence to Pap smear guidelines (refer to Chapter 2, "Healthy Living for the Adult-Geriatric Client").

3. Abnormal sperm counts, motility, and morphology are seen with sulfasalazine.

B. Geriatrics:

1. Due to polypharmacy, a full review of medications, herbals, and over-the-counter (OTC) drugs should be undertaken to identify medications that should be avoided in the geriatric population, duplicate medications, and medications that are able to be decreased or deprescribed (refer to deprescribing section in Chapter 3 "Adult-Geriatric Assessments").

2. For the older adult with existing UC, nurse practitioners (NPs) should practice special consideration of the following *geriatric syndromes*:

a. Delirium is a common early sign of acute illness or infection in older adults. It is more common in clients with existing cognitive impairment. At a minimum, clients and their family should be asked if the client has experienced episodes of altered mental status:

i. The Brief Confusion Assessment Method (bCAM) is a well-documented assessment for delirium. The bCAM is available at www.mnhospitals.org/Portals/0/Documents/ptsafety/LEAPT%20Delirium/HELP%20Program%20 CAM%20Flowsheet.pdf.

b. Pain: Poorly relieved pain is an important cause of functional impairment in any age group:

i. Older adults have a diminished sensorium, allowing pathology to advance to a dangerous point prior to symptom development.

ii. Older adults with abdominal pain may present with altered mental status. Cognitive impairment can make assessment and diagnosis of pain more difficult.

iii. For dementia or noncommunicative clients, the American Medical Directors Association has endorsed the Pain Assessment in Advanced Dementia (PAINAD) Scale, which scores the following items:

1) Breathing: Examples include normal, labored, hyperventilation.

2) Negative vocalization: Examples include moaning, negative language, crying.

3) Facial expression: Examples include smiling, frowning, grimacing.

4) Body language: Examples include relaxed, tense, rigid, or striking out.

5) Consolability: Examples include extent to which a caregiver is able to console, distract, or reassure.

c. Weight loss/appetite suppression: Weight loss is a predictor of mortality. Clinically significant weight loss is considered to be 5% loss of usual body weight in 3 months or 10% in 6 months.

i. Monitor weight patterns/BMI. (Caution: BMI is not used to identify loss of muscle mass.)

ii. Monitoring both serum albumin and anemia: Hypoalbuminemia and anemia are not necessarily associated with low bodyweight. They can be caused by decreases in liver protein synthesis:

1) Serum albumin concentration is commonly recommended.

2) Hemoglobin, prealbumin, and transferrin.

3) Cholesterol.

d. Depression: Anxiety and depression are common in IBD and are factors that lead to decreased health-related quality of life. A depression screen should be done at each office visit:

i. An online Patient Health Questionnaire-9 (PHQ-9) is available at www.mdcalc.com/phq-9-patient-health-questionnaire-9.

ii. The Geriatric Depression Scale, Short Form, a 15-item questionnaire that can be quickly applied in the clinical setting, is available at web.stanford.edu/yesavage/GDS.english.short.html.

RESOURCES

American College of Gastroenterology (ACG): http://gi.org

Crohn's and Colitis Foundation of America (CCFA): www.ccfa.org

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CLIENT TEACHING GUIDE

ABDOMINAL PAIN

PROBLEM

A. Abdominal pain may range from simple gas to appendicitis. Acute pain is pain that has started recently; recurrent pain is present in three or more separate times over at least a 3-month period.

B. It is important that you call if you have pain that lasts 3 hours or longer, have a fever, are vomiting, or if the pain is unusually sharp or intense.

CAUSE Pain may result from inflammation, poor blood supply, bloating, constipation, or blockage. Gastroenteritis is the most common cause of acute pain, and constipation is the most common cause of chronic pain. Urinary tract infections (UTIs) can also cause abdominal pain.

A. Males: Torsion of the testicles or a strangulated inguinal hernia may cause abdominal pain.

B. Females: If you have missed a period or suspect you are pregnant, tell your healthcare provider; ectopic pregnancies are a medical emergency.

PREVENTION The following suggestions can prevent abdominal pain from constipation:

A. Go to the bathroom as soon as you have the urge to have a bowel movement (BM).

B. Establish a regular toilet time, such as after breakfast; 15 to 20 minutes after breakfast is a good time because your gastrointestinal (GI) motility is greatest during this period.

TREATMENT PLAN Do not take laxatives, use enemas, or take drugs, food, or liquids (including water) until calling your healthcare provider to report suspected abdominal pain and the following:

A. Increased or odd-looking vomit or stools.

B. Hard, swollen abdomen.

C. Lump in scrotum, groin, or lower abdomen.

D. Missed period or suspected pregnancy.

Activity: As tolerated. Abdominal pain with nausea and vomiting, with fever, or that lasts more than 3 hours and makes you stop doing daily activities should be reported.

Diet: Do not eat food or drink liquids until you see a healthcare provider if you have pain with nausea and vomiting, with fever, or pain that lasts longer than 3 hours.

Medications: Narcotic pain medications may or may not be prescribed depending on your physical examination.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Any change in first symptoms that brought you to the office.

B. Fever higher than _____ degrees.

C. You are unable to have a BM and unable to pass gas, especially if you have had surgery.

D. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

CROHN'S DISEASE AND ULCERATIVE COLITIS

PROBLEM Crohn's disease (CD) and ulcerative colitis (UC) are inflammatory disorders of the gastrointestinal (GI) tract that produce abdominal pain, ulceration, formation of fibrous tissue, and malabsorption of important vitamins and minerals.

CAUSE The cause is unknown, but it can be aggravated by bacterial infection or inflammation.

TREATMENT PLAN

A. Adequate nutrition is critical to the promotion of healing.
B. Vitamins, minerals, and folic acid are necessary for proper healing and to avoid complications, such as bone disease and low blood counts.
C. To relieve pain, apply a heating pad or warm compress to your abdomen.
D. Check your bowel movements (BMs) daily for signs of bleeding.
E. You may need to have blood tests.
F. You may need to see a specialist.
G. Surgery may be required to help control symptoms.
H. There may be a support group near where you live. The Crohn's and Colitis Foundation website—at www.ccfa.org/living-with-crohns-colitis/find-a-support-group—has an area to find a support group.

Activity: During acute attacks, rest in bed or in a chair. Get up only to go to the bathroom, bathe, or eat. Between attacks, resume normal activities, as tolerated.

Diet:

A. When you have diarrhea, increase the fiber content of your diet. Increasing fiber in your diet is used for diarrhea and constipation.
B. Restricting milk products may stop the diarrhea. Omit milk products for a short time, then try them again in a few weeks.
C. Reducing the amount of fat and gluten in your diet may help.
D. Ensure, Sustacal, and Isocal have been found to induce remission and improve symptoms.
E. Severe relapse may require partial bowel rest. Contact the office for instruction.

Medications: You may be prescribed vitamins and minerals, medicine to control pain and relieve diarrhea, and a steroid to reduce the inflammation. Do not stop taking the steroid abruptly. Your healthcare provider can tell you how to taper the dose over several days.

You Have Been Prescribed the Following Vitamins and Minerals:

A. _____
B. _____
C. _____
D. _____

You Need to Take: _____

You Have Been Prescribed the Following to Relieve Diarrhea: _____

You Need to Take: _____

You Have Been Prescribed the Following Steroid: _____

You Need to Take: _____

Other medications to treat inflammatory bowel diseases include oral medication, intravenous infusions, and injectable medications to control the severity of symptoms. Your healthcare provider will discuss these with you and the need for close follow-up monitoring.

You Have Been Prescribed: _____

(*continued*)

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CLIENT TEACHING GUIDE

CROHN'S DISEASE AND ULCERATIVE COLITIS (*CONTINUED*)

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Black, tarry stools, or blood in the stool.
B. A swollen abdomen.
C. A temperature of 101.0°F or higher.
D. Other: _____

Phone: _____

You Need to Return to the Office for Blood Work: _____

RESOURCE Crohn's and Colitis Foundation of America: www.ccfa.org, 386 Park Avenue South, 17th Floor, New York, NY 10013, 800-932-2423, info@ccfa.org

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CLIENT TEACHING GUIDE

DIARRHEA

PROBLEM Diarrhea is loose, watery bowel movements (BMs).

CAUSE There are many causes of diarrhea, including viral and bacterial infections and parasites.

PREVENTION

A. Avoid raw seafood and undercooked foods.
B. Store food in the refrigerator within 1 hour of cooking to prevent the growth of bacteria. Especially avoid buffet or picnic foods left out for several hours and food served by street vendors.
C. Wash your hands well before preparing foods, after going to the bathroom, and after handling diapers.
D. When traveling:
  1. Avoid local water supplies (including ice): Drink bottled water instead.
  2. Wash fresh fruits and vegetables before eating.
  3. If possible, travel with antiseptic hand sanitizer and wipes.
E. When hiking or camping, do not drink from streams, springs, or untested wells. Boil all water used for drinking or cooking.
F. Do not allow people with diarrhea to handle food.
G. Thoroughly cook eggs and other foods of animal origin. Do not eat raw eggs or foods containing raw eggs, such as cookie dough.
H. Keep your child away from childcare centers if their diarrhea is too much for a diaper or they are unable to get to the toilet.

TREATMENT PLAN

A. Spontaneous recovery usually occurs in 24 to 48 hours.
B. Keep drinking liquids.
C. If you think a prescription drug is causing diarrhea, talk with your healthcare provider before stopping the medication.
D. Clean toys and hard surfaces with soap and water. Chlorine-based disinfectants inactivate rotavirus and may help prevent disease transmission in childcare centers.

Activity: Decrease activities until diarrhea stops.

Diet:

A. If diarrhea is accompanied by nausea, suck ice chips.
B. Drink clear liquids frequently, such as 7UP, Gatorade, ginger ale, broth, or gelatin, until diarrhea stops.
C. Use popsicles for added liquid.
D. After symptoms disappear, eat soft foods, such as cooked cereal, rice, baked potatoes, and yogurt, for 1 to 2 days.
E. You can eat normal foods in 2 to 3 days after the diarrhea stops.
F. Avoid fruit, alcohol, and highly seasoned foods for several more days.
G. Avoid raw foods such as salads for 2 to 3 days after diarrhea ends.

Medications: _____

You Have Been Prescribed the Following Antidiarrheal Medication: _____

You Need to Take: _____

You Need to Notify the Office If:

A. You have diarrhea lasting more than 2 days or chronic diarrhea (especially if you are a diabetic).
B. You have mucus, blood, or worms in the stool.
C. You have a fever of 101.0°F or higher.
D. You have severe pain in the stomach or rectum.
E. You have dehydration symptoms, including dry mouth, wrinkled skin, excessive thirst, and little or no urine.
F. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

FECAL INCONTINENCE

PROBLEM Fecal incontinence is losing mucus or stool while passing gas or completely losing stool before getting to a toilet.

CAUSES There are many causes of fecal incontinence, including having diarrhea around a hard solid impacted stool from constipation, not being active, problems after delivery of a baby, taking too many laxatives, hemorrhoids, and postradiation treatment.

PREVENTION

A. Go to the bathroom as soon as you feel that you need to have a bowel movement (BM).
B. Have a scheduled time to go to the toilet; after eating is a good time.
C. Tips to help deal with uncontrolled BMs:
1. Use the toilet before leaving home.
2. Identify public restrooms before they are needed.
3. Carry a bag of supplies.
4. Change your clothes as soon as possible.
5. Pants with elastic bands are easier to pull down than pants that have buttons.
6. Wear disposable underwear/absorbent pads.
7. Use water without soap. Soap dries and irritates.
8. Use baby wipes instead of toilet paper.
9. Use a moisture-barrier cream such as zinc oxide.
10. Use a special deodorant for your bowels.

TREATMENT PLAN

A. You may be asked to keep a stool diary for 1 week (Table 1).

TABLE 1 BOWEL CONTROL STOOL DIARY

PLEASE RECORD YOUR STOOL HABIT FOR 1 WEEK				**Name**				
Date	**Time of BM**	**Incontinence**	**Stool Seepage or Staining**	**Stool Consistency (Type 1–5, see below)**	**Urgency/ Unable to Postpone BM for More Than 15 Minutes**	**Use of Pads**	**Medications**	**Comments**
		Yes/No	Yes/No		Yes/No	Yes/No		

Type 1, hard to pass—marble-like stool; **Type 2,** somewhat difficult to pass—stool is shaped like a chain; **Type 3,** easy to pass—stool comes out in a smooth long shape; **Type 4,** easy to pass—stool is in soft separate pieces; **Type 5,** diarrhea—with no formed pieces of stool, comes out like water.

BM, bowel movement.

Source: From National Institute of Diabetes and Digestive and Kidney Diseases, National Institute of Health Let's Talk about Bowel Control" available at https://www.niddk.nih.gov/-/media/Files/Weight-Management/Stool_Diary_508.pdf.

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CLIENT TEACHING GUIDE

FECAL INCONTINENCE (*CONTINUED*)

B. If you are constipated:
  1. You may need to have the hardened impacted stool removed.
  2. Drink 8 to 10 glasses of water a day.

Activity: It is important to remain active, such as walking for exercise.

Diet:
A. You may be asked to keep a food diary for 1 week.
B. Eat more fiber in your diet. Fiber helps diarrhea and constipation.

You Have Been Instructed to Take the Following Fiber Supplement: _____

You Need to Take: _____

You Have Been Prescribed the Following Medication: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. Blood in your bowel movements.
B. Fever of 101.0°F or higher.
C. Severe pain in the stomach or rectum.
D. Other: _____

Phone: _____

FIBER SUGGESTIONS High-fiber diets are used to help prevent constipation as well as diarrhea. Fiber has been used to help several medical conditions such as diabetes, diverticulosis, irritable bowel syndrome (IBS), and high cholesterol, as well as weight loss. If you have a chronic health condition, check with your healthcare provider about starting any dietary change.

Fiber provides a full feeling that can help with spacing meals further apart (3–4 hours). Fiber recommendations change with your age.

TIPS FOR INCREASING FIBER IN YOUR DIET

A. Read the Nutrition Facts food labels for fiber content per food serving:
  1. Cereals that provide 5 g of fiber per serving give you 20% of your daily fiber.
  2. Look for "whole grain" on the label. Just because bread is brown does not mean it is whole grain.
B. Increase fiber in your diet gradually to prevent gas. Adding too much fiber too quickly may give you abdominal pain, bloating, and constipation. Increase your fiber over several weeks so that it gives time for you to adjust.
C. As you increase fiber, it is also important to increase the amount of fluids you drink up to six to eight glasses a day, including tea, milk, fruit juices, coffee, and even soft drinks. The extra fluids that you drink along with the extra fiber make you feel fuller, which can help control snacking.
D. Keep a food diary and review it periodically to decide on other diet adjustments that need to be made.
E. Several fiber supplements are available over the counter to help you get your daily recommendation of fiber.

GOOD FOOD SOURCES OF FIBER

A. Bran: Add one teaspoon of whole-grain bran to food three times a day, or take an over-the-counter fiber supplement such as psyllium (Metamucil), as directed.
B. Whole-grain cereals and breads: Eat oat, bran, multigrain, light, wheat, or rye breads rather than pure white bread or breads that list eggs as a major ingredient. Grains are not only a good source of fiber but also contain vitamins and minerals. Folic acid has been added to breads and cereals to help reduce neural tube defects.

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FECAL INCONTINENCE (*CONTINUED*)

C. Fresh or frozen fruits and vegetables: Citrus fruits are especially good sources of fiber. Eat raw or minimally cooked vegetables, especially squash, cabbage, lettuce, other greens, and beans. Leave the skins on fruits and vegetables; eating the whole fruit is better than drinking the juice. Whole tomatoes offer more fiber than peeling the skin off. The more colorful the fruit and vegetable (dark green, reds, blue, yellow) the better; they are a good source of antioxidants that are good for the heart and the prevention of some cancers. Apples are a good source of both fiber and water.

D. Legumes (pods): Peas and beans are a good source of fiber. Add chickpeas and kidney beans to salads for extra fiber and flavor. Add baked beans as a delicious side item to your meal.

E. Coffee is another source of fiber.

F. Nuts are an excellent source of fiber. They are considered nutrient-dense and are a good source of vitamins and folic acid. Sprinkle sunflower seeds on a salad to add flavor and fiber. The amount of nuts eaten should be limited to 1 to 2 oz because they are also high in calories.

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CLIENT TEACHING GUIDE

GASTROESOPHAGEAL REFLUX DISEASE (GERD)

PROBLEM You have stomach acid or a feeling of "heartburn" and "acid brash."

CAUSE Heartburn happens when stomach contents come back up into the throat. Diet, spicy foods, alcohol, pregnancy, and nervous tension can make it worse.

PREVENTION

A. Avoid smoking.
B. Avoid things that increase pressure on your stomach:
  1. Straining causes more pressure on your stomach.
  2. Coughing causes more pressure on your stomach.
  3. Do not lie down or bend over for 3 hours after eating.
  4. Do not wear tight clothes and belts.
C. Avoid medications such as aspirin or ibuprofen, which may irritate your stomach.
D. . Avoid alcohol.

TREATMENT PLAN

A. Weight loss is advised to relieve symptoms.
B. Stop smoking. **It is never too late to stop smoking.**
C. You may be instructed to elevate the head of your bed on 6-in. blocks, or sleep on a wedge bolster pillow.
D. Make a list of all of your medications, over-the-counter medications, and herbal products so that you can review them with your healthcare provider to help find any side effects that may cause your symptoms.
E. Take your medications at the correct times.

Activity: Postpone vigorous exercise until your stomach is likely to be empty, about 2 hours after eating.

Diet:
A. Eat a lower fat, bland diet.
B. Eat four to six small meals a day instead of three larger meals.
C. Do not eat 2 to 3 hours before bedtime.
D. Avoid chocolate, garlic, onions, citrus fruits, coffee (including decaffeinated), alcohol, highly seasoned foods, and carbonated beverages.
E. Eat slowly.

Medications:
A. Sit or stand when taking solid medications (pills or capsules).
B. Drink at least 1 cup of liquid to help swallow pills.
C. When over-the-counter antacids and other medicines stop making your symptoms better, call the office to make an appointment for medications that decrease stomach acid.

You Have Been Prescribed: _____

You Need to Take: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. No relief from antacids or second medication, so that the next step of medication therapy may be prescribed.
B. New symptoms, such as blood in regurgitated stomach contents.
C. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

HEMORRHOIDS

PROBLEM A hemorrhoid is a varicose vein of the rectum. You may not know you have a hemorrhoid until bleeding occurs. Hemorrhoids are often found after painless rectal bleeding with a bowel movement (BM). They may cause rectal pain, itching, or a sensation that you have not emptied completely after a BM.

CAUSE Repeated pressure in the anal or rectal veins causes hemorrhoids. Hemorrhoids are commonly seen with obesity, pregnancy, constipation, lack of exercise, and liver disease.

PREVENTION

A. Avoid heavy lifting.
B. Try to prevent constipation or straining.
C. Eat a high-fiber diet.
D. Drink 8 to 10 glasses of water a day.
E. Lose weight if you are overweight.
F. Exercise regularly.

TREATMENT PLAN

A. Spend less time sitting on the toilet to reduce pressure in the veins around the anus.
B. Pat with toilet paper instead of rubbing.
C. Take sitz baths or soak in a warm bathtub three or four times a day for comfort.
D. Apply cold packs or witch hazel (Tucks) compress for symptom relief.
E. Take a stool softener two to three times a day to prevent straining, then take one a day.
F. A hemorrhoid treatment may need to be done for severe cases.

Activity: No restrictions are required. Bowel function improves with good physical activity.

Diet: A high fiber diet helps to prevent constipation.

TIPS FOR INCREASING FIBER IN YOUR DIET

A. Read the Nutrition Facts food labels for fiber content per food serving:
    1. Cereals that provide 5 g of fiber per serving give you 20% of your daily fiber.
    2. Look for "whole grain" on the label. Just because bread is brown does not mean it is whole grain.
B. Increase fiber in your diet gradually to prevent gas. Adding too much fiber too quickly may give you abdominal pain, bloating, and constipation. Increase your fiber over several weeks so that it gives time for you to adjust.
C. As you increase fiber, it is also important to increase the amount of fluids you drink up to six to eight glasses a day, including tea, milk, fruit juices, coffee, and even soft drinks. The extra fluids that you drink along with the extra fiber make you feel fuller, which can help control snacking.
D. Keep a food diary and review it periodically to decide on other diet adjustments that need to be made.
E. Several fiber supplements are available over the counter to help you get your daily recommendation of fiber.

GOOD FOOD SOURCES OF FIBER

A. Bran: Add one teaspoon of whole-grain bran to food three times a day, or take an over-the-counter fiber supplement such as psyllium (Metamucil), as directed.
B. Whole-grain cereals and breads: Eat oat, bran, multigrain, light, wheat, or rye breads rather than pure white bread or breads that list eggs as a major ingredient. Grains are not only a good source of fiber but also contain vitamins and minerals. Folic acid has been added to breads and cereals to help reduce neural tube defects.
C. Fresh or frozen fruits and vegetables: Citrus fruits are especially good sources of fiber. Eat raw or minimally cooked vegetables, especially squash, cabbage, lettuce, other greens, and beans. Leave the skins on fruits and vegetables; eating the whole fruit is better than drinking the juice. Whole tomatoes offer more fiber than peeling the skin off. The more colorful the fruit and vegetable

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CLIENT TEACHING GUIDE

HEMORRHOIDS (*CONTINUED*)

(dark green, reds, blue, yellow) the better; they are a good source of antioxidants that are good for the heart and the prevention of some cancers. Apples are a good source of both fiber and water.

D. Legumes (pods): Peas and beans are a good source of fiber. Add chickpeas and kidney beans to salads for extra fiber and flavor. Add baked beans as a delicious side item to your meal.

E. Coffee is another source of fiber.

F. Nuts are an excellent source of fiber. They are considered nutrient-dense and are a good source of vitamins and folic acid. Sprinkle sunflower seeds on a salad to add flavor and fiber. The amount of nuts eaten should be limited to 1 to 2 oz because they are also high in calories.

Medications: _____

You Have Been Prescribed the Following Stool Softener: _____

You Need to Take: _____

You Have Been Prescribed the Following Local Cream: _____

You Need to Apply: _____

Emergent Instructions:

A. Had a procedure to remove a hemorrhoid and a hard lump develops where the hemorrhoid has been.

B. Excessive rectal bleeding needs to be evaluated.

You Need to Notify the Office If You Have:

A. Hemorrhoids that cause severe pain that is not relieved by the previous treatment.

B. Excessive rectal bleeding: more than a trace or streak on the toilet paper or BM.

C. Rectal bleeding may be an early sign of cancer.

D. Other: _____

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CLIENT TEACHING GUIDE

IRRITABLE BOWEL SYNDROME (IBS)

PROBLEM Irritable bowel syndrome (IBS) is an irritative and inflammatory disorder of the intestine. You may have diarrhea, constipation, or problems with alternating constipation and diarrhea.

CAUSE IBS is **not contagious.** IBS can flare with severe stress and may also be triggered by eating certain foods.

PREVENTION

A. Try to reduce stress or modify your response to it. Keep a stress diary to avoid stress triggers.
B. Good food habits help. Eat healthier foods that do not cause irritation and foods that are less likely to bother your stomach.
C. Keep a food diary to identify and avoid your food triggers.
D. Change your diet by decreasing carbohydrates such as fructose, fructans, lactose, and those called FODMAPs (fermentable oligosaccharides, disaccharides, monosaccharides, and polyols). FODMAPs are also found in certain grains, vegetables, fruits, and dairy products. See Appendix B, "Fermentable Oligosaccharides, Disaccharides, Monosaccharides, and Polyols (FODMAP) Diet."
E. When your IBS symptoms are getting better, reintroduce one food at a time to see if it makes your IBS symptoms come back or get worse:
1. After you feel that the new food does not cause increased IBS symptoms, try a second food.
2. Foods that make your IBS symptoms worse should continue to be avoided.

TREATMENT PLAN

A. Quit smoking: Nicotine may contribute to the problem. **It is never too late to stop smoking.**
B. Apply a warm heat compress to your abdomen for comfort.

Activity: Exercise, such as walking 20 minutes a day, improves bowel function and helps reduce stress. Other stress-reduction techniques include self-hypnosis and biofeedback.

Diet:

A. Eat a high-fiber diet. Fiber is good for both diarrhea and constipation.
B. Avoid sorbitol-containing (sugar-free) candies and gum as well as lactose-containing milk products to see if this eases your diarrhea symptoms.
C. Do not eat or drink anything that aggravates your symptoms. Common triggers may include coffee and spicy and gas-producing foods.
D. Avoid large meals, but eat regularly.
E. Limit alcohol intake to avoid symptoms.

Medications:_____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Fever.
B. Black or tarry-looking bowel movements.
C. Vomiting.
D. Unexplained weight loss of 5 lbs or more.
E. Symptoms that do not get better with changes in diet, exercise, and medication.
F. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

IRRITABLE BOWEL SYNDROME (IBS) (*CONTINUED*)

FODMAP DIET **Low FODMAP foods (good to eat):**

A. Vegetables (bean sprouts, kale, tomato, zucchini, spinach, eggplant, carrots).
B. Legumes (eggs, nuts, seeds, tofu).
C. Fruits (higher glucose than fructose); eat only one serving per meal, 3 oz (bananas, blueberries, pineapple, strawberries, oranges, mandarins, kiwi).
D. Meats, poultry, and meat substitutes.
E. Fish and seafood.
F. Cereals, grains, breads, biscuits, pasta, nuts, and cakes (brown rice, oats, polenta, quinoa, and tapioca).
G. Condiments, dips, sweets, sweeteners, and spreads (glucose, maple syrup, sugar, artificial sweeteners including aspartame, saccharin, and stevia).
H. Dairy foods and eggs (cheddar cheese, feta cheese, Parmesan cheese, cream, lactose-free milk).
I. Beverages (black tea, coffee, gin, green tea, vodka, water, white tea).

High FODMAP foods (avoid eating):

A. Wheat (main ingredient in food), barley, and rye.
B. Garlic.
C. Onions.
D. Fruits (higher in fructose than glucose, including apples, apricots, peaches, pears, watermelon, plums, nectarines, mangoes, cherries).
E. Vegetables (asparagus, Brussels sprouts, artichokes, leeks, mushrooms, snow peas, cauliflower).
F. Legumes (baked beans, black-eyed peas, butter beans, kidney beans, lentils, soybeans, split peas).
G. Sweeteners (agave nectar, high-fructose corn syrup, honey, sugar-free gum/mints).
H. Dairy (cottage cheese, cream cheese, milk, ricotta, yogurt).
I. Beverages (chai tea, chamomile tea, coconut water, dessert wine, and rum).

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CLIENT TEACHING GUIDE

JAUNDICE AND HEPATITIS

PROBLEM Jaundice is a yellow tinge of the skin. Your blood contains too much bilirubin—a yellow pigment found in bile, which is a fluid secreted by the liver.

Other symptoms that occur with jaundice are dark urine, light-colored bowel movements (BMs), fatigue, fever, chills, loss of appetite, nausea, and vomiting.

CAUSE Jaundice usually comes from a liver disorder, such as cirrhosis, hepatitis, or a disease of the gallbladder or pancreas. Sometimes jaundice results from taking a drug that damages the liver.

PREVENTION Although hepatitis is considered contagious, you do not need to stay in your home. To help prevent the spread of hepatitis:

A. Do not prepare or handle food for others until cleared by your healthcare provider.
B. Wash your hands well after using the toilet and changing diapers.
C. If you have hepatitis A or B, avoid intimate sexual contact until cleared by your healthcare provider.
D. Hepatitis A and B can be killed by cleaning household surfaces with a solution of 1:10 dilution of bleach to water.
E. If you have hepatitis B or C, do not share razors, toothbrushes, and other personal items.
F. Do not share personal equipment, such as glucose testing/injection equipment.
G. Cover open cuts and scratches.
H. Never donate blood, organs, or sperm after a hepatitis B or C infection.
I. Your family and your sexual partners need evaluation for hepatitis A or hepatitis B vaccinations.

TREATMENT PLAN Treatment for jaundice includes the following:

A. Use good hygiene techniques with bathing, using the bathroom, and handwashing.
B. Apply anti-itch lotions, such as calamine.
C. Rest.
D. Make a list of all of the medications, over-the-counter medications, and herbal products you are taking so that you and your healthcare provider can see if any of them are causing the jaundice.
E. A procedure may need to be done to crush a gallstone blocking the bile duct, causing jaundice.
F. If you have hepatitis, you will be referred to a specialist and the public health department will be notified by your healthcare provider.
G. Your healthcare specialist may discuss the need for a liver biopsy and treatment for the virus.
H. Avoid alcohol. Alcohol is associated with an increase of cirrhosis and hepatocellular carcinoma (HCC).

Activity: Plan rest periods throughout the day. Avoid strenuous exercise. Gradually resume activities and mild exercise during this time.

Diet: Eat small, frequent, low-fat, high-calorie meals. You may be instructed to limit protein during acute phases of some types of hepatitis. Sit down to eat to decrease pressure on your liver. Drink 8 to 10 glasses of liquids a day.

Medications:_____

You Have Been Prescribed: _____

You Need to Take: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Mild confusion.
B. Personality changes.
C. Worsening symptoms.

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CLIENT TEACHING GUIDE

JAUNDICE AND HEPATITIS (*CONTINUED*)

D. Tremors.
E. Other:_____

Phone: _____

RESOURCES Centers for Disease Control and Prevention: www.cdc.gov/hepatitis

Hepatitis B information is available from the Hepatitis B Foundation: www.hepb.org

Hepatitis C information is available from the American Liver Foundation: https://liverfoundation.org/for-patients/about-the-liver/diseases-of-the-liver/hepatitis-c.

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CLIENT TEACHING GUIDE

LACTOSE INTOLERANCE AND MALABSORPTION

PROBLEM You have problems digesting milk (lactose) products or have problems with malabsorption. Lactose intolerance can cause gas, bloating, abdominal cramps, diarrhea, and nausea or vomiting. You may be able to eat small portions without problems or be unable to tolerate any foods containing milk.

CAUSE Lactose is the sugar present in milk. Lactose intolerance is very common; it occurs when the body is not able to digest this milk sugar content and causes diarrhea.

PREVENTION Follow a lactose-free or lactose-controlled diet.

Activity: No restrictions are required. Resume normal activities as soon as diarrhea symptoms improve. You may be sent to see a registered dietitian to assist in your dietary plan.

Diet:

A. Limit or omit foods that contain milk, lactose, whey, or casein.
B. Lactose-controlled diets allow up to one cup of milk per day for cooking or drinking, if you can tolerate it.
C. If you cannot tolerate any lactose, choose lactose-free foods with lactate, lactic acid, lactalbumin, whey protein, sodium caseinate, casein hydrolysates, and calcium compounds. Read labels carefully.
D. You may also choose kosher foods marked "pareve" or "parve," which do not contain lactose. Read labels carefully.
E. A low-fat diet is important if you have fat malabsorption.
F. To help with diarrhea caused by malabsorption, avoid more than one serving a day of caffeine-containing drinks.
G. Beverages with high sugar content, such as soft drinks and fruit juices, may increase diarrhea. Juices and fruits with high amounts of fructose include apples, pears, sweet cherries, prunes, and dates.
H. Sugar-free (Sorbitol) candies and gums may cause diarrhea.

Medications: You may need the enzyme lactase to help you digest your food. You may need vitamins and minerals if you are having problems with malabsorption/diarrhea.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Severe abdominal pain.
B. Diarrhea causing dehydration.
C. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

MANAGEMENT OF ULCERS

PROBLEM An ulcer is a sore in the lining of the stomach or intestine due to stomach acid.

CAUSE The cause of ulcers is not fully understood. A bacteria called *Helicobacter pylori* and certain drugs have been identified as possible causes.

PREVENTION Modify your lifestyle to include health practices that prevent recurrences of ulcer pain and bleeding.

TREATMENT PLAN

A. If aspirin or ibuprofen is suspected as the cause of the ulcer, you will have to stop taking it. If you need the medicine for other health problems, your healthcare provider will help with a new dose or therapy.
B. Avoid caffeine, colas, alcohol, and chocolate because they may increase acid production.
C. Stop smoking: It decreases the ulcer's healing rate and increases its recurrence. **It is never too late to stop smoking.**
D. Be sure to tell healthcare providers about your history of an ulcer and stomach pain if you need new prescriptions or are sent to the hospital.
E. Make a list of all of your medications and over-the-counter and herbal products so that you and your healthcare provider can review your list for possible causes or ulcer irritants.

Activity: Exercise daily. Plan rest periods, avoid fatigue, and learn to cope with or avoid stressful situations.

Diet:
A. Eat a well-balanced diet with high fiber content.
B. Eat meals at regular intervals. Frequent small feedings are unnecessary. Avoid bedtime snacks.
C. Eliminate foods that cause pain or distress; otherwise, your diet is usually not restricted. Examples of foods that cause worse pain are the following:
1. Peppermint.
2. Spicy food.
3. Alcohol.

D. Avoid extremely hot or cold food or fluids, chew thoroughly, and eat slowly and while relaxed for better digestion.

Medications:
A. Some of the medicines that help the pain and acid can be purchased at the drugstore without a prescription. H2 blockers and proton pump inhibitors (PPIs) reduce stomach acid.
B. Antibiotics fight *H. pylori* infection.
C. Other medications may be prescribed to coat the ulcer area. Antacids can be taken during the ulcer treatment but should not be used 1 hour before or 2 hours after the ulcer treatments because antacids can interfere with absorption.
D. Take the entire prescription: Do not stop when you feel better.
E. If you have been prescribed metronidazole (Flagyl) or clarithromycin, you may notice a metallic taste in your mouth.
F. Alcohol (including wine) should be avoided when taking Flagyl. The interaction can cause skin flushing, headache, nausea, and vomiting.
G. If you are prescribed bismuth, you may notice black bowel movements (BMs).

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. Worsening symptoms while taking your medication.
B. Vomiting that is bloody or looks like coffee grounds.
C. Tar-colored or "grape jelly" BMs. If this occurs, bring a stool sample to the office.
D. Diarrhea and/or severe pain despite treatment.
E. Unusual weakness or paleness.
F. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

TIPS TO RELIEVE CONSTIPATION

PROBLEM Constipation is an infrequent and difficult passing of hard stools and sensation of incomplete emptying or straining.

CAUSE There are many reasons why you have constipation. You may have just one or a combination of causes:

A. Not drinking enough liquids.
B. Eating a low-fiber diet.
C. Sedentary lifestyle, lack of exercise.
D. Ignoring the urge to go to the bathroom.
E. Taking drugs, including blood pressure medications, antidepressants, pain medications, and antacids, or overusing laxatives.
F. Depression.
G. Other medical conditions.

PREVENTION

A. Go to the bathroom as soon as you have the urge to have a bowel movement (BM). Do not wait.
B. Make a regular toilet time, such as after breakfast; 15 to 20 minutes after breakfast is a good time because spontaneous movement is greatest during this period.
C. Use a footrest during elimination to provide support and decrease straining.
D. Do not rely on laxatives; use prune juice as a natural substitute.
E. Stimulate the intestine by drinking hot or cold water or prune juice before eating.
F. Decrease eating sweets, which increase bacterial growth in the intestine and can lead to gas.
G. Make a list of all of your medicines, over-the-counter medicines, and herbals so that you and your healthcare provider can review them and discuss what can be stopped.

TREATMENT PLAN

A. Follow the suggested prevention tips.
B. Change to a high-fiber diet.
C. Get daily exercise.

Activity: Daily exercise such as walking helps to maintain healthy bowel patterns.

Diet:

A. Eat a high-fiber diet.
B. Restrict cheese: It causes constipation.
C. Drink at least eight glasses of water each day.
D. Avoid refined cereals and breads, pastries, and sugar.
E. Coffee, tea, and alcohol decrease water to the colon. Limit to two drinks per day.

Medications: You may be prescribed a stool softener or a laxative for short-term use only. You may be prescribed a bulk-forming agent to take on a regular basis to increase the bulk of your stool.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Fever.
B. Severe abdominal pain.
C. Nausea and vomiting.
D. Not had a BM and you are not able to pass gas.
E. Constipation that continues after diet changes and exercise.
F. Noticed blood in your BM.
G. Other: _____

Phone: _____

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CHAPTER 15

GENITOURINARY GUIDELINES

Kristina A. Potts and Nancy Pesta Walsh

BENIGN PROSTATIC HYPERPLASIA

DEFINITION

A. Benign prostatic hyperplasia (BPH) is enlargement of the prostate gland that constricts the urethra causing urinary symptoms. BPH is not believed to be a risk factor for prostate cancer. BPH occurs primarily in the central or transitional zone of the prostate, while prostate cancer originates primarily in the peripheral part of the prostate.

B. The voiding dysfunction that results from prostate enlargement and bladder outlet obstruction (BOO) are termed lower urinary tract symptoms (LUTS).

INCIDENCE

A. BPH increases progressively with age. The prevalence of prostatic hyperplasia increases from 8% in males aged 31 to 40 years to 40% to 50% in males aged 51 to 60 years, to over 80% in males older than 80 years.

PATHOGENESIS

A. The exact cause is unknown; BPH may be a response to the androgen hormone. The process of aging and the presence of circulating androgens are required for the development of BPH. Hyperplasia, in which the normally thin and fibrous outer capsule of the prostate becomes spongy and thick, and the contraction of muscle fibers cause pressure on the urethra. This pressure requires the bladder musculature to work harder to empty urine.

PREDISPOSING FACTORS

A. Advancing age.

B. Race: African American males younger than 65 years of age may need treatment more often than White males.

C. Genetic predisposition: increases with a positive family history of BPH having moderate to severe LUTS.

D. Obesity.

E. Diabetes.

F. High levels of alcohol consumption.

G. Physical inactivity.

COMMON COMPLAINTS

A. The clinical manifestations of BPH are LUTS that typically appear slowly and progress gradually over a period of years:

1. Difficulty starting urine flow.
2. Dribbling.
3. Bladder that does not feel it completely empties.
4. Frequency of urination.

OTHER SIGNS AND SYMPTOMS

A. Obstructive symptoms:

1. Hesitancy.
2. Diminution in size and force of urinary stream.
3. Stream interruption (double voiding).
4. Urinary retention.
5. Straining/Valsalva maneuver to fully empty the bladder.

B. Irritative voiding symptoms:

1. Urgency.
2. Frequency.
3. Nocturia.
4. Painless hematuria: an early symptom; may also indicate malignancy.

C. Severe late symptoms with untreated BPH:

1. Acute urinary retention.
2. Recurrent urinary tract infections (UTIs).
3. Hydronephrosis.
4. Loss of renal concentrating ability.
5. Systemic acidosis and renal failure.

SUBJECTIVE DATA

A. Have the client complete the American Urological Association Symptom Score (AUASS) assessment tool at each visit to track symptoms. The AUASS (Table 15.1) is used to assess the severity of symptoms of BPH.

B. Review the onset, duration, and course of symptoms. The AUASS assessment tool can be used to quantitatively assess BPH symptoms over time.

C. Does the client have signs of a UTI?

D. Is there any blood in the urine or pain in the bladder region? (Evaluate bladder tumor or calculi.)

E. Does the client have new symptoms such as bone or back pain, loss of appetite, or weight loss (rules out cancer)?

F. Review the client's history for medical illness, including diabetes and neurologic problems.

G. Review previous urinary problems, surgeries, infections, treatments, success of treatments, and testing.

H. Review the client's history of sexual dysfunction and any new sexual partners (sexually transmitted infections [STIs]).

I. Review the client's history of urethral trauma, urethritis, or urethral instrumentation that could have led to urethral stricture.

J. Review medications, both prescription and over-the-counter (OTC) drugs, including sinus or cold products, anticholinergic drugs (for impaired bladder function), and sympathomimetic drugs (to increase outflow resistance). ▶

The contributions of Cheryl A. Glass to this chapter in prior editions are acknowledged here.

TABLE 15.1 AMERICAN UROLOGICAL ASSOCIATION SYMPTOM SCORE

PATIENT NAME:_____ **TODAY'S DATE:** _____

(Circle One Number on Each Line)	Not at All	Less Than 1 Time in 5	Less Than Half the Time	About Half the Time	More Than Half the Time	Almost Always
Over the past month or so, how often have you had a sensation of not emptying your bladder completely after you finished urinating?	0	1	2	3	4	5
During the past month or so, how often have you had to urinate again less than 2 hours after you finished urinating?	0	1	2	3	4	5
During the past month or so, how often have you found you stopped and started again several times when you urinated?	0	1	2	3	4	5
During the past month or so, how often have you found it difficult to postpone urination?	0	1	2	3	4	5
During the past month or so, how often have you had a weak urinary stream?	0	1	2	3	4	5
During the past month or so, how often have you had to push or strain to begin urination?	0	1	2	3	4	5
	None	**One Time**	**Two Times**	**Three Times**	**Four Times**	**Five or More Times**
Over the past month, how many times per night did you most typically get up to urinate from the time you went to bed at night until the time you got up in the morning?	0	1	2	3	4	5
Add the score for each number above and write the total in the space to the right.				**TOTAL:** _____		
SYMPTOM SCORE: 0–7 (Mild) 8–19 (Moderate) 20–35 (Severe)						
QUALITY OF LIFE (QOL)						

	Delighted	Pleased	Mostly Satisfied	Mixed	Mostly Dissatisfied	Unhappy	Terrible
How would you feel if you had to live with your urinary condition the way it is now, no better, no worse, for the rest of your life?	0	1	2	3	4	5	6

Source: Used with permission from the American Urological Association. www.auanet.org.

K. Review family history of BPH and prostate cancer.

L. Review fluid intake, especially caffeinated/carbonated drinks.

M. Evaluate how bothersome the symptoms are to the client's quality of life:

1. How often does the client have interrupted sleep to get up to go to the bathroom?

2. How often does the client urinate?

3. Does the client have to wear an absorptive underwear pad?

PHYSICAL EXAMINATION

A. Check temperature (if indicated), blood pressure (BP), and weight (if indicated).

B. Inspect:

1. Evaluate general appearance for discomfort or acute discomfort with urinary retention.

2. Consider having the client void: Normal urination for a male is the ability to empty the bladder of 300 mL of urine in 12 to 15 seconds.

3. Examine the urethral meatus for discharge. ▶

4. Retract foreskin (if present) and assess for hygiene and smegma.
5. Check the shaft of the penis, glans, and prepuce for lesions.
6. Check inguinal and femoral areas for bulges or hernias; have the client bear down and cough and reexamine them.
7. Perform a neurologic examination (evaluate sensory and motor deficits).

C. Palpate:
1. Palpate the abdomen for masses or bladder distension.
2. Palpate lymph nodes in the groin for enlargement.
3. Check costovertebral angle (CVA) tenderness.
4. Palpate the testes and epididymides for inflammation, tenderness, and masses.
5. Palpate the scrotum for hydrocele or varicocele.

D. Digital rectal exam (DRE): Use the index finger of the dominant hand for the DRE:
1. Note sphincter tone, nodules or masses, and tenderness. **Decreased anal sphincter tone or the lack of muscle reflex may indicate an underlying neurologic disorder.**
2. Palpate the two lateral lobes of the prostate gland and its median sulcus for irregularities, nodules, induration, swelling, or tenderness just above the prostate anteriorly; determine whether the rectum lies adjacent to the peritoneal cavity. If possible, palpate this region for peritoneal masses and tenderness.
3. The U.S. Preventive Services Task Force (USPSTF) does not recommend DRE as a screening test in primary care due to lack of evidence on the benefits.

DIAGNOSTIC TESTS

A. Urinalysis: Evaluate for infection and hematuria.
B. Urine culture if indicated (clients with BPH are more susceptible to UTIs).
C. Optional studies:
1. Prostate-specific antigen (PSA): Reference ranges vary by age and ethnicity and may be elevated with BPH.
2. Urodynamic testing, including maximal urinary flow rate.
3. Postvoid residual (PVR; as shown by in–out catheterization, radiography, or ultrasound).
4. Cystourethroscopy (assists in planning for surgical therapy).
D. The American Urological Association (AUA) recommends that the routine measurement of serum creatinine levels is *not* indicated in the initial evaluation.

DIFFERENTIAL DIAGNOSES

A. BPH: Classifications of BPH from the score of the AUA symptom assessment tool:
1. Mild = total AUASS 0 to 7.
2. Moderate = total AUASS 8 to 19.
3. Severe = total AUASS 20 to 35.
B. Other obstructive causes: prostate cancer, urethral obstruction, urethral stricture, and vesical neck obstruction.
C. Neurogenic bladder.
D. Cystitis.
E. Prostatitis.
F. Bladder calculi.

PLAN

A. General interventions:
1. Have the client complete a 24-hour voiding chart with assessment of frequency and volume.
2. Any client with other than mild symptoms needs referral to a urologist to discuss treatment options (surgery or drugs).
3. Monitor the client with mild symptoms every 3 to 6 months to determine the progression of symptoms. Imaging studies are not routinely necessary in typical cases of BPH unless there is hematuria, an elevated creatinine, or another indication.
4. Treat concurrent UTI and STIs.

B. Client teaching: *See Client Teaching Guide for this chapter, "Benign Prostatic Hyperplasia."*
1. Clients should be instructed about the hypotensive effect, asthenia, nasal congestion, and effect on ejaculation of the long-acting alpha-1 antagonists. The hypotensive effects can be potentiated by concomitant use of phosphodiesterase-5 (PDE5) inhibitors sildenafil (Viagra), avanafil (Stendra), tadalafil (Cialis), or vardenafil (Levitra).
2. Alpha-1 antagonists have been associated with intraoperative floppy iris syndrome. Clients need to discuss using these meds with their ophthalmologist prior to eye surgery (e.g., cataract).

C. Pharmaceutical therapy:
1. Four long-acting alpha-1 antagonists are Food and Drug Administration (FDA)-approved for treatment of BPH:
a. Terazosin (Hytrin) initial: 1 mg at bedtime, increasing as needed; most clients require 10 mg/d. If no response after 4 to 6 weeks of 10 mg/d, may increase to 20 mg/d. Side effect increases hypotensive effect with PDE5 inhibitor. Terazosin requires dose titration to minimize side effects.
b. Doxazosin (Cardura) goal: 4 to 8 mg/d; maximum dose 8 mg/d. Start 1 mg/d in the morning or evening; may be increased to 2 mg/d. Thereafter titrate upward, if needed, over several weeks, balancing therapeutic benefit with doxazosin-induced postural hypotension. Side effect increases hypotensive effect with PDE5 inhibitor. Doxazosin requires dose titration to minimize side effects.
c. Tamsulosin (Flomax): 0.4 mg/d; dose may be increased after 2 to 4 weeks to 0.8 mg/d. Side effect decreases ejaculate volume. Tamsulosin capsules are not intended for use as an antihypertensive.
d. Alfuzosin (Uroxatral): 10 mg/d; it generally does not cause ejaculation problems. Dosage adjustment in renal impairment: Bioavailability and maximum serum concentrations are increased by 50% with mild, moderate, or severe renal impairment.
2. Two 5-alpha-reductase inhibitors are FDA-approved for BPH with an enlarged prostate. The major side effects of these drugs are decreased libido and ejaculatory or erectile dysfunction (ED):
a. **The FDA has advised about safety information for use of 5-alpha-reductase inhibitors due to an increased risk of being diagnosed with a more serious form of prostate cancer (high-grade prostate cancer).**
b. **Individuals who are, or may become, pregnant should not handle the 5-alpha-reductase inhibitors. They are pregnancy category X, known to cause birth defects.**
c. Finasteride (Proscar): 5 mg/d as a single dose. Clinical responses occur within 12 weeks to 6 months of initiation of therapy.
d. Dutasteride (Avodart): 0.5 mg/d.

3. Other drugs for treatment of BPH:
   a. Alfuzosin (Uroxatral): 10 mg/d. Bioavailability and maximum serum concentrations are increased by 50% with mild, moderate, or severe renal impairment.
   b. Silodosin (Rapaflo): 4 to 8 mg/d (4 mg/d for moderate renal impairment, creatinine clearance 30–50 mL/min). Silodosin is an alpha-1 adrenergic receptor antagonist for treatment of BPH and is not indicated for treatment of hypertension (HTN). Side effects may cause retrograde ejaculation.
4. Dual–drug combination:
   a. Dutasteride–tamsulosin (Jalyn): Each capsule contains 0.5 mg dutasteride and 0.4 mg tamsulosin.
5. There are no herbal supplements that have been approved by the FDA for treatment of BPH; however, clients may report taking saw palmetto. The AUA does not endorse supplements.

FOLLOW-UP

A. See the client in 2 to 3 weeks to monitor symptoms after specialty referral.

B. PSA testing:
1. Baseline testing should begin at age 40.
2. The USPSTF recommends that PSA testing be discontinued at age 70, including African American males and males with a family history of prostate cancer.
3. PSA should not be measured in older adult males if life expectancy is less than 10 years.

C. PSA is an amino acid glycoprotein specific to prostate disease, but not exclusive to prostate cancer. **Once the client has exhibited an elevated PSA level, repeat the test yearly. Clients who have undergone treatment for prostate cancer are monitored for recurrence by the PSA levels.**

D. The concept of "watchful waiting" may be appropriate for clients with mild symptoms. The client should be seen yearly for evaluation and an examination.

CONSULTATION

A. Refer to a urologist for any complicated LUTS, including:
1. History of prostate cancer.
2. Elevated PSA.
3. Urethral stricture.
4. Spinal cord injury.
5. Stroke.
6. Recurrent/persistent UTI.

B. The presence of microscopic hematuria requires an evaluation of the complete urinary system and a referral to a urologist.

C. Refer for a procedure after failed medication therapy:
1. Minimally invasive therapy:
   a. Transurethral microwave therapy (TUMT).
   b. Transurethral incision of the prostate (TUIP).
2. Surgical therapies:
   a. **Transurethral resection of the prostate (TURP) is considered the gold standard of surgical treatment of BPH.** Sexual dysfunction may occur after a TURP, including decreased libido, impotence, and ejaculatory difficulties. Balloon dilation may be used to reduce symptoms; however, relapse is common.
   b. Open prostatectomy.
   c. Laser procedures:
      i. Laser vaporization of the prostate.
      ii. Laser enucleation of the prostate.
   d. Transurethral needle ablation (TUNA).
   e. Photoselective vaporization of the prostate (PVP).

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. Elderly males require special attention because their symptoms may be poorly expressed or confusing. Always screen for UTI if these are newly reported symptoms.
2. Geriatric syndrome assessments:
   a. Incontinence: In clients with signs and symptoms of BPH, assess for incontinence, especially related to nighttime frequency. A validated survey tool is the International Consultation on Incontinence Questionnaire (ICIQ). The ICIQ Short Form for urinary incontinence (UI) survey can be requested from www.iciq.net/background.htm.
   b. Falls: Consistently assess for falls. Educate clients that incontinence and urinary frequency have a strong association with falls in elderly males. Avoid use of diuretics in the 4 hours prior to bedtime (caffeine, alcohol, diuretic medications). Avoid fluids 1 to 2 hours before bedtime. Safety measures to reduce falls in the home include removing small area carpets, keeping night lights along the path to the bathroom, and installing a toilet seat with grab bars or nearby grab bars (search Americans with Disabilities Act bathroom grab bar for installation information).
3. Pharmacologic caution in geriatrics:
   a. First-generation alpha-1 blockers are linked with falls in older adults due to orthostatic hypotension. Educate older males to use caution, getting up slowly from seated or lying positions. Avoid first-generation alpha-1 blockers (Hytrin, Cardura) in older males with a history of syncope. Avoid using antimuscarinics in frail older adults, especially those with persistent incontinence because the risk from anticholinergic effects (sedation, delirium/confusion/dementia, constipation, dryness) outweighs the benefit.
4. Beers list caution: Avoid use of desmopressin for nocturia or nocturnal polyuria due to high risk of hyponatremia in older adults. This is a potentially inappropriate medication to use in the elderly; safer alternative treatments are available.

RESOURCES

American Urological Association (AUA): www.auanet.org

National Kidney Urologic Diseases Information Clearinghouse (NKUDIC): kidney.niddk.nih.gov/health-information/communication-programs/information-clearinghouses

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CHRONIC KIDNEY DISEASE

DEFINITION

A. Chronic kidney disease (CKD) is defined as an abnormality of kidney structure or function that is present for greater than 3 months. CKD is specifically defined as follows:

1. The persistent and usually progressive reduction in glomerular filtration rate (GFR) less than 60 mL/min/1.73 m^2. and/or

2. Albuminuria: more than 30 mg of urinary albumin per gram of urinary creatinine.

3. CKD is a disorder that leads to progressive kidney damage from a variety of causes, including diabetes, hypertension (HTN), cardiovascular (CV) disease, urinary obstructions, prolonged use of nephrotoxic medications, and inherited diseases such as polycystic kidney disease. Associated comorbidities of CKD include renal osteodystrophy, anemia, metabolic acidosis, and malnutrition. Early recognition of CKD as well as treatment of complications can improve long-term outcomes. The five stages of CKD are:

a. Stage 1: kidney damage with normal kidney function (estimated glomerular filtration rate [eGFR] ≥90 mL/min/1.73 m^2) and persistent (≥3 months) proteinuria.

b. Stage 2: kidney damage with mild loss of kidney function (eGFR 60–89 mL/min/1.73 m^2) and persistent (≥3 months) proteinuria.

c. Stage 3: mild to severe loss of kidney function (eGFR 30–59 mL/min/1.73 m^2).

d. Stage 4: severe loss of kidney function (eGFR 15–29 mL/min/1.73 m^2).

e. Stage 5: kidney failure requiring dialysis or transplant for survival. Also known as end-stage renal disease (ESRD; eGFR <15 mL/min/1.73 m^2).

INCIDENCE

A. Kidney disease kills more people than breast or prostate cancer. The overall prevalence of CKD in the general population is 14%.

B. CKD is more common in people aged 65 years or older (38%) than in people aged 45 to 64 years (12%) or 18 to 44 years (6%).

C. CKD is slightly more common in females (14%) than in males (12%).

D. CVD contributes to more than half of all deaths among clients with ESRD.

E. African Americans are about 3.5 times more likely to develop ESRD than whites. Hispanics are about 1.5 times more likely to develop ESRD than non-Hispanics.

F. CKD prevalence is greater among persons with diabetes than among those without diabetes (40.2% vs. 15.4%) and among persons with CV disease than among those without CV disease (28.2% vs. 15.4%).

G. CKD is higher among persons with HTN than among those without HTN (24.6% vs. 12.5%).

PATHOGENESIS

A. Blood from the renal arteries and their subdivisions is delivered to the glomeruli. The glomeruli form an ultrafiltrate, nearly free of protein and blood elements, which subsequently flows into the renal tubules. The tubules reabsorb and secrete solute and/or water from the ultrafiltrate. The final tubular fluid, the urine, leaves the kidney, draining sequentially into the renal pelvis, ureter, and bladder, from which it is excreted through the urethra. The causes of CKD are traditionally classified by which portion of the renal anatomy is most affected by the disorder.

1. Vascular disease: Vascular disorders of the kidneys may involve partial or complete occlusion of large, medium, or small renal vessels.

a. Renal artery stenosis: narrowing or blockage of an artery to the kidneys.

b. Renal artery thrombosis: a blood clot in a renal artery.

c. Renal vein thrombosis: a bulging, weak area in the wall of a renal artery.

d. Atheroembolic renal disease: when a piece of plaque from a larger artery breaks off and travels through the blood causing blockage to the small renal arteries.

e. Benign hypertensive arteriolar nephrosclerosis results when chronic HTN damages small blood vessels, glomeruli, renal tubules, and interstitial tissues.

f. Glomerulosclerosis is a severe microvascular or small vessel kidney disease caused by diabetes and uncontrolled HTN in which glomerular function of blood filtration is lost as fibrous scar tissue replaces the glomeruli. This leads to proteinuria, hematuria, HTN, and nephrosis with variable progression to ESRD. Proteinuria occurs due to changes to capillary endothelial cells, the glomerular basement membrane (GBM), or podocytes, which normally filter serum protein selectively by size and charge.

2. Tubular and interstitial disease: As with vascular disease, chronic tubulointerstitial nephritis (CTIN) can be primary or secondary to glomerular damage and renovascular disease. CTIN arises when chronic tubular insults cause gradual interstitial infiltration and fibrosis, tubular atrophy and dysfunction, and a gradual deterioration of renal function, usually over years. Causes of CTIN are immune disorders, infections, reflux or obstructive nephropathy, and drugs. Analgesic abuse nephropathy (AAN) is a type of CTIN caused by cumulative lifetime use of large amounts of certain analgesics such as nonsteroidal anti-inflammatory drugs (NSAIDs).

PREDISPOSING FACTORS

A. Diabetes.

B. HTN.

C. CV disease.

D. Chronic use of analgesics such as NSAIDs.

E. Autoimmune disorder.

F. Polycystic kidney disease.

G. Urinary tract obstructions such as benign prostatic hyperplasia or kidney stones.

H. Recurrent urinary tract infections (UTIs).

I. Older than 60 years of age (age alone is not a reason for screening).

J. High-risk ethnic groups: African American, Native American, Pacific Islanders, Asian descent, or Hispanic smokers.

K. Exposure to toxins.

L. Family history of kidney disease.

COMMON COMPLAINTS

A. For CKD stages 1 and 2, there are usually no presenting complaints; however, HTN is usually present. CKD is usually identified through routine screening of kidney function and urine tests for microalbumin.

SIGNS AND SYMPTOMS

A. In CKD stage 3, the person will develop CKD complications but will still not usually have identifiable signs/symptoms:

1. HTN.
2. Decreased dietary calcium absorption.
3. Reduced renal phosphate excretion.
4. Elevation of parathyroid hormone.
5. Altered lipoprotein metabolism.
6. Reduced spontaneous protein intake.
7. Anemia.
8. Left ventricular hypertrophy.
9. Salt and water retention.
10. Decreased renal potassium excretion; elevated serum potassium >5.5 mEq/L.

B. Signs/symptoms gradually worsen as the person moves to stage 4 CKD. The person will begin to display signs and symptoms of complications:

1. Changes in bone density.
2. Fatigue and pallor related to anemia.
3. Edema.
4. Decreases in muscle mass.

C. As the CKD progresses to stage 5, in addition to gradual worsening of the signs/symptoms noted in stages 3 and 4, the person will experience symptoms indicating chronic uremia:

1. Impaired sleep.
2. Nocturia.
3. Fatigue.
4. Anorexia, nausea, vomiting, and weight change.
5. Decreased mental acuity.
6. Pruritus.
7. Edema.
8. Respiratory symptoms, including orthopnea and dyspnea.
9. Muscle cramps, twitching, and restless legs.
10. Peripheral neuropathy.

SUBJECTIVE DATA

A. Review the client's medical history to identify risk factors for CKD (previously noted in the section "Predisposing Factors").

B. Elicit information about how well risk factors such as diabetes mellitus (DM) and HTN are controlled.

C. Review onset and duration of signs/symptoms of CKD complications and uremia.

D. Review all medications, including all over-the-counter (OTC) medications, especially NSAIDs, and supplements. Obtain specific information about dosing and length of time the drug was used.

E. Elicit smoking history.

F. Determine if client was dehydrated during lab draw, this could cause a falsely low GFR. If suspected, repeat in 1 to 2 weeks and make sure client is fully hydrated.

PHYSICAL EXAMINATION

A. Observe general demeanor, attentiveness, and signs of fatigue.

B. Measure vital signs:

1. Vital signs, including orthostatic blood pressure (BP) and pulse.
2. Height and weight to calculate body mass index (BMI). A BMI calculator is located at www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/english_bmi_calculator/bmi_calculator.html:
   a. Record weight and BMI on each visit for comparison.

C. Inspect:

1. Skin for color, moisture, turgor, and signs of scratching caused by chronic pruritus.
2. Neck for jugular venous distension.
3. Abdomen for distension.
4. Extremities for edema, muscle mass, and signs of pain disorders such as arthritis.

D. Auscultate:

1. Lungs for rales.
2. Heart for cardiac heave, gallop, or rub.
3. Abdominal or femoral bruit.

E. Palpate:

1. Abdomen for masses, distension.
2. Bladder or flank for tenderness.

F. Neurologic exam:

1. Assess for sensation and vibratory sense on both feet.

DIAGNOSTIC TESTS

A. Age alone is not a reason for CKD screening.

B. Laboratory testing is critical in ascertaining the stage, course, chronicity, and complications (and associated comorbid conditions) of CKD.

C. Routine kidney function tests:

1. Serum creatinine.
2. Blood urea nitrogen (BUN).
3. Urinalysis.
4. eGFR is the best marker for CKD. Measuring GFR: The severity of CKD should be classified based on the level of the eGFR. Serum creatinine alone should *not* be used as a measure of kidney function. Kidney function in clients with CKD should be assessed by formula-based estimation of GFR (eGFR), preferably using the four-variable Modification of Diet in Renal Disease (MDRD) equation. Clinicians who do not have access to an automated tool may use the web-based tool at www.nkdep.nih.gov/professionals/gfr_calculators/index.htm. Calculate eGFRs using the actual MDRD equation: eGFR = 186 × [SCr] − 1.154 × [age] − 0.203 × [0.742 if female] × [1.210 if black], where SCr is serum creatinine concentration.

D. Assessing proteinuria:

1. When screening adults at increased risk for CKD, albumin in the urine should be measured in a spot urine sample using either:
   a. Albumin-specific dipstick. OR
   b. Albumin-to-creatinine ratio (ACR).
      i. It is usually not necessary to obtain a timed urine collection (overnight or 24 hours).
      ii. First morning specimens are preferred, but random specimens are acceptable if first morning specimens are not available.
      iii. In most cases, screening with urine sticks is acceptable for detecting proteinuria.
      iv. Standard urine dipsticks are acceptable for detecting increased total urine protein.
      v. Albumin-specific dipsticks are acceptable for detecting albuminuria.
2. Clients with a positive protein dipstick test (1 or greater) should undergo confirmation of proteinuria by ▶

a quantitative measurement (protein-to-creatinine ratio [PCR] or ACR) within 3 months.

3. Clients with two or more positive quantitative tests spaced apart by 1 to 2 weeks should be diagnosed as having persistent proteinuria and undergo further evaluation and management for CKD.

OTHER TESTS TO DETERMINE CHRONIC KIDNEY DISEASE COMPLICATIONS

A. Complete blood count (CBC).

B. If hemoglobin (Hgb) level is less than 12 g/dL in females, and Hgb levels are less than 13.5 g/dL in adult males, also do blood cell indices, absolute reticulocyte count, serum iron, total iron-binding capacity, percent transferrin saturation, serum ferritin, white blood cell (WBC) count and differential, platelet count, and testing for blood in stool.

C. Lipid profile and triglycerides.

D. Comprehensive metabolic profile (serum total protein, serum albumin, glucose, calcium, sodium, potassium, chloride, bicarbonate, BUN, creatinine, alkaline phosphatase, alanine aminotransferase [ALT], aspartate aminotransferase [AST], bilirubin).

E. Prealbumin.

F. Phosphorus.

G. Parathyroid hormone.

H. Urine immunofixation study.

I. Antinuclear antibody (ANA), antineutrophil cytoplasmic autoantibody (ANCA), and complement 3 and complement 4 (C3–C4):

1. Test is done to rule out autoimmune disorder, lupus, vasculitis, and cancer.

2. No fasting is needed.

J. Hepatitis B surface antigen and hepatitis C antibody.

K. Anti-GBM antibody.

L. Renal ultrasound to rule out postobstructive uropathy.

M. Renal Doppler to rule out renal artery stenosis.

DIFFERENTIAL DIAGNOSES

A. Evaluation is meant to determine whether kidney disease is acute or chronic and to determine prerenal, intrarenal, or postrenal causation.

1. CKD.

2. Acute kidney disease.

3. Chronic nephropathy.

4. Multiple myeloma.

5. Nephrolithiasis.

6. Nephrosclerosis.

PLAN

A. The treatment plan will focus on clients in earlier stages of CKD: stages 1, 2, and 3. Persons with stages 4 and 5 CKD need specialized interventions provided by nephrologists and should be referred immediately.

B. General interventions:

1. Provide support to the client.

2. Initiate appropriate referrals for a nephrologist, client education, and social and financial support as soon as possible.

3. For stage 4 or 5 CKD, access devices for hemodialysis, such as a primary arteriovenous fistula or graft, require months to mature and should be in place for 6 months prior to the start of dialysis.

C. Client teaching: *See Client Teaching Guide for this chapter, "Chronic Kidney Disease"*

1. Instruct the client on how kidneys work and how their body is affected by the CKD.

2. Emphasize the importance of following instructions to prevent further kidney damage:

a. Follow medication management instructions as carefully as possible.

b. No OTC medications should be taken that are not approved by the provider.

c. Keeping diabetes and HTN under control is crucial to maintaining kidney function.

d. Preventing CV complications by lowering cholesterol and BP is more important when you have CKD.

e. Smoking cessation is an important way to prevent worsening kidney function.

f. Routine follow-ups with the provider are necessary to monitor kidney function. Clients should be educated and encouraged to keep all appointments.

g. Follow dietary instructions regarding protein, fats, sodium, and minerals.

h. Dietary intake of protein is usually restricted to 0.8 to 1.0 g/kg/d of high biologic value protein.

i. Dietary sodium should be restricted to no more than 2 g daily.

j. Potassium should be restricted to 40 to 70 mEq/d.

k. Calories should be restricted to 35 kcal/kg/d; if the body weight is greater than 120% of normal or the client is older than 60 years of age, a lower amount may be prescribed.

l. Fat intake should be about 30% to 40% of total daily caloric intake.

m. Phosphorus should be restricted to 600 to 800 mg/d.

n. Calcium should be restricted to 1,400 to 1,600 mg/d.

o. Magnesium should be restricted to 200 to 300 mg/d.

3. Instruct when to notify the provider with urgent signs/symptoms or changes in kidney function:

a. Changes in urine volume.

b. Anorexia, nausea, and vomiting.

c. Increased edema.

d. Shortness of breath (SOB).

e. Increased fatigue.

f. Difficulty concentrating.

g. Muscle weakness, cramping, or twitching.

h. Fever.

i. Chest pain.

j. Hematuria.

D. Pharmaceutical therapy: General principles of medications used to prevent progression of CKD and to manage symptoms of complications of CKD:

1. Always prescribe the smallest effective dose of any medication.

2. Start with a low dose and gradually increase. Adjust drugs according to kidney function using the eGFR.

a. D osage int ervals may need to be extended.

3. Obtain regular measurements of creatinine/eGFR, at least every 6 months after any change in medications (e.g., angiotensin-converting enzyme [ACE] inhibitor, angiotensin II receptor blocker [ARB], or diuretics), medical intervention, or clinical status.

4. Monitor potassium:

a. Measure after change in medications, medical intervention, or clinical status with particular attention to potassium.

b. Check Cr and potassium prior to starting ACE inhibitors or ARBs, within 7 to14 days of starting, and within 7 to14 days after dose increase.

c. Cr rise greater than 20% or eGFR decrease greater than 15% after dose increase should be followed by further measurements within 7 to 14 days.

5. Provide education to the client of signs/symptoms to report to the provider immediately regarding drug therapy.

6. Diabetes:

a. Long-acting sulfonylureas may be associated by hypoglycemia with an unstable eGFR, especially those below 45.

b. If recurrent hypoglycemia, or unstable eGFR, consider using short-acting sulfonylureas or nonsulfonylureas.

c. In clients with an unstable eGFR or acute changes in clinical condition, metformin should be held, avoided, or reduced.

7. Avoid use of nephrotoxic radiographic contrast materials to prevent nephrogenic systemic fibrosis. If radiocontrast material use cannot be avoided because the benefit outweighs the risks, protect the kidney with acetylcysteine (Mucomyst) 600 mg orally BID on the day of intravenous (IV) contrast.

8. For clients with an extensive list of drugs, evaluate the need for dosage adjustment, contraindications, or nephrotoxicity. There are several classifications of the medications requiring dose adjustment. Classes may be used with the mnemonic BANDD CAMP.

a. **B:** Beta-blockers.

b. **A:** ACE inhibitors/ARBs.

c. **N:** NSAIDs, cyclooxygenase-2 (COX-2) inhibitors, opioids.

d. **D:** Diuretics.

e. **D:** Diabetic medications.

f. **C:** Cholesterol medications.

g. **A:** Antibiotics-aminoglycosides should be avoided or used in caution in clients with any degree of CKD. Kidney function should be monitored if they are used.

h. **M:** Miscellaneous.

i. **P:** Psychotropics.

9. Immunizations: Some vaccines in usual doses provide protection, whereas other vaccines require more frequent dosing, or larger doses, to achieve and maintain protective antibodies. Protective antibody titers may fall, and booster doses should be given if appropriate. In general, the recommendations are the following:

a. Annual influenza vaccination.

b. Pneumococcal vaccine with a single booster dose 5 years after the initial dose.

c. Hepatitis B vaccine series for clients prior to starting dialysis.

10. HTN control and kidney protection: It is recommended that HTN therapy achieve a goal of BP of 130/80 or less. Use of an ACE inhibitor or ARB therapy in early stages of CKD with persons who have proteinuria can preserve kidney function. The clinician should monitor serum potassium upon initiation of the therapy. It is common for the serum potassium to initially rise, then return to normal levels in 2 to 3 months. Follow-up serum potassium levels are recommended. In the early stages of CKD, with no proteinuria, the ACE inhibitors and ARBs have not been shown to be effective in protecting kidney function. HTN control can still be achieved with the ACE/ARB drugs, but other antihypertensive drugs can be used as well.

11. Fluid overload occurs when sodium intake exceeds sodium excretion. The combination of sodium restrictions and a loop diuretic can lower intraglomerular pressure and provide some kidney protection.

12. Hyperkalemia:

a. Hyperkalemia is managed with a low-potassium diet in combination with prescribing a loop diuretic such as furosemide. If a client is on an ACE or ARB, the addition of the loop diuretic will compensate for the elevation of serum potassium related to the ACE/ARB treatment.

13. Metabolic acidosis: Buildup of hydrogen ions causes bicarbonate levels to fall below acceptable levels. Sodium bicarbonate in a daily dose of 0.5 to 1 mEq/kg/d is often given. This prevents the symptoms of metabolic acidosis, which can increase muscle mass loss and worsen bone disease.

14. Renal osteodystrophy: The development of renal osteodystrophy is caused by hyperphosphatemia and hypocalcemia that are secondary to the decreased kidney function. In order to compensate, the client will develop secondary hyperparathyroidism. Dietary restriction of phosphate to 800 mg/d is recommended. In stage 3 CKD, the client will usually require an oral phosphate binder like calcium carbonate or calcium acetate to prevent hyperphosphatemia. Oral phosphate binders must be taken with meals to be effective. It is imperative to avoid phosphate binders that contain aluminum or magnesium. To suppress parathyroid hormone secretion, the client is given calcitriol, a vitamin D analog.

15. Anemia:

a. Use of elemental iron 200 mg such as ferrous sulfate 325 mg TID (65 mg elemental iron per dose) is recommended to maintain the percent transferrin saturation greater than 20% and the serum ferritin level to be greater than 100 ng/mL.

b. Although primarily used in clients with ESRD, erythropoietic agents (EPO), such as epoetin alfa (Procrit), Epogen, and darbepoetin alfa (Aranesp), are also used to correct the anemia in those with CKD who do not yet require dialysis.

16. Dyslipidemia: Management of dyslipidemia has been shown to slow progression of CKD. Statins and fibrates are commonly prescribed to lower total cholesterol, low-density lipoprotein (LDL), and triglycerides. The incidence of untoward side effects with statins and fibrates is increased in persons with CKD; therefore, lower dosages and careful monitoring are required.

FOLLOW-UP

A. Measure at least annually (more frequently with advanced CKD):

1. CBC and iron profiles (percent iron saturation).

2. Mineral metabolism (calcium, phosphorus).

3. Nutrition profile (albumin).

B. Diabetes:

1. Measure A1C every 3 to 6 months or as clinically indicated.

2. The American Diabetes Association 2018 Standards of Medical Care in Diabetes states that all hypertensive clients with diabetes monitor their BP at home to help identify potential discrepancies between office versus home BP, and to improve medication-taking behavior.

CONSULTATION/REFERRAL

A. Rapid and sustained deterioration in kidney function (a decline of eGFR >10%–15% within a 12-month period) warrants urgent referral to a nephrologist or internist, irrespective of eGFR values.

B. Early referral to a nephrologist is recommended for anyone who has CKD. Referral to a nephrologist must be done as quickly as possible for symptomatic clients with stage 4 or 5. Consultation with an endocrinologist or HTN specialist may be helpful in cases where DM and HTN continue to be poorly controlled.

C. Counseling on renal transplantation should be completed by a nephrologist, after the client is evaluated as a candidate for kidney transplantation, on what types of transplant are available and how the transplant process works.

D. Referral to a dietitian for nutritional counseling and education to assist the client to understand and follow complex dietary instructions is recommended.

E. Clients with stage 4 or 5 CKD need counseling regarding the psychosocial and financial impact of progressive renal disease. Referral to a renal social worker or case manager can help the client understand their health insurance benefits, how the transition to Medicare occurs after the health insurance benefit changes, and to help the client deal with concerns about work or family life.

F. Clients should be referred to educational and support organizations for further education regarding CKD.

INDIVIDUAL CONSIDERATIONS

A. Although renal replacement therapy is widely available, some clients, especially the more debilitated elderly or those who have a terminal illness, may request end-of-life counseling, including advance directives.

B. Serum creatinine may be elevated after intense exercise or heavy intake of protein in the form of meat. Instruct clients who exercise vigorously to withhold strenuous exercise and large intake of meat protein at least 24 hours prior to evaluation of serum creatinine.

C. Geriatrics:

1. Risk for renal artery stenosis increases with age; if a sharp increase in creatinine occurs after beginning treatment with an ACE or ARB, obtain a duplex Doppler ultrasound.

2. Older adults treated for ESRD should be referred for a palliative care consult to provide guidance in discussing/determining goals of care for this life-limiting condition.

3. Clients on dialysis with limited life expectancies should not routinely be screened for cancer without active signs and symptoms.

4. Beers list caution: Refer to Appendix C, Tables C.2 and C.5 on medications that should be avoided or have their dosage reduced with varying levels of kidney function in older adults.

RESOURCES

American Diabetes Association Standards of Medical Care in Diabetes: care.diabetesjournals.org/content/41/Supplement_1

Department of Veterans Affairs (VA)/Department of Defense (DoD) Clinical Practice Guidelines: www.healthquality.va.gov/guidelines/CD/CKD/GlobalRPh

CDC. (2021, March 9). Chronic Kidney Disease in the United States, 2021: www.cdc.gov. https://www.cdc.gov/kidneydisease/publications-resources/ckd-national-facts.html

Commonly used agents for renal failure: www.globalrph.com/renalfx.htm

Creatinine clearance and CKD staging calculator: www.globalrph.com/multiple_crcl_2012.htm

Renal Dosing Database: www.globalrph.com/index_renal.htm

National Kidney Disease Education Program: www.nkdep.nih.gov

National Kidney Foundation: www.kidney.org

National Kidney Foundation: Kidney Disease Outcomes Quality Initiative (KDOQI) Clinical Practice Guidelines: www.ajkd.org

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EPIDIDYMITIS

DEFINITION

A. The epididymitis is part of the genitourinary tract that includes the testes, the vas deferens, the prostate, the urethra, and the bladder. Epididymitis is an infection or inflammation of the epididymis, the tubular structure located on the posterior and superior aspect of the testis where sperms mature prior to ejaculation. Epididymitis is managed medically, whereas testicular torsion is a surgical emergency and should be considered in all cases.

1. Acute epididymitis lasts less than 6 weeks in duration of symptoms:

a. Acute epididymitis often involves the testis (epididymo-orchitis).

2. Chronic epididymitis lasts more than 6 in weeks duration of symptoms:

a. Inflammation chronic.
b. Obstructive chronic.
c. Chronic epididymalgia.

INCIDENCE

A. Epididymitis is the fifth most common urologic diagnosis in males aged 18 to 50 years. There are approximately 600,000 medical visits per year related to epididymitis. An estimated 1 in 1,000 males develop epididymitis annually. Chronic epididymitis may account for up to 80% of scrotal pain noted in the outpatient setting.

PATHOGENESIS

A. The exact pathophysiology is unclear. The cause may be the retrograde passage of infected urine from the prostatic ▶

urethra to the epididymis from the ejaculatory ducts and vas deferens. Reflux may be induced by having the client perform Valsalva or may be from strenuous exertion. Pathogens include *Chlamydia trachomatis, Neisseria gonorrhoeae, Escherichia coli, Proteus* species, *Klebsiella* species, *Pseudomonas, Mycoplasma* species, and *Treponema pallidum.*

PREDISPOSING FACTORS

A. Age:
  1. Age younger than 35 years is generally associated with urethritis with the following organisms:
    a. *C. trachomatis* (chlamydia).
    b. *N. gonorrhoeae* (gonorrhea).
  2. Benign prostatic hyperplasia (BPH) is more common in males older than 35 years and common organisms include the following:
    a. *E. coli.*
    b. *Pseudomonas* species.
    c. *Proteus* species.
    d. *Klebsiella* species.
  3. The older population of males usually has nonsexual epididymitis related to urinary tract instrumentation, surgery, and immunosuppression.

B. Men having sex with men (MSM) who are the insertive partner during anal intercourse have epididymitis with the following organisms:
  1. *E. coli.*
  2. *Pseudomonas.*
  3. Coliform bacteria.

C. Urinary tract infections (UTIs).

D. Tuberculosis (TB; should be considered if there is a history of or recent exposure to).

E. Vasectomy.

F. Indwelling urethral catheter.

G. Urethral stricture.

H. Amiodarone: high drug concentrations (dose-dependent).

I. Prolonged sitting (sedentary job, travel).

J. Mumps.

COMMON COMPLAINTS

A. Swelling and tenderness of the scrotum (usually located on one side).

B. Fever.

C. Chronic epididymitis:
  1. Epididymal pain and inflammation that last more than 6 weeks.
  2. May be accompanied by scrotal induration.

OTHER SIGNS AND SYMPTOMS

A. Gradual onset of localized, unilateral testicular pain. ***The client may get relief with elevation of the scrotum, which is a positive Prehn sign.***

B. Urethral discharge.

C. Dysuria.

D. Hematuria.

E. Fever and chills (in only 25% of adults with acute epididymitis).

SUBJECTIVE DATA

A. Elicit the onset, duration, and course of the client's symptoms.

B. Review the client's history for vasectomy or trauma to the groin.

C. Are there any other symptoms, including fever, dysuria, or discharge?

D. What helps relieve the pain? Ask about elevating the scrotum.

E. Does the client's sexual partner(s) have any symptoms or discharge?

F. Has there been any recent instrumentation or catheterization?

G. Is the pain unilateral or bilateral?

H. Review medication history for amiodarone.

I. Does the client have a recent TB exposure?

PHYSICAL EXAMINATION

A. Check temperature, blood pressure (BP), and pulse.

B. Inspection:
  1. Examine the client generally for discomfort before and during examination.
  2. Check the urethral meatus for discharge. Retract foreskin (if present) and assess for hygiene and smegma. Check the shaft of the penis, glans, and prepuce for lesions.
  3. Check the inguinal and femoral areas for bulges and hernias; have the client bear down and cough and reexamine them.

C. Palpate:
  1. Palpate testes and epididymides for inflammation, tenderness, and masses. In chronic cases, epididymis feels firm and lumpy. Vas deferens may be beaded.
  2. Check Prehn sign by elevating the affected hemiscrotum. This action relieves the pain of epididymitis but exacerbates the pain of torsion.
  3. Elicit a cremasteric reflex. Stroking the inner thigh should result in rise of the testicle and scrotum on the affected side. A normal cremasteric reflex indicates that testicular torsion is less likely.
  4. Palpate scrotum for hydrocele or varicocele.
  5. Check for costovertebral angle (CVA) tenderness.
  6. Examine the abdomen for masses, urinary distension, tenderness, and organomegaly.
  7. Palpate lymph nodes in the groin.
  8. Evaluate for an inguinal hernia.

D. Rectal exam:
  1. Check for symmetry, swelling, tenderness, and enlarged prostate.

DIAGNOSTIC TESTS

A. Gram stain of urethral secretions.

B. Urinalysis and urine cultures:
  1. Positive leukocyte esterase test (LET) on first-void urine.
  2. Microscopic examination of sediment from a spun-first void urine demonstrating ≥10 white blood cells (WBC) per high-power field.
  3. Urine testing for gonorrhea and chlamydia culture by nucleic acid amplification tests (NAATs).

C. Urethra swab (before void, after prostate massage) for gonorrhea and chlamydia culture.

D. In clients older than 40, express prostatic secretions.

E. TB skin test to rule out TB.

F. Complete blood count (CBC).

DIFFERENTIAL DIAGNOSES

A. Epididymitis:
  1. Bacterial.
  2. Viral epididymo-orchitis (mumps and *Haemophilus influenzae*).

B. Testicular torsion *(surgical emergency).*

C. Testicular tumor.

▶

D. Prostatitis.
E. Incarcerated inguinal hernia.
F. Orchitis (occurs with parotitis).
G. Trauma.
H. Epididymal congestion following vasectomy.
I. Folliculitis.
J. Herpes outbreak.
K. Hydrocele.
L. Spermatocele.

PLAN

A. General interventions:
1. Encourage and stress the importance of adequate fluid intake.
2. Stress the importance of taking all antibiotics as directed. Complete resolution of discomfort might not occur until a few weeks after completion of the antibiotic regimen.
B. Client teaching**:** *See Client Teaching Guide for this chapter, "Epididymitis."*
1. Client teaching for supportive therapy.
C. Pharmaceutical therapy:
1. Antibiotic therapy (both partners must be treated for a sexually transmitted infection [STI]). Treat empirically until laboratory test results are available.
2. Acute epididymitis should be treated for 10 days (Table 15.2).
3. Chronic epididymitis should be treated for 4 to 6 weeks for bacterial pathogens, especially chlamydia.
4. Nonsteroidal anti-inflammatory drugs (NSAIDs) for pain management.
5. Antitubercular treatment per the Centers for Disease Control and Prevention guidelines. www.cdc.gov/tb/topic/treatment/tbdisease.htm.
6. Amiodarone epididymitis usually responds to a dosage reduction or discontinuation.

FOLLOW-UP

A. See the client within 72 hours of initiation of treatment if the symptoms fail to improve. Signs and symptoms of epididymitis that do not subside within 3 days require reevaluation of the diagnosis and therapy.

TABLE 15.2 THE 2015 CDC RECOMMENDATION REGIMENS FOR ACUTE EPIDIDYMITIS

For acute epididymitis most likely caused by sexually transmitted chlamydia and gonorrhea: Ceftriaxone 250 mg IM in a single dose PLUS Doxycycline 100 mg orally BID for 10 days
For acute epididymitis most likely caused by sexually transmitted chlamydia and gonorrhea and enteric organisms (males who practice insertive anal sex): Ceftriaxone 250 mg IM in a single dose PLUS Levofloxacin 500 mg orally once a day for 10 days OR Ofloxacin 300 mg orally BID for 10 days
For acute epididymitis most likely caused by enteric organisms: Levofloxacin 500 mg orally once a day for 10 days OR Ofloxacin 300 mg orally BID for 10 days

CDC, Centers for Disease Control and Prevention; IM, intramuscular.
Source: From Centers for Disease Control and Prevention. (2015). 2015 sexually transmitted diseases treatment guidelines: Epididymitis. https://www.cdc.gov/std/tg2015/epididymitis.htm.

1. Pain typically improves within 1 to 3 days but may take up to 2 to 4 weeks.
2. Inadequate treatment can result in abscess formation and decreased fertility.
3. Males who experience swelling and tenderness that persist after completion of antimicrobial therapy should be evaluated for alternative diagnoses, including tumor, abscess, infarction, testicular cancer, TB, and fungal epididymitis.
B. Culture urine at the end of treatment (test of cure).
C. Treat both partners for STIs; test for all STIs and do not just focus on chlamydia and gonorrhea.
D. Consider testing for HIV.
E. Tuberculous epididymitis should be suspected if clinical signs worsen despite appropriate antibiotic therapy.
F. Males older than 50 years should be evaluated for urethral obstruction secondary to prostatic enlargement.
G. Those with HIV who have uncomplicated acute epididymitis should receive the same treatment regimen as those who are HIV-negative. Acute epididymitis in those with HIV infections may have other etiologies including cytomegalovirus (CMV), salmonella, toxoplasmosis, *Ureaplasma urealyticum*, *Corynebacterium* species, *Mycoplasma* species, and *Mima polymorpha*. Fungi and mycobacteria also are more likely to cause acute epididymitis in those with HIV infection than in those who are immunocompetent.

EMERGENT ISSUES/INSTRUCTIONS

A. Obtain an immediate consultation with a urologist if testicular torsion, scrotal abscess, or failed medical treatment is suspected.

CONSULTATION/REFERRAL

A. Consult a specialist for the following:
1. Intravenous pyelography (IVP).
2. Doppler ultrasonography.
3. Scrotal ultrasonography.
4. Radionuclide scrotal imaging.

INDIVIDUAL CONSIDERATIONS

A. Partner:
1. Treat sexual partners for STI.
2. Consider testing for HIV.
B. Geriatrics:
1. Pharmacologic caution in geriatrics:
a. Fluoroquinolone use in the elderly has the potential to cause neuropsychiatric symptoms, including seizures to worsening dementia.
2. Epididymitis in the elderly is often caused by an enlarged prostate gland.
3. Trouble voiding can be an early finding.
4. Retrograde flow of urine.

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HEMATURIA

DEFINITION

A. Hematuria is blood in the urine. Hematuria is a symptom of an underlying disease/condition; however, **routine screening is not recommended.** Microscopic hematuria is defined as greater than three red blood cells (RBCs) per high-power microscope field (HPF) in urinary sediment clean-catch midstream urine specimens.

B. Asymptomatic microscopic hematuria can range from minor findings that do not require treatment to highly significant, life-threatening lesions. Microscopic hematuria is an incidental finding. The American Urological Association (AUA) recommends an appropriate renal or urologic evaluation with asymptomatic microscopic hematuria for clients who are at risk for urologic disease or primary renal disease.

C. If the excretion rate exceeds one million RBCs, macroscopic or gross hematuria is noted. Gross hematuria (macroscopic hematuria) is suspected when red or brown urine is present. Color change does not necessarily reflect the degree of blood loss. Glomerulonephritis is associated with brown urine, whereas bleeding from the lower urinary tract is suggested by pink or red urine. Gross hematuria with passage of clots almost always indicates a lower urinary tract source.

INCIDENCE

A. The prevalence of asymptomatic hematuria is from 1% to 20% of the general population. Less than 3% excrete 10 RBC/HPF. Every disease of the genitourinary (GU) tract can produce hematuria.

PATHOGENESIS

A. Prerenal pathology:
- **1.** Coagulopathy: hemophilia or idiopathic thrombocytopenia purpura (ITP).
- **2.** Drugs:
  - a. Anticoagulants (warfarin or dabigatran).
  - b. Aspirin.
- **3.** Sickle cell disease or trait.
- **4.** Collagen vascular disease; lupus.
- **5.** Wilms tumor.

B. Renal pathology:
- **1.** Nonglomerular pathology:
  - a. Pyelonephritis.
  - b. Polycystic kidney disease.
  - c. Granulomatous disease; tuberculosis (TB).
  - d. Malignant neoplasm.
  - e. Congenital and vascular anomalies.
- **2.** Glomerular pathology:
  - a. Glomerulonephritis.
  - b. Berger disease.
  - c. Lupus nephritis.
  - d. Benign familial hematuria.
  - e. Vascular abnormalities; vasculitis.
  - f. Alport syndrome; familial nephritis.

C. Postrenal pathology:
- **1.** Renal calculi.
- **2.** Ureteritis.
- **3.** Cystitis.
- **4.** Prostatitis.
- **5.** Benign prostatic hypertrophy (BPH).
- **6.** Epididymitis.
- **7.** Urethritis.
- **8.** Malignant neoplasm.

D. False hematuria:
- **1.** Vaginal bleeding.
- **2.** Recent circumcision.
- **3.** Pigmentation:
  - a. Food: beets, blackberries.
  - b. Medications: quinine sulfate, phenazopyridine, and rifampin.

E. Other causes:
- **1.** Trauma.
- **2.** Strenuous exercise (marathons).
- **3.** Fever.

PREDISPOSING FACTORS

A. See previous section, "Pathogenesis."

B. Risk factors for malignancy:
- **1.** Age older than 35.
- **2.** Smoking (current use or history).
- **3.** Chemical exposure (cyclophosphamide, benzenes, aromatic amines).
- **4.** History of pelvic irradiation.
- **5.** Chronic analgesic abuse.
- **6.** Chronic urinary tract infections (UTIs).
- **7.** Prior urologic disease or treatment.
- **8.** Male.

COMMON COMPLAINT

A. Pink or red urine (clots may be present) or brown cola-colored urine on toilet tissue is the common complaint.

OTHER SIGNS AND SYMPTOMS

A. Pain may or may not be present. Colicky flank pain radiating to the groin suggests a kidney stone. Significant flank pain of renal colic is usually secondary to renal calculi, but may occasionally be associated with passage of clots.

B. Frequency, dysuria, urgency, and suprapubic pain occur with cystitis and inflammatory lesions of the lower urinary tract.

C. Dull flank pain with fever and chills may accompany pyelonephritis.

D. Hesitancy and dribbling of the urine suggest BPH.

SUBJECTIVE DATA

A. Elicit onset, duration, and occurrence (beginning, ending, or during voiding) of hematuria. Describe the color and amount: Is it "pink on tissue" or bright red in the toilet and tissue?

B. Question the client regarding medical history of renal disease, systemic disease such as lupus, or sickle cell disease.

C. Review all medications including over-the-counter (OTC) and herbal products. Evaluate specifically for the use of aspirin, ibuprofen, warfarin (Coumadin), and laxatives containing phenolphthalein. Rifampin and phenazopyridine HCl (Pyridium) can change the color of urine to orange or red.

D. Review other symptoms, such as dysuria, fever, chills, pain, and hesitancy with voiding.

E. Female clients:
- **1.** Establish whether the blood was urinary or from the vagina (after intercourse or during menstruation).
- **2.** Is the client postpartum?
- **3.** Is there a history of endometriosis?

F. Does the client bruise easily? Does the client have bleeding noted when flossing teeth or with brushing?

G. Has the client had a recent bout of pharyngitis with a rash, hematuria, edema, or hypertension (HTN; glomerulonephritis)? ▶

H. Have they had any recent trauma, car accident, or strenuous exercise (e.g., running a marathon)?
I. Does the client's occupation include risks for trauma, such as occupational truck driver (e.g., bumps in road)?
J. Does the client know if they have had any exposure to TB?
K. Is there any family history of kidney disease, stones, and familial nephritis?
L. Does the client have any current outbreaks of herpes or other sexually transmitted infections (STIs)?
M. Review the client's smoking history.
N. Review occupation exposure to chemicals or dyes (benzenes or aromatic amines).
O. Review intake of foods such as beets and blackberries.
P. Does the male client have any hesitancy and dribbling (signs of prostatic obstruction)?
Q. Evaluate if there is rectal bleeding from hemorrhoids for strain with a bowel movement (BM).

PHYSICAL EXAMINATION

A. Check temperature, blood pressure, and weight in the presence of recent weight gain or edema.
B. Male or female clients:
1. Inspect:
a. Inspect mouth: Check tonsils for enlargement and check gums for petechiae.
b. Examine skin for signs of bleeding or bruises and pallor.
c. Examine for edema.
2. Palpate:
a. Check the back and abdomen for costovertebral angle (CVA) tenderness.
b. Check the abdomen for masses, urinary distension, tenderness, and organomegaly.
c. Palpate groin lymph nodes for enlargement.
3. Auscultate:
a. Heart and lungs.
b. For abdominal bruits.
C. Female clients:
1. Inspect:
a. Direct visualization of the external genitalia for inflammation, ulcerations, nodules, lesions, and hemorrhoids.
b. Ask the client to bear down to check for cystocele and rectocele.
c. Speculum exam: Observe for atrophic vaginitis, torn tissue, discharge, and friable cervix.
2. Palpate:
a. Milk urethra for discharge.
b. Bimanual exam: Check for cervical motion tenderness and adnexal masses.
c. Rectal exam: Check for the presence of hemorrhoids.
D. Male clients:
1. Inspect:
a. Direct visualization of the genitals; check the urethral meatus for discharge.
b. Retract the foreskin (if present) and assess for hygiene and smegma. Check the shaft of the penis, glans, and prepuce for lesions or urethral meatal erosion.
2. Palpate:
a. Palpate the testes and epididymides for inflammation, tenderness, and masses; palpate the scrotum for hydrocele or varicocele.
b. Check the inguinal and femoral areas for bulges and hernias; have the client bear down and cough and reexamine client.
c. Rectal exam:
i. Check for swollen or tender prostate.
ii. Check for presence of hemorrhoids.

DIAGNOSTIC TESTS

A. Urinalysis:
1. The initial step in the evaluation of clients with red urine is to centrifuge the urine specimen to see if the red or brown color is in the urine sediment or supernatant.
2. If the supernatant is red to brown, test for heme (hemoglobin [Hgb] or myoglobin) with a urine dipstick. Semen is in urine after ejaculation and may cause a positive heme reaction on the dipstick.
3. A positive dipstick must always be confirmed with a microscopic examination.
4. Urine culture and sensitivity.
5. Urine cytology.
B. After the physical exam and consultation, consider the following:
1. Complete blood count (CBC) with differential.
2. Blood urea nitrogen (BUN).
3. Creatinine.
4. Prothrombin time (PT), partial thromboplastin time (PTT), platelet count, and bleeding time (if indicated).
5. Sickle cell testing (if indicated).
6. The Nickel premassage and postmassage two-glass test for the male client (see Section II: Procedures, "Prostatic Massage Technique: Two-Glass Test").
7. Collect 24-hour urine for calcium, uric acid, protein, and creatinine. Follow with a 24-hour urine collection for creatinine and protein to assess renal function and quantitatively assess the degree of proteinuria.
8. Culture for gonorrhea and chlamydia.
9. Urine culture for acid-fast bacillus.
10. CT urography (CTU) is considered the preferred initial imaging in most clients for any unexplained persistent hematuria. CT is considered the best imaging modality for evaluation of urinary stones, renal and perirenal infections, and associated complications. Intravenous pyelogram (IVP) and ultrasound are not as sensitive in the evaluation.
11. Cystoscopy: The combination of a CTU and the cystoscopy provides a complete evaluation.
12. A CT scan of the abdomen or pelvis should be considered with a history of trauma to determine the source of blood.
C. Based on history, consider the following tests:
1. Strep testing to detect poststreptococcal glomerulonephritis.
2. Antinuclear antibody (ANA) test to detect lupus nephritis.
D. Urologic referral testing includes:
1. CTU and cystoscopy.
2. MRI if a mass is suspected.
3. Renal biopsy.

DIFFERENTIAL DIAGNOSES

A. See section on "Pathogenesis" for differential diagnoses.

PLAN

A. General interventions:

1. Investigate and diagnose cause(s). Only a limited workup (electrolytes, CBC) is needed in clients younger than 35 with normal physical exam.

2. Clients older than 35 need a detailed investigation and referral, especially if a smoker.

3. Microhematuria with clients on an anticoagulant requires a urologic/nephrology workup regardless of the type or level of anticoagulation.

4. Repeat urinalysis in 2 weeks.

B. Client teaching:

1. There is no one specific treatment for all cases of hematuria.

2. Treatment is aimed at the specific underlying cause, if a cause can be identified.

C. Pharmaceutical therapy:

1. None is recommended for hematuria unless an infection is diagnosed.

FOLLOW-UP

A. For clients who are at risk for malignancy and have a negative workup:

1. Evaluate in 1 year.

2. After two consecutive negative urinalysis, discontinue the follow-up.

3. If gross hematuria after the initial negative urinalysis, repeat a full evaluation.

B. For clients with HTN, proteinuria and an increase in creatinine need to be reevaluated for renal disease.

C. For persistent asymptomatic microhematuria after a negative workup, a yearly urinalysis is needed.

D. For persistent or recurrent asymptomatic microhematuria after the initial negative workup, consider repeating the evaluation within 3 to 5 years.

E. Culture urine for acid-fast bacillus if sterile pyuria and hematuria persist.

CONSULTATION/REFERRAL

A. If all benign causes for hematuria have been ruled out, acute conditions treated and hematuria still persists, referral to a urologist or a nephrologist is recommended.

B. The presence of significant proteinuria (excretion of more than 1,000 mg per 24 hours), red cell cast or renal insufficiency, or a predominance of dysmorphic RBCs in the urine should prompt an evaluation for renal parenchymal disease by a nephrologist. Red cell casts are considered virtually pathognomonic of glomerular bleeding.

C. New gross hematuria should be promptly reevaluated.

INDIVIDUAL CONSIDERATIONS

A. Females:

1. Nonpregnant: Rule out menstruation and sexual activity.

2. Pregnant: Rule out vaginal bleeding such as threatened abortion, abruption, or placenta previa.

3. Ultrasound can be used to evaluate the pregnant client. CTU should not be used secondary to the radiation exposure.

4. Females with a history of smoking should be screened for bladder cancer.

B. Geriatrics:

1. Hematuria is commonly associated with UTI in high-risk elderly individuals.

2. High-risk individuals:

a. Have a history of kidney stones.

b. Females with atrophic vaginitis (bleeding from friable tissues). Postmenopausal females with atrophic vaginitis and frequent UTIs can benefit significantly from use of topical estrogens.

c. Clients who use an indwelling or intermittent catheter may be presumed to have a complicated UTI when hematuria is present accompanied by another significant sign or symptoms such as fever, tachycardia, or altered mental status.

C. The risk of malignancy increases among older individuals with a significant history of smoking or analgesic abuse.

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INTERSTITIAL CYSTITIS

DEFINITION

A. Interstitial cystitis (IC) is a chronic condition that results in recurring discomfort or suprapubic pain, as well as pressure in the bladder and surrounding pelvic region related to bladder filling. IC is also commonly known as painful bladder syndrome (PBS). IC/PBS includes all cases of urinary pain with persistent urge to void or urinary frequency that cannot be attributed to other causes (e.g., infection, stones, or other pathology).

B. The persistent urge to void helps to distinguish the symptoms of IC/PBS from those of overactive bladder (OAB). IC/PBS affects quality of life related to social activities, lost work productivity, sleep deprivation due to urinary frequency, fatigue, and even depression.

C. The Society for Urodynamics and Female Urology (SUFU) has stated that the symptoms should last more than 6 weeks in order for therapy to begin.

D. IC/PBS clients void to avoid or relieve pain, whereas clients with OAB void to avoid incontinence.

INCIDENCE

A. Actual prevalence is unknown due to the variability of diagnostic criteria. It is not uncommon for clients to experience a lag time of 5 to 7 years before diagnosis. It is estimated that in the United States IC/PBS affects 3 to 8 million females and 1 to 4 million males. The symptom complex is the same for females and males.

PATHOGENESIS

A. The pathophysiology of IC/PBS remains unclear. It is not established whether IC/PBS is a localized condition just involving the bladder or whether it is a systemic disease that affects the bladder.

B. Clients frequently exhibit several mental health disorders and negative personality traits. Therefore, in addition to targeting the bladder pathologic condition, psychological intervention focusing on personality traits and anxiety mood status should be provided to improve quality of life of IC clients.

PREDISPOSING FACTORS

A. In both sexes, with a higher prevalence in females.
B. Mean age of diagnosis is 42 to 45 years.
C. Urinary tract infection (UTI).
D. Prostatitis.
E. Chronic yeast infections.
F. Posthysterectomy or other pelvic surgery.
G. Medications:
  1. Calcium channel blockers.
  2. Cardiac glycosides.
H. Other hypersensitivity conditions that coexist with IC:
  1. Fibromyalgia.
  2. Irritable bowel syndrome (IBS).
  3. Chronic headaches.
  4. Vulvodynia.
  5. Sjögren syndrome.
I. Depression/mood disorders.
J. Hypothyroidism.

COMMON COMPLAINTS

A. Mild discomfort to intense pain with bladder filling and/or emptying is the hallmark symptom. The pain is not limited to the bladder/suprapubic area but includes symptoms throughout the pelvic area, lower abdomen, and back.
B. Persistent urge to void.
C. Frequency.
D. Urgency.
E. Nocturia.

OTHER SIGNS AND SYMPTOMS

A. Combination of urgency and frequency.
B. Pressure.
C. Increase in symptoms during menstruation.
D. Pain during vaginal intercourse.
E. Low back pain with bladder filling.

SUBJECTIVE DATA

A. Review onset, frequency, duration, and severity of symptoms.
B. Evaluate if the pain of bladder filling is partially or completely relieved by voiding.
C. Does the client void frequently in order to maintain a low bladder volume and avoid discomfort versus voiding frequently to avoid urge incontinence (OAB)?
D. Are there any OAB triggers (e.g., citrus, beer, coffee) that exacerbate symptoms?
E. Are symptoms increased after stress, exercise, intercourse, being seated for long periods of time, or during the menstrual cycle?
F. How much do the symptoms affect the client's quality of life (e.g., sleep disturbance, loss of work, avoiding activities)?
G. Does the client have any other chronic pain syndromes such as IBS, chronic fatigue, dyspareunia, or fibromyalgia?
H. Review the client's surgical history and history of genitourinary (GU) cancers.
I. Has the client had any GU trauma or falls onto the coccyx?
J. Review the client's history of UTIs, urinary retention, and urinary tract stones.
K. Review all medications, including over-the-counter (OTC) and herbal products.
L. Administer a pain/symptom evaluation tool at each visit:
  1. The Pelvic Pain and Urgency/Frequency (PUF) Patient Symptom Scale is available at www.ichelp.org/wp-content/uploads/2015/06/PUF_Questionnaire.pdf.
  2. The Interstitial Cystitis Symptoms Index (ICSI) is available at www.essic.eu/pdf/ICSIandICPI.pdf.

PHYSICAL EXAMINATION

A. Check temperature (if indicated to rule out infection; fever is not associated with IC) and blood pressure (BP).
B. Inspect:
  1. Note general appearance for signs of depression and discomfort before and during examination.
  2. Inspect the male external genitalia for redness, edema, lesions, and discharge.
  3. Inspect the female genitalia for discharge, lesions, fissures; inspect cervix for cervicitis.
  4. Pelvic exam to test pelvic floor muscles to determine if any of the pain is related to spasms.
C. Auscultate:
  1. Auscultate the heart and lungs.
  2. Auscultate bowel sounds in all four quadrants.
D. Palpate:
  1. Palpate back; note costovertebral angle (CVA) tenderness.
  2. Palpate the abdomen for suprapubic tenderness, rebound masses, or pain.
  3. Females: Perform bimanual exam to rule out other infections and pelvic inflammatory disease (PID; tenderness of the cervix, uterus, and adnexal should be absent. During the pelvic examination, evaluate locations of tenderness and trigger points.
  4. Males: Complete palpation of external genitalia, prostate, and rectal exam.
E. Percuss:
  1. Percuss the bladder.
  2. Percuss the back for CVA tenderness.
F. Neurologic exam:
  1. Perform a limited neurologic examination to rule out an occult problem.

DIAGNOSTIC TESTS

A. Urinalysis with microscopy to exclude hematuria.
B. Urine culture and sensitivity may be ordered even with a negative urinalysis to evaluate low levels of bacteria.
C. Postvoid residual (PVR) volume by straight catheter or ultrasound.
D. Urodynamic testing is not currently considered to have a role in the diagnosis of IC/PBS; however, urodynamic testing should be used for complex presentations.
E. Cystoscopy is usually reserved for gross or microscopic hematuria.
F. Hydrodistention is not required for diagnosis or treatment.
G. Bladder biopsy is not required for diagnosis; however, it is used for exclusion of other disorders.
H. The potassium sensitivity test is not recommended for routine use since the results are nonspecific for IC/PBS.

DIFFERENTIAL DIAGNOSES

A. IC.
B. UTI.
C. IBS.
D. Females:
  1. Endometriosis.
  2. Vulvodynia.
E. Males:
  1. Chronic prostatitis (CP).
  2. Benign prostatic hypertrophy (BPH).

PLAN

A. General interventions:

1. Behavioral modifications are recommended. Restrict fluids to 64 oz/d, divided into 16 oz per meal and 8 oz between meals.

2. Progressive timed voiding on a 2- to 3-hour schedule. If the client is unable to hold urine for this interval, progressively increase urine storage time between voids by 15 minutes per week until the goal of a 2- to 3-hour interval is reached.

3. Kegel exercises should be avoided with IC.

4. Psychosocial support is an integral part of chronic pain disorders.

5. Pelvic floor physical therapy is recommended to help strengthen pelvic floor muscles.

B. Client teaching:

1. Educate about normal bladder function, the benefits versus risks/burdens of the available treatment alternatives, and the fact that no single treatment has been found to be effective for the majority of clients.

2. Several foods have been identified as bladder irritants, including foods rich in potassium. Clients may try to eliminate foods/drinks and reintroduce them one at a time to identify any items that make their symptoms worse. Examples are:

a. Alcohol.
b. Tomatoes.
c. Spices/spicy foods.
d. Chocolate.
e. Caffeinated beverages.
f. Coffee.
g. Artificial sweeteners.
h. Citrus: lemons, limes, and oranges (including citrus-flavored beverages).
i. Cranberry/cranberry juice.

C. Pharmacologic therapies:

1. Pentosan polysulfate sodium (Elmiron) is the only oral medication approved by the Food and Drug Administration (FDA) for treatment of IC.

a. Dosage: 100 mg orally TID.
b. Clients should be evaluated at 3 months and may be continued an additional 3 months if there has been no improvement and if there are no therapy-limiting side effects.
c. The risks and benefits of continued use beyond 6 months in clients who have not responded are unknown.

2. Amitriptyline (Elavil) is used in the treatment of other pain syndromes, including IC. A self-titration dose of 25 mg orally every night may be utilized and increase in increments of 25 mg every week to a maximum dose of 100 mg orally per day.

3. Cimetidine 400 mg BID.

4. Hydroxyzine hydrochloride (Vistaril, Atarax) 25 to 75 mg orally at bedtime.

5. Uro Blue medications for short-term bladder spasms.

6. Nonsteroidal anti-inflammatory drugs for pain control.

7. Medications may be instituted for treating any comorbid depression.

8. Treat any comorbid infections and conditions (e.g., UTIs, sexually transmitted infections, inflammatory bowel disease, or endometriosis).

9. Bladder instillation:

a. Intravesical dimethyl sulfoxide (DMSO; Rimso-50) is the only drug approved by the FDA for bladder instillation.
b. Heparin instillation.
c. Lidocaine instillation.
d. "Bladder cocktail" combination of sodium bicarbonate, heparin, lidocaine, and/or triamcinolone. There are various formulas/combinations.

10. Cyclosporine A has been utilized when other treatments have not provided adequate relief or control of symptoms and quality of life. The American Urological Association (AUA) guidelines consider cyclosporine as the fifth-line treatment option.

D. Intradetrusor botulinum toxin A (BTX-A) therapy may require posttreatment intermittent self-catheterization.

E. Laser or electrocautery if Hunner ulcers are present.

F. Surgical options are available if all other therapies have failed.

G. Some alternative treatments such as guided imagery and acupuncture have shown some success but have not been studied.

H. Therapies that are not recommended/should not be offered include the following:

1. Long-term antibiotics.

2. High-pressure, long-duration hydrodistention.

3. Systemic steroids.

4. Intravesical resiniferatoxin (ultrapotent capsaicin analogue).

5. Intravesical Bacillus Calmette–Guerin (BCG).

6. Potassium sensitivity test is not recommended for routine use. It is a nonspecific and painful test.

FOLLOW-UP

A. The IC/BPS diagnosis should be reconsidered if no improvement occurs after multiple treatment approaches.

B. Ineffective treatments should be stopped once a clinically meaningful interval has elapsed.

C. Allodynia, the perception of stimuli causing pain from objects that should not be painful, such as brushing hair/scalp, may be present; therefore, an adequate pelvic examination may not be possible. Consider empiric treatment and have the client return for a pelvic examination to finish the evaluation.

D. Have the client keep a 1-day bladder diary prior to visits to evaluate a pattern of low urine volume frequency characteristic of IC/PBS.

E. As with all medications, start at the lowest dose and titrate/increase doses if there is an improvement in symptoms.

CONSULTATION/REFERRAL

A. Refer to a urologist for a more thorough workup and testing.

B. Refer to a pain management specialist if indicated. Pain management should be continually assessed due to the importance of quality of life. If pain management is inadequate, then consideration should be given to a multidisciplinary approach.

C. Electrical stimulation therapy may be considered. The implanted sacral neuromodulation device is FDA-approved for treatment of urinary urgency and frequency but not specifically for treatment of IC/PBS.

D. Provide clients with information on support groups, online resources, and professional organization client guidelines.

RESOURCES

American Urological Association (AUA): www.auanet.org
International Painful Bladder Foundation: www.painful-bladder.org
Interstitial Cystitis: www.mayoclinic.org/diseases-conditions/interstitial-cystitis
Interstitial Cystitis Association (ICA): www.ichelp.org
Interstitial Cystitis Network: www.ic-network.com
Pelvic Pain and Urgency/Frequency Patient Symptom Scale available at www.ichelp.org/wp-content/uploads/2015/06/PUF_Questionnaire.pdf
Society of Urodynamics, Female Pelvic Medicine & Urogenital Reconstruction: www.sufuorg.com

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NOCTURIA

DEFINITION

A. Nocturia is frequent wakefulness at night to urinate. The American Urological Association (AUA) and the International Continence Society (ICS) define nocturia as "the need to awake in order to urinate at least once or twice during the night." Most individuals describe their nocturia progressing from having an episode occasionally to having one or more episodes nightly.

B. Nocturia in the older population is associated with higher rates of accidental falls and fractures.

C. Nocturia is a leading cause of sleep disturbance affecting sleep onset, difficulty returning to sleep following nighttime awakening, and sleep deprivation associated with morning fatigue having a negative impact on quality of life.

D. Nocturia is associated with increased rates of depression and work absenteeism.

E. Occasionally nocturia can be an indicator of worsening clinical status of an underlying disease, such as chronic kidney disease (CKD), lithium toxicity, diabetes, or congestive heart failure (CHF).

INCIDENCE

A. The prevalence of nocturia increases with age.

B. Some studies suggest that nocturia is present in 50% of both males and females older than 50 years of age.

C. Among those 18 to 49 years old, more females than males experience nocturia.

D. Males older than 60 years of age experience nocturia more than females.

E. The prevalence of twice nightly or greater nocturia among males between the ages of 70 and 79 years is nearly 50%.

F. Nocturia associated with pregnancy nearly always resolves by 3 months postpartum.

G. The incidence of nocturia is highest in blacks and does not appear to be related to socioeconomic factors. Hispanics tend to have a somewhat higher incidence of nocturia than Whites but less than the Black population.

PATHOGENESIS

A. Aging is a major factor, and nocturia occurs more commonly in older adults than in younger individuals.

B. Nocturia can be attributed to any disorder or condition that causes one of the following:

1. Low-volume bladder void due to reduced bladder capacity or impaired bladder function.
2. Increased volume of nighttime urinary output.
3. Sleep disturbance.

C. In both females and males, the occurrence is much different due to the dissimilarity of the respective anatomy.

1. In females, nocturia is generally experienced as a result of childbirth, menopause, and pelvic organ prolapse.
2. Nocturia in males, however, can be directly attributed to enlarged prostate causing a bladder outlet obstruction (BOO).

PREDISPOSING FACTORS

A. Multiple factors could cause nocturia in both males and females, including the following:

1. Chronic diseases:
   a. Diabetes.
   b. Heart failure/venous status (third spacing of fluids).
   c. Diabetes insipidus and uncontrolled diabetes mellitus (DM).
   d. Primary polydipsia (nocturnal polyuria).
   e. CKD.
   f. Obstructive sleep apnea (OSA).
   g. Neurologic diseases.
   h. Obesity.
   i. Age-related defect in diurnal secretion of arginine vasopressin (AVP) in the elderly.
2. Medications:
   a. Diuretics.
   b. Calcium channel blockers.
   c. Beta-blockers (associated with bladder storage problems).
   d. Xanthines (associated with bladder storage problems).
   e. Selective serotonin reuptake inhibitors (SSRIs).
   f. Cholinesterase inhibitors.
   g. Lithium.
   h. Corticosteroids.
3. Caffeine.
4. Alcohol.
5. Nocturnal polyuria.
6. Low nocturnal bladder capacity.
7. Combination of the nocturnal polyuria and low nocturnal bladder capacity.
8. Overactive bladder (OAB).
9. Sex:
   a. Male:
      i. Benign prostatic hypertrophy (BPH).
      ii. Prostate cancer.
   b. Female:
      i. Pregnancy.
      ii. Estrogen deficiency.
      iii. Pelvic floor laxity (e.g., cystocele, uterine prolapse).
10. Excessive fluid intake in the evening.

▶

11. Larger fluid intake alone (greater than 40 mL/kg/d) may be responsible for the client's nocturia without other identifiable causes.
12. Interstitial cystitis/painful bladder syndrome (IC/PBS).
13. Chronic or recurrent urinary tract infections (UTIs).
14. Idiopathic.
15. Fecal impaction.

COMMON COMPLAINTS

A. Morning fatigue.
B. Frequent wakefulness.
C. Insomnia.
D. Urinary frequency.
E. Urinary urgency.
F. Geriatrics:
1. May not present with classic symptoms or be asymptomatic.
2. Incontinence.
3. Mental confusion.
4. History of falls.

OTHER SIGNS AND SYMPTOMS

A. Asymptomatic.
B. Bladder spasms.
C. Suprapubic pain or suprapubic discomfort.
D. Depression.

SUBJECTIVE DATA

A. Review the onset, course, and duration of symptoms.
B. Review the number of sleep interruptions or frequent wakefulness. Ask an open-ended question: "Over the past month, how often did you typically get up at night to urinate, from the time you went to bed until the time you got up in the morning?"
C. Are there any other genital problems such as herpes lesions or vaginal/penile discharge?
D. Review all medications, prescriptions, over-the-counter (OTC) drugs, and herbals for how often and what time of the day they are taken.
E. Does the client have a fever and chills or back or flank pain (unilateral or bilateral)?
F. Is the client pregnant? If not, what type of birth control do they use?
G. Is there any history of previous UTIs? How often and how were they treated? Were any tests performed in a workup by a urologist?
H. Is there any history of IC? Were any tests performed in a workup by a urologist? What treatments?
I. In the postmenopausal client, review whether they had a known prolapse and/or vaginal atrophy. Do they use any systemic or local estrogen medications?
J. How much liquid or water does the client drink every day?
1. Note the amount of caffeine and alcohol.
2. Note the total amount of fluids.
3. At what time in the evening are the last fluids taken?
4. Does the client awake at night and drink more liquids?
K. In older males, review the strength of the urinary flow, dribbling, hesitancy, and so forth.
L. Is there any history of other medical diseases (noted in the section "Predisposing Factors")?
M. Does the client have a mobility issue and use any aids, such as a cane or a walker?
N. In the elderly, what is the client's ability to toilet independently?

PHYSICAL EXAMINATION

A. Both sexes:
1. Check temperature (if an infection is suspected), pulse, blood pressure (BP), and respirations. Measure height and weight to calculate body mass index (BMI).
2. Inspection begins as the client comes into the examination room:
a. Assess cognitive and functional status.
b. Observe mobility (including use of a walker, cane, or other aid).
c. Assess for risk of falls.
d. Observe client transfer.
e. Assess manual dexterity (ability to use buttons and zippers).
f. Inspect for evidence of cardiac overload: pedal edema.
3. Auscultate:
a. Auscultate the lungs for evidence of fluid overload: rales.
4. Palpate:
a. Palpate the abdomen.
b. Palpate suprapubic and back for costovertebral angle (CVA) tenderness.
c. Palpate inguinal nodes for CVA tenderness.
d. Palpate bladder fullness and need for catheterization.
e. Perform a neurologic examination to determine presence of perineal sensation and anal wink (visual or palpated anal contraction in response to a light scratch of the perineal skin lateral to the anus).
5. Screen for depression.
B. Females:
1. Inspect perineal skin for irritation, thinning, vaginal atrophy, and vaginal discharge.
2. Perform a bimanual pelvic exam for prolapse, masses, or tenderness:
a. Remove the top blade of the speculum and evaluate the vaginal wall support.
b. Ask the client to cough to reevaluate the vaginal wall support.
3. Palpate:
a. Perform rectal exam for sphincter tone, masses, and fecal impaction.
C. Males:
1. Inspect:
a. Inspect the glans penis for abnormalities in urethral meatus. (Hypospadias may cause postvoid dribbling.)
b. Uncircumcised males should be evaluated for phimosis and balanitis.
2. Palpate:
a. Perform rectal exam for sphincter tone, masses, fecal impaction, prostate size, and contour.
b. Palpate the scrotum to evaluate masses.
c. Evaluate the presence of an inguinal hernia since straining with a partial urinary obstruction can worsen an inguinal hernia.

DIAGNOSTIC TESTS

A. History, physical examination, and urinalysis may be sufficient to guide initial therapy and to rule out other causative factors. Other tests include the following:
1. Urine culture and sensitivity if infection is suspected.
2. Urine cytology if hematuria or pelvic pain is present.
3. Urodynamic testing is strongly recommended.
4. A prostate-specific antigen (PSA) should be considered. ▶

5. Cystoscopy/ureteroscopy.
6. Renal ultrasound with Doppler.
7. Renal function, electrolytes, glucose, and hemoglobin (Hgb) A1C.
8. Sleep study may be indicated, especially if positive for nocturnal hypoxemia.
9. Depression screen.
10. Neurologic evaluation may be needed.
11. Nocturnal pulse oximetry study.
12. Clients with neurologic conditions affecting the bladder (diabetic neuropathy), older males, and clients with a history of genitourinary (GU) surgery are at risk for urinary retention.
1. Postvoid residual (PVR) by catheterization or ultrasound (a PVR of <50 mL is considered adequate emptying, and a PVR >200 mL may require referral for further evaluation).

DIFFERENTIAL DIAGNOSES

A. Nocturia (idiopathic).
B. Nocturnal polyuria.
C. Low nocturnal bladder capacity.
D. DM/insipidus.
E. Medication(s) side effect.
F. Fluid intake near bedtime (includes caffeine and alcohol).
G. Chronic or recurrent UTI.
H. Enlarged prostate.
I. Pregnancy.
J. IC/PBS.
K. CHF/vascular stasis.
L. Heart disease.
M. Sleep disturbance.

PLAN

A. General interventions:
1. Have the client complete a 24-hour voiding diary before their next visit. The 24-hour voiding diary is available in *Client Teaching Guide* for this chapter, *"Urinary Incontinence: Females."*
2. Evaluate proper usage of continuous positive airway pressure (CPAP) machine if sleep apnea is present.
3. Percutaneous tibial nerve stimulator (PTNS) has been used for OAB. The use for nocturia remains unclear.
4. Implantable neuromodulation therapy (InterStim) has been helpful.
5. Electrical stimulation may be used to teach the proper use of pelvic muscles.

B. Client teaching: *See Client Teaching Guide for Chapter 3, "Safety Issues: Fall Prevention," and the Client Teaching Guide for this chapter, "Nocturia."*
1. Limit fluid intake at night, especially alcohol and caffeine.
2. Reduce dietary salt intake especially late in the day.
3. Move the nighttime diuretic dose to the midafternoon if the client requires twice-daily diuretic medications.
4. If edema and venous stasis in the lower extremities are a problem, have client wear compression stockings and elevate legs during the waking hours and remove stockings prior to retiring.
5. Maintain blood glucose control.

C. Pharmaceutical therapy:
1. Oxybutynin (Ditropan) 5 to 15 mg tablets: anticholinergic medicine that relaxes the detrusor muscle of the bladder. It is available in time-release tablets, as a syrup, as well as in transdermal patches.
a. A lower starting dose of 2.5 mg two or three times a day is recommended for the frail elderly.
b. May be combined with bladder volume training. It reduces wet nights in those with nocturnal enuresis.
2. Trospium chloride 20 mg tablet: anticholinergic medication that blocks cholinergic receptors that are found on muscle cells in the wall of the bladder. Use to treat OAB.
3. Solifenacin (VESIcare) 5 mg and 10 mg tablets: anticholinergic and selective antimuscarinic with fewer anticholinergic side effects.
4. Darifenacin (Enablex) 7.5 to 15 mg extended-release (ER) tablets: anticholinergic medication that relieves bladder spasms and treats OAB, and decreases bladder contractions and increases bladder capacity. The effects of darifenacin for nocturia vary.
5. Tolterodine (Detrol) 2 mg tablet: The dose may be lowered to 1 mg based on individual response and tolerability. Anticholinergic/antimuscarinic functions much like oxybutynin antagonist that is available in both short- and long-acting forms.
6. Fesoterodine (Toviaz) 4 and 8 mg: anticholinergic, muscarinic receptor antagonist. The recommended starting dose is 4 mg once a day (maximum 8 mg/d). The daily dose should not exceed 4 mg in the following populations:
a. Clients with severe renal impairment (creatinine clearance [ClCr] <30 mL/min).
b. Clients taking potent CYP3A4 inhibitors, such as ketoconazole, itraconazole, and clarithromycin.
i. Fesoterodine is not recommended for use in clients with severe hepatic impairment (Child-Pugh C).
7. Desmopressin (DDAVP) 0.1 to 0.4 mg tablets, subcutaneous (SC), intravenous (IV), and nasal spray: antidiuretic; for nocturnal polyuria and vasopressin disorder. Immediate reassessment for effectiveness is recommended.
8. Hydrochlorothiazide (HCTZ) and furosemide antidiuretics: Use if the client is unable to tolerate DDAVP. Used as a second-line treatment option. Give 6 to 8 hours before bedtime.
9. Cyclooxygenase-2 inhibitor has been used occasionally due to its effect on urine production.
10. Botulinum toxin: Use only as refractory to first-line treatment. Further adjuvant therapy must be under direction after a full evaluation.
11. Melatonin OTC: Using a combination treatment may mitigate rather than reduce symptoms.

FOLLOW-UP

A. Follow-up is determined by the client's needs, severity of symptoms, and whether complications are present. Adequate time should be allowed for treatments involving the nonpharmacologic and pharmacologic approach.

CONSULTATION/REFERRAL

A. Consider referral to a urologist.
B. Refer to a pulmonologist if client is positive for nocturnal hypoxemia.
C. Urogynecologist for females with a structural problem and pregnancy.
D. Neurology.
E. Endocrinology.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Older clients may not present with classic symptoms; instead they may present with a history of falls, incontinence, and mental confusion.

2. Avoid the use of anticholinergics if glomerular filtration rate (GFR) is less than 30.

3. Melatonin secretion is impaired in the elderly, which can contribute to disruption of the circadian rhythm of micturition, leading to nocturia.

4. Geriatric syndrome assessments:

a. Falls: Nocturia in the elderly is a risk factor for falls. Many falls occur en route to the bathroom, especially at night.

i. Avoid consumption of diuretics in the hours prior to bedtime (caffeine, alcohol, diuretic medications). Avoid fluids 1 to 2 hours before bedtime.

ii. Safety measures to reduce falls in the home include removing small area carpets, keeping night lights along the path to the bathroom, and installing a toilet seat with grab bars or nearby grab bars.

iii. Durable medical equipment such as a bedside commode or a urinal should be considered.

b. Delirium/behavioral disturbance: Older clients given anticholinergic medications for bladder symptoms may experience worsening cognitive impairment or delirium.

c. Depression: Nocturia is associated with a decreased quality of life.

d. Constipation: Fecal impaction is a risk factor for nocturia and urinary incontinence. Constipation is a common side effect of anticholinergic and antispasmodic drugs.

5. Pharmacologic caution in geriatrics:

a. Antimuscarinic medications may increase confusion and should be avoided in frail older adults or adults with dementia and are contraindicated in people with narrow angle-closure glaucoma. Ask about the effectiveness of the medications versus the appearance of side effects such as constipation, dry eye/mouth, blurred near-vision, tachycardia, drowsiness, or reduced cognitive function. Carefully consider benefits versus risks in older adults.

b. First-generation alpha-1 blockers are linked with falls in the older adults due to orthostatic hypotension. Educate older males prescribed alpha-1 blockers for BPH to use caution, getting up slowly from seated or lying positions. Avoid first-generation alpha-1 blockers (Hytrin, Cardura) in older males with a history of syncope.

6. Beers list caution: Avoid use of desmopressin for nocturia or nocturnal polyuria; high risk for hyponatremia in older adults. Desmopressin is a potentially inappropriate medication to use in the elderly; safer alternative treatments are available.

RESOURCE

National Association for Continence (NAFC): http:// www.nafc.org/nocturia

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PROSTATITIS

DEFINITION

A. Prostatitis is an acute or chronic infection of the prostate gland. Prostatitis is the most important cause of urinary infection in males.

1. Clients can be considered to be in the early stages of the disease if they have experienced persistent, recurrent symptoms for less than 6 months and are antibiotic-naïve.

2. Clients can be considered in the later stages of the disease if they have persistent, recurrent symptoms for ≥3 of the previous 6 months and are refractory to initial lines of pharmacotherapy (Table 15.3).

TABLE 15.3 NIH CLASSIFICATION OF PROSTATITIS

NIH Classification	Definition
Bacterial prostatitis	Acute infection of the prostate gland
CBP	Chronic or recurrent infection of the prostate
CP/CPPS • Inflammatory CPPS[a] • Noninflammatory CPPS[a]	No demonstrated infection White blood cells in semen and/or EPS or VB3 after prostatic massage No white cells in semen/EPS/VB3
Asymptomatic inflammatory prostatitis	No subjective symptoms detected Inflammation shown either by prostate biopsy or the presence of white cells in EPS/semen during evaluation for infertility or other disorders

[a]During CP/CPPS, it is possible for clients to switch between the two subcategories), but this has little effect on subsequent clinical management.

CBP, chronic bacterial prostatitis; CP, chronic prostatitis; CPPS, chronic pelvic pain syndrome; EPS, expressed prostatic secretions; NIH, National Institutes of Health; VB, voided bladder; VB3, postprostatic massage voided bladder urine.

Source: Rees, J., Abrahams, M., Doble, A., Cooper, A., & the Prostatitis Expert Reference Group. (2015). Diagnosis and treatment of chronic bacterial prostatitis and chronic prostatitis/chronic pelvic pain syndrome: a consensus guideline. *BJU International, 116*(4), 509–525. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5008168/.

INCIDENCE

A. About 50% of adult males in the United States will be treated for prostate conditions during their lifetime.
B. Acute and chronic bacterial prostatitis (CBP) occurs in about 1 in 10 males.
C. Nonbacterial prostatitis occurs in about 6 in 10 males.
D. Prostatodynia occurs in about 3 in 10 males.

PATHOGENESIS

A. Nonbacterial prostatitis is an inflammatory condition with an unknown etiology. Infection results in prostatitis in four ways:
1. Ascending infection of urethra.
2. Reflux of infected urine into the prostate through ejaculatory and prostatic ducts that empty into the prostatic urethra.
3. Hematogenous spread causing bacterial prostatitis.
4. Invasion by rectal bacteria through direct extension or lymph system spread.
B. Causative organisms: *Escherichia coli, Klebsiella, Pseudomonas, Enterococci, Ureaplasma, Gardnerella vaginalis, Trichomonas vaginalis, Chlamydia trachomatis, Chlamydia, Mycoplasma*, or *Neisseria gonorrhoeae. Cytomegalovirus (CMV), Mycobacterium tuberculosis*, and fungi have been associated with prostatitis in HIV-infected clients.
C. Incubation period depends on pathogen.

PREDISPOSING FACTORS

A. More common in younger and middle-aged males.
B. Sexual transmission of bacteria.
C. Neuromuscular dysfunction.
D. Structural voiding dysfunction.
E. Benign prostatic hypertrophy (BPH).
F. History of allergies and asthma (increase in nonbacterial prostatitis).
G. Genetics.
H. Ethnicity; more common in African Americans.
I. Socioeconomic conditions such as lower socioeconomic status and/or those experiencing homelessness.

COMMON COMPLAINTS

A. Dysuria.
B. Perineal, rectal, or suprapubic pain (chronic pain syndrome).
C. Less urine flow.
D. Spiking fever.
E. Back pain.
F. Sexual dysfunction symptoms:
1. Erectile dysfunction (ED).
2. Ejaculatory dysfunction (premature, delayed, or pain during or after ejaculation).
3. Decreased libido.

OTHER SIGNS AND SYMPTOMS

A. Acute bacterial prostatitis:
1. Fever and chills, malaise.
2. Acute onset of dysuria.
3. Hesitancy.
4. Urinary frequency and low back pain.
5. Pain with intercourse and with defecation.
6. Initial or terminal hematuria and edema with acute urinary retention.
7. Arthralgia or myalgia.
8. Nocturia.
9. Neuropathic pain.
B. CBP:
1. Usually presents with recurrent urinary tract infection (UTI).
2. May be asymptomatic between acute episodes; some males have large fluctuation in symptom severity.
3. Perineal, inguinal, or suprapubic pain, or irritative symptoms on voiding such as frequency and urgency.
4. Hematuria, hematospermia, or painful ejaculations.
5. Prostatic calculi.
C. Nonbacterial prostatitis (most common):
1. Vague discomfort to pain: prostatic, lower back, perineum, groin, scrotum, or suprapubic pain; ejaculatory pain.
2. Dysuria, urinary frequency, urgency, hesitancy, and decreased urine flow.
3. Penile discharge, especially noted during the first bowel movement (BM) of the day.
4. Sexual difficulty.
5. Low sperm count.
6. Blood or urine in ejaculate.
D. Asymptomatic inflammatory prostatitis is found when looking for causes of infertility and testing for prostate cancer.
E. Prostatodynia (cause is unknown):
1. Prostate irritation.
2. Pain and discomfort in the prostate, testicles, penis, and urethra.
3. Difficulty urinating.

SUBJECTIVE DATA

A. Ask the client to complete the National Institutes of Health Chronic Prostatitis Symptom Index (NIH-CPSI) self-evaluation form (Table 15.4). The assessment tool evaluates pain, urinary symptoms, and the impact on quality of life. An online NIH symptom index that self-scores is available at www.prostatitis.org/symptomindex.html.
1. Mild = 0 to 14 total score.
2. Moderate = 15 to 29 total score.
3. Severe = 30 to 43 total score.
B. Review the onset, duration, and course of symptoms.
C. Are there any other symptoms, such as discharge, pain, hematuria, hesitancy, back pain, or weight loss?
D. Has the client ever had the same symptoms? If so, how were they treated?
E. Does any sexual partner(s) have any symptoms, lesions, or known sexually transmitted infections (STIs)?
F. Does the client engage in anal intercourse?
G. Has the client noted any impaired urinary flow?
H. Has the client required any recent urethral catheterization or instrumentation?

PHYSICAL EXAMINATION

A. Check temperature and blood pressure (BP).
B. Inspect:
1. Examine the client generally for discomfort before and during examination.
2. Check the urethral meatus for discharge.
3. Retract foreskin (if present) and assess for hygiene and smegma.
4. Check the shaft of the penis, glans, and prepuce for lesions.
C. Palpate:
1. Palpate testes and epididymides for inflammation, tenderness, and masses; palpate scrotum for hydrocele or varicocele.
2. Check back for costovertebral angle (CVA) tenderness. ▶

TABLE 15.4 NATIONAL INSTITUTES OF HEALTH CHRONIC PROSTATITIS SYMPTOM INDEX

Pain or Discomfort	Yes	No
1. In the past week, have you experienced any pain or discomfort in the following areas?		
a. Area between rectum and testicles (perineum)	☐1	☐0
b. Testicles	☐1	☐0
c. Tip of the penis (not related to urination)	☐1	☐0
d. Below your waist, in your pubic or bladder area	☐1	☐0
2. In the past week, have you experienced:		
a. Pain or burning during urination?	☐1	☐0
b. Pain or discomfort during or after sexual climax (ejaculation)?	☐1	☐0

3. How often have you had pain or discomfort in any of these areas over the past week?
   - ☐ 0 Never
   - ☐ 1 Rarely
   - ☐ 2 Sometimes
   - ☐ 3 Often
   - ☐ 4 Usually
   - ☐ 5 Always

4. Which number best describes your average pain or discomfort, on the days that you had it, over the past week?
   ☐☐☐☐☐☐☐☐☐☐☐
   0 1 2 3 4 5 6 7 8 9 10
   0 = No pain; 10 = Pain as bad as you can imagine

Urination

5. How often have you had a sensation of not emptying your bladder completely after you finished urinating, over the past week?
   - ☐ 0 Not at all
   - ☐ 1 Less than 1 time in 5
   - ☐ 2 Less than half the time
   - ☐ 3 About half the time
   - ☐ 4 More than half the time
   - ☐ 5 Almost always

Urination

6. How often have you had to urinate again less than 2 hours after you finished urinating, over the past week?
   - ☐ 0 Not at all
   - ☐ 1 Less than 1 time in 5
   - ☐ 2 Less than half the time
   - ☐ 3 About half the time
   - ☐ 4 More than half the time
   - ☐ 5 Almost always

Impact of Symptoms

7. How much have your symptoms kept you from doing the kinds of things you would usually do, over the past week?
   - ☐ 0 None
   - ☐ 1 Only a little
   - ☐ 2 Some
   - ☐ 3 A lot

8. How much did you think about your symptoms, over the past week?
   - ☐ 0 None
   - ☐ 1 Only a little
   - ☐ 2 Some
   - ☐ 3 A lot

Quality of Life

9. If you were to spend the rest of your life with your symptoms just the way they have been during the past week, how would you feel about that?
   - ☐ 0 Delighted
   - ☐ 1 Pleased
   - ☐ 2 Mostly satisfied
   - ☐ 3 Mixed (about equally satisfied and dissatisfied)
   - ☐ 4 Mostly dissatisfied
   - ☐ 5 Unhappy
   - ☐ 6 Terrible

Scoring the NIH-CPSI Domains:

Pain: Total of items 1a, 1b, 1c, 1d, 2a, 2b, 3, and 4 = _____

Urinary symptoms: Total of items 5 and 6 = _____

Quality-of-life impact: Total of items 7, 8, and 9 = _____

NIH-CPSI, National Institutes of Health Chronic Prostatitis Symptom Index.

3. Evaluate for an enlarged, tender bladder caused by urinary retention.
4. Palpate the abdomen for masses, urinary distension, suprapubic tenderness, and organomegaly.
5. Palpate inguinal lymph nodes, check the inguinal and femoral areas for bulges and hernias, have the client bear down and cough, and reexamine them.
6. Rectal exam:
   a. Prior to the rectal examination, have the client obtain a clean-catch urine specimen for culture. Check for symmetry, swelling, tenderness, and enlarged prostate.
   b. In acute prostatitis, rectal examination reveals the prostate gland to be exquisitely tender and boggy.
   c. A fluctuant prostatic mass suggests an abscess that may require surgical intervention.
   d. Perform prostate massage for postmassage urine sample (see Section II: Procedures, "Prostatic Massage Technique: Two-Glass Test").

DIAGNOSTIC TESTS

A. Acute infection:
1. Complete blood count (CBC) with differential.
2. Urinalysis and urine culture.
3. Culture for STIs.
4. The four-glass test is considered the diagnostic standard but is not practiced in a clinical setting; the two-glass premassage and postmassage test is more commonly used.
5. Gram stain, culture of expressed prostatic specimen (EPS):
   a. **Avoid vigorous massage when obtaining specimen due to the risk of inducing bacteremia.**
   b. Gram stain of EPS demonstrates infectious organisms or white blood cells (WBCs) typical of an immune response (>10 WBC/high-power microscope field [HPF] is abnormal). Clients with abnormal WBC but no bacterial growth may have chlamydial or *Ureaplasma* infection and need to be tested or treated empirically.

B. Chronic infection:
1. Blood urea nitrogen (BUN).
2. CBC with differential.
3. Creatinine.
4. Uroflowmetry, retrograde urethrography, or cystoscopy to exclude bladder outlet obstruction, urethral stricture, or bladder neck stenosis.

C. CBP:
1. If recurrent infections are confirmed, evaluate for structural or functional abnormality with CT scan.
2. Measure residual urine after voiding.
3. If no urologic abnormalities are found and repeated cultures indicate the same bacterial strain, CBP is likely.

D. Other tests such as ultrasound, MRI, and biopsies are used as required to rule out other pathology.

DIFFERENTIAL DIAGNOSES

A. Prostatitis:
1. Acute: readily evident by clinical presentation and exam.
2. Chronic: more difficult to diagnose. The hallmark symptom is recurrent UTI. It often resembles prostatic hyperplasia, strictures, and prostatic carcinoma.
3. Chronic pelvic pain syndrome (CPPS).

B. Pyelonephritis.
C. Epididymitis.
D. Anal fistulas and fissures.
E. BPH causes urinary retention due to obstruction.
F. Urethral stricture or stone.
G. Chronic pain syndromes/back pain.

PLAN

A. General interventions:
1. Clients with acute prostatitis may need hospitalization and intravenous therapy for severe infection (high fever, increased WBC, dehydration). Less toxic clients may be treated on an outpatient basis.
2. Older clients without evidence of infection with lower tract symptoms should have urine cytology to rule out malignancy.
3. Review the client's current medications. Discontinue or reduce the dosage (if possible) of the client's anticholinergics, sedatives, and antidepressants because they may impair bladder function.

B. Client teaching: *See Client Teaching Guide for this chapter, "Prostatitis."*
1. Having the client self-massage to reduce symptoms is questionable; the massage of an acutely infected gland is contraindicated due to the risk of bacteremia.
2. Recommend sitz baths two to three times daily.
3. Stool softeners may be needed.

C. Dietary management:
1. Increase fluid intake.
2. Decrease caffeine and alcohol, which can irritate the urethra.

D. Pharmaceutical therapy: Full treatment guideline for the "Diagnosis and treatment of chronic bacterial prostatitis and chronic prostatitis/chronic pelvic pain syndrome: A consensus guideline" is available with open access through the Wiley Online Library at onlinelibrary.wiley.com/doi/abs/10.1111/bju.13101.
1. Pain relief:
   a. Analgesics:
      i. Nonsteroidal anti-inflammatory drugs (NSAIDs) should be offered only for short-term treatment of pain to clients with early stage CBP or chronic prostatitis (CP)/CPPS whose symptoms are suspected to be caused by an inflammatory process/flare. NSAIDs should be stopped within 4 to 6 weeks of treatment if they do not reduce symptoms.
      ii. Opioids should not be used in clients with early-stage CBP or CP/CPPS due to the risk of dependency.
      iii. If pain is considered neuropathic in origin, treatment may include a gabapentinoid (e.g., pregabalin or gabapentin), a tricyclic antidepressant (e.g., amitriptyline, nortriptyline, or trimipramine), or a selective serotonin and norepinephrine reuptake inhibitor (SNRI; e.g., duloxetine).
      iv. When pain is severe and refractory to the treatments or is significantly impairing the client's quality of life in daily activities, referral to a pain specialist should be considered.
2. Alpha-adrenergic antagonists:
   a. Due to the adverse side effect profiles, consider offering uroselective alpha-adrenergic antagonists (e.g., tamsulosin, alfuzosin, and silodosin) as first-line treatment in clients with CBP and CP/CPPS who present with voiding lower urinary tract symptoms (LUTS).
3. Antibiotics: Antimicrobial therapy should be guided by bacterial cultures and sensitivities, taking into consideration drug interactions and/or contraindications. Dose and duration should be sufficient to eradicate the infection. ▶

Treatment typically consists of 4 to 8 weeks of antibiotic therapy but may need to be extended to 12 weeks.

a. First-line therapy for early-stage CBP and CP/CPPS, offer a quinolone:
   i. Ciprofloxacin 500 mg BID × 4 weeks.
   ii. Ofloxacin 400 mg BID × 4 to 6 weeks.
   iii. Levofloxacin 500 to 750 mg daily × 4 weeks.

b. Second-line therapy:
   i. Trimethoprim-sulfamethoxazole (TMP-SMX): 1 double strength (DS) BID × 14 days or 2 to 3 months if chronic infection.
   ii. Tetracyclines: doxycycline 100 mg BID × 4 weeks.

c. Macrolides are reserved for special indications, based on advice from microbiological findings.

4. There is insufficient evidence to warrant recommending 5 alpha-reductase inhibitors as monotherapy in CP/CPPS, unless coexisting BPH is present.

FOLLOW-UP

A. See the client in 2 to 10 days depending on the client's symptoms and course.

B. Culture urine at completion of drug therapy. Test of cure for antibiotics requires elimination of bacteria from prostatic fluid to prevent chronic flares. Some clients may not achieve cure even after 6 to 12 weeks of therapy.

C. Clients who achieve a partial response may be given a second course of antibiotics. Those failing to demonstrate an organism may benefit from a course of doxycycline or erythromycin for *Chlamydia* and/or *Ureaplasma* coverage.

EMERGENT ISSUES/INSTRUCTIONS

A. If the client is unable to void from urinary retention, they should go to the hospital for catheterization.

CONSULTATION/REFERRAL

A. Obtain a referral to a urologist for recurrent acute bacteria prostatic infections or infections that persist.

B. Cystoscopy may be required to rule out interstitial cystitis (IC).

C. Urinary retention concomitant with acute prostatitis may require hospitalization.

INDIVIDUAL CONSIDERATIONS

A. Pharmacologic caution in geriatrics:

1. Fluoroquinolone use in the elderly has the potential to cause neuropsychiatric symptoms, including seizures to worsening dementia.

2. CP causes a significant decrease in storage function in the elderly, such as an increase in detrusor overactivity.

RESOURCES

American Urological Association (AUA): www.auanet.org

National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK): www.niddk.nih.gov

The Prostatitis Foundation: www.prostatitis.org

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PROTEINURIA

DEFINITION

A. Proteinuria is excess protein (albumin) in urine. Proteinuria may be an incidental finding and have no symptoms. Use of a urine dipstick for screening is acceptable for first detecting proteinuria (Table 15.5); however, the dipstick should not be used to quantify the amount of urinary protein. Protein concentration is a function of urine volume as well as the quantity of protein present.

B. The measurement of protein excretion is used to establish the diagnosis and to follow the course of glomerular disease. The normal rate of albumin excretion is less than 30 mg/d; the rate is about 4 to 7 mg/d in healthy young adults and increases with age and an increase in body weight. Persistent albumin excretion between 30 and 300 mg/d is called microalbuminuria. Values of 300 mg/d of protein are considered overt proteinuria or macroalbuminuria.

C. When proteinuria coexists with hematuria, the likelihood of clinically significant renal disease is high. In clients with diabetes, microalbuminuria usually indicates incipient diabetic nephropathy. In nondiabetics, the presence of microalbuminuria is associated with left ventricular hypertrophy and cardiovascular (CV) disease. Protein is also the cardinal sign of pregnancy-induced hypertension (PIH).

D. Functional/transient proteinuria is associated with fever, exercise, dehydration, cold exposure, and stress, and is not associated with underlying renal disease. Orthostatic proteinuria, a transient proteinuria condition, is related to postural changes that affect the glomerular hemodynamics. Orthostatic proteinuria rarely exceeds 1 g/d. Significant renal disease is not usually found upon further testing and workup.

E. Persistent proteinuria is defined as greater than 4 mg/m^2/hr of protein in a 24-hour urine collection or greater than 0.02 mg/mg of protein creatinine ratio on a spot urine. Persistent proteinuria requires further evaluation to rule out underlying renal pathology. There are four basic types of proteinuria:

1. Glomerular proteinuria (albuminuria) caused by increased filtration of macromolecules across the glomerular capillary wall. The standard urine dipstick is able to detect glomerular proteinuria. Some causes of glomerular proteinuria include diabetes; hypertension (HTN); nephrotic syndrome; infections including hepatitis, HIV, cytomegalovirus (CMV), malaria, syphilis, and streptococcal infections; chemotherapeutic agents; Alport syndrome; and hemolytic uremic syndrome:

a. In nonnephrotic proteinuria, the amount of proteinuria is <3.5 g per 24 hours and is persistent. These clients require close follow-up and may need a renal biopsy if they have abnormal urine microscopy results and/or impairment of kidney function.

b. Orthostatic proteinuria is characterized by increased protein excretion in the upright position but normal protein excretion when the client is supine. The exact ▶

mechanism is unclear. Total protein excretion is generally less than 1 g/d; however, in orthostatic proteinuria it may exceed 3.5 g/d in selected clients.

c. **Nephrotic range proteinuria is defined as >3.5 g of proteinuria on a spot urine protein-to-creatinine ratio. This finding denotes significant glomerular disease and requires a renal biopsy for diagnosis and management.**

2. Tubular proteinuria is nonglomerular (i.e., nonalbumin form of protein), related to interference with proximal tubular reabsorption. The amount of proteinuria is <2 g and urinary dipstick is generally unable to detect tubular proteinuria. Some causes of tubulointerstitial proteinuria include toxins, pyelonephritis, nonsteroidal anti-inflammatory drugs (NSAIDs), antibiotics, and inherited causes such as Lowe syndrome and Wilson disease.

3. Secretory (overflow) proteinuria is nonglomerular (i.e., nonalbumin form of protein). Increased excretion from the tubules is secondary to an overproduction of a particular protein, most commonly noted in interstitial nephritis. A urinary dipstick is unable to detect overflow protein.

4. Postrenal proteinuria: Inflammation in the urinary tract and urinary tract infection (UTI) can cause increases in urinary protein excretion. The excreted proteins are often nonalbumin, and only small amounts are excreted. Leukocyturia is frequently present. Clients with nephrolithiasis or tumors of the urinary tract may also have proteinuria.

5. It is currently recommended that a diagnosis of kidney damage can only be made if at least two separate measurements are elevated.

INCIDENCE

A. Approximately 6.1% of males and 9.7% of females have proteinuria detected by a single routine dipstick test.

B. The prevalence of proteinuria is higher in the elderly and in clients with comorbidities.

1. The prevalence of proteinuria starts increasing at 40 years of age.

2. The incidence of HTN and diabetes increases with age. In consequence, the incidence of persistent proteinuria (and microalbuminuria) also increases with age.

3. The prevalence of microalbuminuria is 28.8% in persons with diabetes and 16% with HTN.

4. The incidence of microalbuminuria is only 5.1% in clients without diabetes, HTN, CV disease, or elevated serum creatinine levels.

TABLE 15.5 DIPSTICK ANALYSIS: DETECTING AND QUANTIFYING PROTEINURIA

Dipstick Grade	Quantity of Protein
Negative	<10 mg/dL
Trace	10–20 mg/dL
1+	15–30 mg/dL
2+	40–100 mg/dL
3+	150–300 mg/dL
4+	>500 mg/dL

PATHOGENESIS

A. Pathogenesis depends on the underlying etiology. An alteration in glomerular filtration that increases excretion (filtration) of plasma proteins occurs. Increased glomerular permeability, increased production of abnormal proteins (Bence Jones protein), decreased tubular reabsorption, surgical traumas, and infections may increase urinary protein. Urinary protein may also be affected by dietary protein intake.

PREDISPOSING FACTORS

A. Fever.
B. Age 65 years or older.
C. Ethnic groups including African Americans, Hispanics, Asians, and American Indians.
D. Increased exercise.
E. UTI/pyelonephritis.
F. Medications:
1. Penicillamine.
2. NSAIDs.
3. Angiotensin-converting enzyme (ACE) inhibitors.
4. Aminoglycosides.
5. Cisplatin.
6. Amphotericin B.
7. Quinolones.
8. Sulfonamides.
9. Cimetidine (Tagamet).
10. Allopurinol (Zyloprim).
11. Antiretroviral drugs can be nephrotoxic.
G. Heavy metal exposure:
1. Gold.
2. Cadmium.
3. Mercury.
4. Lead.
5. Copper.
H. Collagen vascular disease or vasculitis.
I. Family history of proteinuria or pyelonephritis.
J. HTN.
K. Renal disease.
L. Congestive heart failure (CHF) or endocarditis.
M. Diabetes.
N. Lupus.
O. Infections:
1. HIV.
2. Syphilis.
3. Hepatitis B and C.
4. Group A beta-hemolytic *Streptococcus*.
5. Viral infection (e.g., mononucleosis).
6. Malaria.
P. Malignancy:
1. Lymphoma.
2. Hodgkin disease.
3. Breast tumor.
4. Lung tumor.
5. Colon tumor.
Q. Heroin use.
R. PIH.
S. Radiocontrast media.
T. Elderly.

COMMON COMPLAINTS

A. Asymptomatic.
B. Increased weight.
C. Decreased urine output.

OTHER SIGNS AND SYMPTOMS

A. Edema: periorbital, presacral, genital, or ankle.
B. Nephrotic syndrome: hypercholesterolemia and hypertriglyceridemia.
C. Protein malnutrition: anorexia and vomiting.
D. "Frothy" urine.

SUBJECTIVE DATA

A. Review the onset, course, and duration of presenting complaints.
B. Question the client concerning urinary output, thirst or fluid intake, edema, or increase in weight. Establish usual weight history.
C. Establish the first day of the client's last period. Are they pregnant? If so, what is the fetus gestational age? Is there edema, HTN, headache, visual changes (scotoma), hyperreflexia, and/or right upper quadrant pain?
D. Review the client's medical history for renal disease, diabetes, CHF, systemic disorders such as lupus, and substance abuse.
E. Does the client have signs or symptoms of a UTI or pyelonephritis?
F. Review the client's recent history for exertion, emotional stress, surgical trauma, fever, and any acute illness.
G. When was the client's last evaluation for cholesterol? Are they on any special diet?
H. Review medication list, including prescribed and over-the-counter (OTC) medications and herbal products.
I. Review the client's occupational exposure, smoking history, and risk factors for infectious diseases.

PHYSICAL EXAMINATION

A. The client's physical examination may have few abnormalities unless there are features of multisystem disease.
B. Check temperature (if indicated), erect and supine blood pressure (BP), pulse, respirations, and weight (massive weight gain because of fluid is very common).
C. Inspect:
  1. Inspect overall general appearance for edema (pedal, hand, facial, or periorbital edema), butterfly rash (lupus), or ascites.
  2. Evaluate for protein wasting.
  3. Evaluate for jugular vein distension.
  4. Funduscopic exam: Evaluate for retinopathy.
  5. Inspect for pharyngitis.
D. Auscultate:
  1. Assess heart.
  2. Assess lungs (may have decreased breath sounds due to pleural effusions).
E. Palpate:
  1. Examine the abdomen; evaluate bladder distension, suprapubic tenderness, masses, or ascites; check abdominal tenderness.
  2. Palpate for costovertebral angle (CVA) tenderness.
  3. Check deep tendon reflexes, especially in those who are pregnant.

DIAGNOSTIC TESTS

A. Urine dipstick is a screening tool in the outpatient setting.
  1. The dipstick result may be difficult to read if the urine is abnormally colored due to nitrofurantoin, riboflavin, azo-containing sulfonamide antimicrobials, or blood.
B. Single void "spot" urine testing:
  1. The first urine specimen of the morning is optimal and is guideline-recommended. Evaluation of the first morning specimen excludes any postural effect on the protein component.
  2. The gold standard for measurement of protein excretion is a 24-hour urine collection but is now being replaced by the easier to obtain and less complicated spot test. The 24-hour urine is considered impractical for generalized testing.
C. Polymerase chain reaction (PCR) test or albumin-to-creatinine ratio (ACR) test on a first morning or a random spot specimen. The PCR or ACR is useful in following trends in the client's proteinuria.
D. Complete blood count (CBC) and serum electrolytes.
E. Serum creatinine.
F. Estimated glomerular filtration rate (eGFR): uses a blood creatinine level along with age and values assigned to gender and race to calculate the estimate rate of urine filtration. The eGFR rate decreases with progressive kidney damage.
G. Lipid profile (high-density lipoprotein [HDL] and low-density lipoprotein [LDL]).
H. Blood urea nitrogen (BUN): serves as an index of renal excretory capacity. Urea is the nitrogenous end product of protein metabolism.
I. Total protein.
J. Urinalysis, urine culture, and sensitivity (if indicated).
K. Ultrasound of the full urinary tract.
L. Screen for diabetes and other testing related to physical findings.
M. Renal biopsy is required to establish the diagnosis in most cases.
N. Hepatitis B, hepatitis C, and HIV serologies, if indicated.

DIFFERENTIAL DIAGNOSES

A. See section "Predisposing Factors."

PLAN

A. General interventions:
  1. Current guidelines for evaluation screening of albuminuria/proteinuria vary by country, but recommendations include screening at-risk individuals with diabetes, HTN, and/or obesity; those who are smokers, are members of indigenous populations, have a family history of chronic kidney disease (CKD), and/or older than 50 years; and/or have structural renal tract disease, renal calculi, benign prostatic hyperplasia, vascular disease, and autoimmune disease.
  2. Management for nephrotic syndrome includes diet with sodium and protein restriction, loop and distal acting diuretics, control of cholesterol (low-saturated fat, low-cholesterol diet), lipid-lowering agents, pneumococcal and influenza vaccines to prevent infections, and use of steroids and immunosuppressive agents as necessary.
  3. Clients with hematuria and proteinuria need a 24-hour urine collection for protein and creatinine clearance (ClCr).
B. Client teaching:
  1. Encourage low-fat/low-cholesterol diet if hyperlipidemia is present. ▶

2. Encourage sodium- and protein-restricted diet for nephrotic syndrome.

C. Pharmaceutical therapy:

1. There is no specific drug therapy for excess protein.

2. Use drug therapy appropriate to the underlying renal or nonrenal cause and, in particular, whether or not the injury is immune mediated.

3. In clients with CKD, the administration of ACE inhibitors and/or angiotensin II receptor blockers (ARBs) is aimed at reducing the degree of proteinuria.

4. For clients with nephrotic syndrome, the Advisory Committee on Immunization Practices (ACIP) recommends immunization for pneumonia for those not previously immunized with the pneumococcal vaccine as follows:

a. One dose of pneumococcal conjugate vaccine 15 (PCV15) or PCV20. If PCV15 given, administer pneumococcal polysaccharide vaccine (PPSV23) 1 year later. If PCV20 is used, PPSV23 is not indicated.

b. Clients who have received PPSV23 only need one dose of PCV15 or PCV20 1 year after the PPSV23 is given. If they received PPSV23, a second dose of PPSV is not recommended.

c. Clients who have received PCV13, with or without PPSV23, administer PPSV23.

FOLLOW-UP

A. If proteinuria is found on a dipstick and the first morning test results are trace or negative for protein, repeat a first morning test in 1 year. It is recommended that a diagnosis of albuminuria be made only after at least three of the client's urine samples over a 6-month period fall above normal range.

B. There is no consensus on how often to screen for proteinuria. Guidelines include annual screening (especially for diabetics), every 5 years for clients older than 50 years or smokers, and every 3 years for clients who have HTN, obesity, or a family history of kidney disease. Monitoring should always include BP, quantitative testing by PCR or ACR, and a serum creatinine.

C. Clients should be assessed at routine examination appointments for vasculitic skin changes, rashes, retinopathy, lymphadenopathy, signs of heart failure, abdominal masses, organomegaly, guaiac stools, prostatic enlargement, and joint inflammation.

CONSULTATION/REFERRAL

A. Consider client referral if kidney damage is progressing from medical disease (systemic lupus erythematosus, HTN, diabetes).

B. Refer the client as necessary; HTN is a poor prognostic sign for significant renal impairment.

C. Consultation for diagnostic tests to be considered: kidney, ureter, and bladder (KUB); intravenous pyelography (IVP); renal ultrasonography; renal biopsy.

D. Referral to a nephrologist should be considered if a definitive diagnosis is required or a renal biopsy is considered.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Protein excretion is considered abnormal in pregnancy when it exceeds 300 mg per 24 hours or greater than 0.2 g of protein per gram of creatinine in a random urine specimen. A urine specimen with 1+ protein is considered the cutoff for proteinuria.

2. The gestational age at which proteinuria is first documented is important in establishing the likelihood of PIH versus other renal disease. Proteinuria prior to or early in pregnancy suggests preexisting renal disease.

3. Monitor urine protein and BP at each prenatal visit and refer the client if urinary protein remains elevated.

4. Monitor for intrauterine growth restriction (IUGR).

5. Proteinuria (or HTN) that persists longer than 3 months after delivery should be followed closely.

B. Geriatrics:

1. Renal function deteriorates in the elderly who may also have coexisting medical conditions (HTN, diabetes) that may cause nephropathy.

2. Refer to Appendix C, Table C.4 on nonantiinfective medications that should be avoided or reduced in older adults.

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PYELONEPHRITIS

DEFINITION

A. Pyelonephritis is an acute infection and inflammatory disease of the upper urinary tract (renal pelvis, tubules, and interstitial tissue) of one or both kidneys. Acute pyelonephritis is an ascending urinary tract infection (UTI) that has progressed from the lower urinary tract.

B. Fever has been strongly correlated with the diagnosis of acute pyelonephritis; therefore, clients with clinical symptoms of pyelonephritis in the absence of fever should be evaluated for alternative diagnoses.

C. Acute pyelonephritis characteristically causes some scarring to the kidney and may lead to significant damage, kidney failure, abscess formation, and sepsis. Antibiotic therapy is essential to prevent the progression of pyelonephritis.

INCIDENCE

A. Thirty percent of the female population has at least one UTI in their lifetime. Males demonstrate a gradual increase at age 35, peaking at 85 years old.

B. Annual rates of pyelonephritis in females are 15 to 17 cases per 10,000, and 3 to 4 cases per 10,000 in males.

C. The incidence of acute pyelonephritis in pregnancy is 20% to 30%. Most cases develop as a consequence of undiagnosed or inadequately treated lower UTI.

D. Upper UTIs are less common and more serious than lower tract infections. After puberty, the prevalence of UTIs increases slightly in females but remains low in males.
E. After age 65, UTIs are more common, with an equal incidence in both sexes.
F. Rates of bacteremia vary widely. Rates depend on host factors and are higher among clients who are severely ill, who are immunocompromised, who have a urinary tract obstruction, and who are 65 years of age or older.
G. 7% of pyelonephritis cases require hospitalization.

PATHOGENESIS

A. Pyelonephritis is caused by ascending infection from the bladder usually caused by *Escherichia coli* (75%–95%) and other gram-negative bacteria including *Proteus mirabilis* (1%–2%), *Klebsiella pneumoniae* (1%–2%), *Enterobacter* (<1%), and Group B *Streptococcus* (GBS: <1%). Gram-positive causative agents are less common; 10% to 15% of cases are caused by *Staphylococcus saprophyticus.*
B. Bacteria can also reach the kidneys through the bloodstream from intravenous (IVI) drug abuse and endocarditis.
C. In females, the short urethra in close proximity to the perirectal area makes colonization possible. In pregnancy, the increased glycosuria, increase in urinary amino acids, urinary stasis, and the presence of vesicoureteral reflux facilitate bacterial growth.
D. In males, benign prostatic hyperplasia (BPH) causing bladder obstruction is a common pathology.
E. Indwelling catheters increase ascending infections and pyelonephritis.

PREDISPOSING FACTORS

A. Previous UTI, cystitis, and pyelonephritis.
B. Sickle cell disease.
C. Diabetes.
D. Urinary catheterization.
E. Obstruction: calculi, tumors, and urethral strictures.
F. Neurogenic bladder disease: strokes, multiple sclerosis (MS), and spinal cord injuries.
G. Urinary reflux.
H. HIV.
I. Trauma.
J. Chronic constipation/fecal impaction.
K. Incomplete bladder emptying related to medications (e.g., anticholinergics).
L. Sex:
1. Females:
   a. Increased sexual activity, failure to void after intercourse, diaphragms, and nonoxynol-9 spermicide.
   b. Pregnancy.
   c. Atrophic vaginal mucosa predisposes to the colonization of pathogens and UTIs.
   d. Cystocele.
2. males:
   a. Homosexuality.
   b. Sexual partner with colonization.
   c. Obstruction: prostatic hyperplasia.
   d. Age 50 years or older.
   e. Acute or chronic bacterial prostatitis.

COMMON COMPLAINTS

A. Shaking, chills, and fever (fever is not always present).
B. Flank pain or tenderness.
C. Urinary frequency or urgency.
D. Costovertebral angle (CVA) tenderness (may be mild, moderate, or severe).
E. Guarding.
F. Urinary frequency, nocturia, hematuria, and dysuria (up to 20% of clients do have bladder present in upper tract infections).
G. Gross hematuria (hemorrhagic cystitis) occurs in 30% to 40% of females; however, gross hematuria is unusual in males with pyelonephritis.
H. Malaise.

OTHER SIGNS AND SYMPTOMS

A. Adults (particularly the elderly) may be asymptomatic with cystitis.
B. Abdominal pain and suprapubic heaviness.
C. Pregnancy: uterine contractions.
D. Shortness of breath (SOB).
E. Anorexia.
F. Elderly:
1. Fever.
2. Mental status change.
3. Generalized deterioration.
4. Decompensation in another organ system.

SUBJECTIVE DATA

A. Review the onset, course, and duration of symptoms.
B. Are there any problems with voiding, such as frequency, urgency, and dysuria?
C. Review the client's history of fever and any treatment.
D. Are there any other symptoms, odor, or nausea?
E. Have the client point to the area of the backache. Is it unilateral or bilateral? What makes the backache better?
F. Rule out pregnancy; review first day of last menses.
G. Rule out sickle cell disease, diabetes, and MS.
H. Review the client's history of genitourinary (GU) tract problems, stones, UTIs, previous pyelonephritis, any previous testing, and any previous anomalies.
I. Review the strength and characteristics of the urinary stream, especially in older males. Ask if the client has ever been diagnosed with BPH.
J. Review the client's history for active herpes lesion. Does urine flow hurt when urine stream begins? Or is the pain noted when urine passes over the lesion?
K. Review drug allergies.
L. Review all medications including over-the-counter (OTC) and herbal products. Review medications for a recent history of an incomplete course of antibiotics and current use of anticholinergics.

PHYSICAL EXAMINATION

A. Clinical presentations and disease severity vary widely, from mild flank pain with low-grade or no fever to septic shock.
B. Check temperature, pulse, and blood pressure (BP); note orthostatic hypotension. Tachycardia may or may not be present, depending on associated fever, dehydration, and sepsis.
C. Inspect:
1. Note general appearance for respiratory distress and dehydration.
2. Inspect the male external genitalia for redness, edema, lesions, and discharge.
3. Inspect the female genitalia for discharge, lesions, and fissures; inspect cervix for cervicitis. ▶

D. Palpate:
1. Palpate the back; check CVA tenderness (usually unilateral over the involved kidney).
2. Palpate the abdomen for suprapubic tenderness, rebound masses, or pain.
3. Perform a pelvic examination to rule out other infections and pelvic inflammatory disease (PID; tenderness of the cervix, uterus, and adnexal should be absent).
E. Auscultate:
1. Lungs and heart.
F. Pregnancy:
1. Check fetal heart rate; fetal tachycardia may be present with fever.
2. Palpate for uterine tenderness and contractions.
3. Pelvic examination for cervical dilation, if indicated for increased risk of preterm labor.
G. Males:
1. Complete palpation of external genitalia, prostate, and rectal exam.

DIAGNOSTIC TESTS

A. The urine culture is the cardinal confirmatory diagnostic test. Urine culture and sensitivity should always be performed before initial empiric treatment with antibiotics.
B. Urinalysis:
1. Dipstick leukocyte esterase test (LET) screens for pyuria. Pyuria is present in almost all females with acute cystitis and pyelonephritis; its absence strongly suggests an alternative diagnosis.
a. Significant pyuria is greater than two to five leukocytes per high-power microscope field (HPF).
2. Nitrite production test (NPT) screens for bacteriuria.
3. Examination for hematuria (gross and microscopic) and proteinuria.
4. Urine may need to be obtained from straight in-and-out catheterization or suprapubic needle aspiration if the client is incontinent or has dementia.
C. Complete blood count (CBC) with differential or white blood cell (WBC), especially with systemic symptoms.
D. Blood culture, if indicated.
E. Arterial blood gases (ABGs), if indicated.
F. Consider sedimentation rate, especially with severe illness and in the elderly.
G. Culture for gonorrhea and chlamydia if symptoms are associated with sexually transmitted infection (STI).
H. Wet prep if symptoms are associated with STI.
I. Imaging studies are not routinely required for the diagnosis of acute pyelonephritis but can be helpful. Indications for initial and subsequent imaging (e.g., baseline risk factors and clinical worsening or lack or improvement after 24 to 48 hours of therapy) to assess for obstruction, abscess, or necrotizing infection:
1. CT scan to identify altered parenchymal perfusion, hemorrhage, nonrenal disease, inflammatory masses/abscessing, and obstruction. ***Contrast is contraindicated in clients with renal dysfunction.*** CT with contrast medium is considered the imaging modality of choice for nonpregnant individuals.
2. MRI to detect renal infections or masses or obstruction.
3. Renal ultrasonography with Doppler study.
4. Scintigraphy to detect focal renal abnormalities.
5. Voiding cystourethrogram.
6. Intravenous pyelography (IVP), if indicated.
7. CT and magnetic resonance (MR) urography used in the evaluation of hematuria.

DIFFERENTIAL DIAGNOSES

A. Pyelonephritis.
B. Appendicitis/acute abdomen.
C. Acute cholecystitis.
D. Pancreatitis.
E. Diverticulitis.
F. Pneumonia.
G. Prostatitis.
H. Epididymitis.
I. PID.
J. Nephrolithiasis.
K. Ureterolithiasis.
L. Renal vein thrombosis.

PLAN

A. General interventions:
1. Optimal therapy for acute uncomplicated nephritis depends on the severity of the illness at presentation.
2. Many severe infections (increased WBC, dehydration or vomiting, high fever) may need hospital admission for IV therapy. Risk factors include older adult, coexisting illness, pregnancy, and uncontrolled vomiting.
B. Client teaching: *See Client Teaching Guide for this chapter, "Urinary Tract Infection."*
1. Give instruction on early recognition of UTIs.
C. Dietary management:
1. Increase fluids; have the client drink at least one large glass of water every hour while awake (unless contraindicated).
2. Encourage the client to drink cranberry juice to help fight and prevent UTIs. If the taste is objectionable, they may mix cranberry juice 1:1 with another juice such as grape juice.
3. There are no dietary restrictions with pyelonephritis.
D. Pharmaceutical therapy:
1. Acetaminophen (Tylenol) for fever.
2. Urinary analgesic as needed to relieve dysuria. Dysuria is usually diminished fairly quickly after the start of antibiotics.
3. Antiemetics as needed; however, if the client is not able to tolerate oral fluids, they should be hospitalized.
4. Antibiotics: Empiric antibiotic selection should be guided by local antibiotics resistance patterns, allergies, and culture results. Clients with delayed response to therapy should also receive a longer course of antibiotics of 14 to 21 days.
a. Adults:
i. First-line therapy: ciprofloxacin (Cipro) 500 mg BID for 5 to 7 days, *or* ER Cipro XR 1,000 mg once a day for 5 to 7 days.
1) Recommended treatment in pregnancy of standard or high-dose extended-release ciprofloxacin is 7 days.
ii. First-line therapy: levofloxacin (Levaquin) 750 mg once daily for 5 to 7 days.
iii. Second-line therapy: trimethoprim-sulfamethoxazole (TMP-SMX) (Septra DS, Bactrim DS) 160 and 800 mg, respectively, one tablet BID for 7 to 10 days.
1) Recommended treatment in pregnancy of TMP-SMX is 14 days.
2) Due to the high rate of resistance of *E. coli*, the empiric use of TMP/SMX should be avoided in clients who require hospitalization. ▶

iv. Alternative therapy: amoxicillin-clavulanate (Augmentin) 875 mg orally BID for 14 days.

1) Recommended treatment in pregnancy is 10 to 14 days for oral beta-lactams.

Antibiotics that should not be utilized for pyelonephritis:

A. Nitrofurantoin (Macrodantin) and fosfomycin (Monurol) should not be used to treat pyelonephritis in adults. It is excreted in the urine but does not achieve therapeutic serum levels.
B. Fluoroquinolones are not used in pregnancy due to the risk of auditory and vestibular toxicity in the fetus.
C. Aminoglycosides are contraindicated in pregnancy because of the risk of permanent ototoxicity to the fetus.

FOLLOW-UP

A. Follow up with the client in 24 to 48 hours depending on the evaluation of the initial severity of symptoms.

1. Clients with persistent fever or clinical symptoms after 48 to 72 hours of appropriate antibiotic therapy should undergo initiation of another class of antibiotics and consider radiologic evaluation.

B. Follow-up urine cultures are not needed for clients with acute cystitis or pyelonephritis if symptoms resolved on antibiotics. However, repeat cultures are in order for clients with recurrent symptoms or any complicated course of illness.

C. Females with recurrence of pyelonephritis need further urologic investigation.

EMERGENT ISSUES/INSTRUCTIONS

A. Within 24 to 48 hours, if the client feels that they are not progressing well or are getting worse, evaluate the client emergently and consider hospital admission for urgent intervention including IV antibiotics.

CONSULTATION/REFERRAL

A. Consult or refer the client to a physician if they require IVP, cystoscopy, or renal biopsy.

B. Males with persistent bladder infections need a urologic consultation.

C. Pyelonephritis in males suggests structural problems and the need for hospitalization and further evaluation (IVP).

D. Consult an infectious disease specialist for clients with unusual or resistant pathogens.

E. For pregnancy, consultation with an obstetrician is required.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Pyelonephritis is the most common urinary tract complication in pregnancy.

2. Untreated asymptomatic bacteriuria (ASB) is a risk factor for acute cystitis and pyelonephritis in pregnancy.

3. Based on the higher risk of complications in pregnancy, pyelonephritis has traditionally been treated with hospitalization and IV antibiotics until the client is afebrile for 48 hours and symptoms improve.

4. Once the pregnant client is discharged from the hospital, oral antibiotics should continue for 10 to 14 days of treatment.

5. During pregnancy, a urine culture should be obtained 1 to 2 weeks after completion of therapy and monthly thereafter to monitor for recurrent infection.

6. Aminoglycosides should be avoided due to the potential risk of ototoxicity following prolonged fetal exposure.

7. Fluoroquinolones are contraindicated during pregnancy due to the risk of auditory and vestibular toxicity in the fetus.

B. Males:

1. Consider ordering renal function tests (blood urea nitrogen [BUN] and creatinine).

2. Males older than 50 years of age: Consider urologic consultation and IVP.

C. Geriatrics:

1. Geriatric syndrome assessments:

a. Delirium/behavioral disturbance: For fever, mental status changes, and generalized deterioration, evaluate for UTI and/or pyelonephritis. Bladder or kidney infections may be common in clients with long-term urinary catheters and can lead to septicemia if untreated or unrecognized. Septicemia increases with bedridden status. Clients may need hospitalization for IV antibiotics and hydration.

b. Weight loss/appetite suppression: Pyelonephritis is a risk factor for anorexia.

c. Constipation: Fecal impaction increases the risk for an obstruction.

2. Pharmacologic caution in geriatrics:

a. Fluoroquinolone use in the elderly has the potential to cause neuropsychiatric symptoms, including seizures to worsening dementia.

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RENAL CALCULI, OR KIDNEY STONES (NEPHROLITHIASIS)

DEFINITION

A. Renal calculi, or kidney stones, are caused by the formation of crystals in the urinary system from the kidneys to the bladder. Nephrolithiasis refers to renal stone disease; urolithiasis refers to the presence of stones in the urinary system. The majority of stones (80%) consist of calcium usually as calcium oxalate, but they can contain uric acid, struvite (magnesium, ammonium, and phosphate), oxalic acid, phosphate salts, or the amino acid cystine. Spontaneous passage of a stone is related to the stone size and location. Approximately one-half of symptomatic clients require intervention for stone removal. An untreated staghorn (branch-shaped) calculi with persistent renal obstruction can destroy renal tissue with potential for life-threatening sepsis.

INCIDENCE

A. Renal calculi are very common, with a higher incidence noted in males. At least 12% of males and 7% of females have at least one symptomatic stone by age 70. Initial cases typically occur between ages 30 and 40, and the prevalence increases with age. Idiopathic nephrolithiasis is common in males, whereas primary hyperparathyroidism is more common in females. An initial stone attack after the age of 50 is relatively uncommon. ▶

B. Most kidney stones pass spontaneously; however, 10% to 30% do not pass and can cause continuing pain, infection, or obstruction.
C. Stones caused by infection (struvite) are more common in females.
D. The incidence of stones in pregnancy is 1 in every 1,500 to 3,000 pregnancies.
E. The recurrence rate for calculi is 50% within 5 years.

PATHOGENESIS

A. The formation of uric acid stones requires continued and excessive oversaturation of urine with stone-forming constituents, uric acid, calcium, and oxalate. Dehydration, hyperuricosuria, and significantly acidic urine contribute to uric acid supersaturation and stone formation. Struvite stones form only when the urinary tract is infected with urea splitting organisms such as *Proteus* species.
B. Hydroureteronephrosis is the most significant renal alteration in pregnancy. Dilatation is greater on the right side than the left due to pressure caused by physiologic engorgement of the right ovarian vein and dextrorotation of the uterus.

PREDISPOSING FACTORS

A. Male.
B. Dehydration (poor intake and immobility).
C. Chronic obstruction with stasis of urine.
D. Hypercalcemia caused by hyperparathyroidism; renal tubular acidosis; multiple myeloma; or excessive intake of vitamin D, milk, and alkali.
E. Diet high in purines and abnormal purine metabolism (gout).
F. Pregnancy (1 per 1,500).
G. Chronic infections.
H. Foreign bodies.
I. Excessive oxalate absorption in inflammatory bowel disease, bowel resection, or ileostomy.
J. Previous stone formation.
K. Family history of nephrolithiasis.
L. Medications:
1. Vitamins A, C, and D.
2. Loop diuretics.
3. Acetazolamide.
4. Ammonium chloride.
5. Calcium-containing medications, including alkali and antacids.
6. Indinavir.
7. Sulfadiazine.
8. Atazanavir.
9. Guaifenesin.
10. Sulfa drugs.
11. Topiramate.
12. Acyclovir.
13. Triamterene.
M. Obesity.
N. Gastric bypass/bariatric surgical procedures.
O. Diabetes.
P. Anatomic abnormalities.

COMMON COMPLAINTS

A. Severe flank and groin pain.
B. Pelvic pain.
C. Blood in urine.
D. Asymptomatic (dependent on the size of the stone).

OTHER SIGNS AND SYMPTOMS

A. The symptoms of kidney stone are related to their location whether it is in the kidney, ureter, or urinary bladder.
1. Unilateral flank pain that radiates to the groin.
2. Sudden onset of colicky pain.
3. Hematuria.

The timing and appearance of hematuria are both important. Hematuria seen at the beginning of the urine stream may indicate bleeding in the urethra. Terminal hematuria, or blood in the end of the urine stream, denotes bladder neck or the prostate as the source. Lastly, blood throughout the entire urination suggests a lesion.

4. Nausea and vomiting.
5. Restlessness.
6. Symptoms common with cystitis or inflammatory lesions of the lower tract are usually absent: frequency, dysuria, urgency, and suprapubic pain.

SUBJECTIVE DATA

A. Review the onset, duration, and course of symptoms.
B. Review other signs and symptoms of a urinary tract infection (UTI) or pyelonephritis: frequency, dysuria, and fever.
C. Have the client describe pain (colicky); note intensity (use a 0- to 10-point scale, 10 being the worst pain) and the characteristics of pain (constant, intermittent).
D. Has the client ever had a stone before? How was it treated? What tests were performed? Has the client ever seen a urologist?
E. Review dietary intake of high animal protein in the diet, milk, and other calcium-containing products for excessive intake.
F. Review the client's medication history including excessive vitamin C or D supplements, antacids that contain calcium, and other medications noted in the predisposing factors.
G. Ask the client to describe any hematuria or blood clots passed.
H. Ask about recent trauma to the back or abdomen.
I. Is there a family history of stone formation?
J. Is the client pregnant?

PHYSICAL EXAMINATION

A. Check temperature, blood pressure (BP), and pulse (may have tachycardia).
B. Inspect:
1. Inspect general appearance for discomfort before and during exam. Clients with renal colic are extremely restless and exhibit active movement on presentation.
2. During the examination, evaluate voluntary guarding of the abdominal musculature.
3. Inspect external genitalia (man or woman) for lesions, discharge, inflammation, and ulcerations.
4. Assess for peripheral edema.
C. Auscultate:
1. Abdomen, noting bruits if present.
2. Bowel sounds in all four quadrants.
D. Palpate:
1. "Milk" the urethra for discharge.
2. Palpate the abdomen for masses and tenderness, organomegaly, and suprapubic tenderness.
3. Palpate the groin; check lymph nodes.
4. Palpate the back and abdomen.

5. Check for the presence of costovertebral angle (CVA) tenderness.

E. Pelvic or bimanual exam:

1. Perform pelvic or bimanual exam, if indicated, to rule out pelvic inflammatory disease (PID).

DIAGNOSTIC TESTS

A. The diagnosis of nephrolithiasis can be made on the basis of clinical symptoms alone, but diagnostic testing is needed to confirm.

B. Laboratory tests:

1. Serum blood urea nitrogen.
2. Creatinine.
3. Calcium.
4. Uric acid.
5. Serum electrolytes (consider fasting serum calcium and phosphorus and parathyroid hormone).
6. Pregnancy test (if indicated) to rule out an ectopic pregnancy.

C. Stone for analysis.

D. Urinalysis:

1. Urine dipstick for a gross screen.
2. pH determination (pH >7.5 is compatible with infection lithiasis, and a pH of <5.5 favors uric acid lithiasis).
3. Red cell casts strongly suggest glomerulonephritis.
4. Evaluate urine sediment for crystalluria.

E. Urine culture, if indicated.

F. 24-hour urine for creatinine, calcium, uric acid, oxalate, pH, and sodium measurement:

1. The client should be on their usual diet before taking 24-hour specimen.
2. Collection should be 1 to 2 months after any interventions, including shock-wave lithotripsy, ureteroscopy, or percutaneous stone removal.

G. CT of the abdomen and pelvis without contrast is the imaging standard to assess the urinary tract in acute renal colic.

H. Renal ultrasound is the procedure of choice for pregnancy.

I. X-ray of the kidney, ureter, and bladder (KUB) is often ordered with the pelvic CT or ultrasound.

J. Intravenous pyelography (IVP) is used as an adjunct or follow-up exam. IVP is rarely used in the initial diagnosis of nephrolithiasis. IVP reliably detects hydronephrosis but is less sensitive and specific than a CT for the detection of stones.

K. Nuclear renal scan.

DIFFERENTIAL DIAGNOSES

A. Kidney stone(s): associated with colicky flank pain radiating to the groin. Significant flank pain of renal colic is usually secondary to renal calculi but may occasionally be associated with passage of clots.

B. UTI: Passage of large, bulky blood clots implicates the bladder as the source, whereas long, shoestring-shaped specks or thin, stringy clots suggest an upper urinary tract or ureteral origin.

C. Pyelonephritis: associated with dull flank pain with fever and chills. In evaluating urine sediment, the presence of white cells and bacteria favors a diagnosis of pyelonephritis or interstitial nephritis.

D. Acute abdomen/appendicitis.

E. Cholecystitis.

F. PID.

G. Inflammatory bowel disease.

H. Urinary tract obstruction.

I. Constipation.

J. Gynecologic (ectopic pregnancy, ovarian cyst torsion, or rupture).

K. Lobar pneumonia.

L. Rib fractures.

M. Radicular pain (L1 herpes zoster, sciatica).

N. Narcotic seeker with possible self-inflicted hematuria.

PLAN

A. General interventions:

1. Increase fluids to allow passage of stone. Strain all urine to recover stone for analysis.
2. Reduce possibility of recurrence with dietary modifications.
3. Clients are usually referred for imaging after evaluation of creatinine.
4. Clients can be managed on an outpatient basis with close follow-up if stones are small (<6 mm).

B. Client teaching:

1. Force fluids to maintain a daily output of 2 to 3 L of urine. Fluid intake that increases urinary production of at least 2 L of urine per day increases the flow rate and lowers the urine solute concentration.
2. Dietary consultation may be needed secondary to stone analysis.
   a. Calcium stone: Reduce sodium. A 2,000-mg low sodium diet may be advised.
   b. Oxalate stone: Limit oxalate. Oxalate is found in many fruits, vegetable, nuts, seeds, grains, legumes, and chocolate.
   c. Uric acid stone:
      i. Reduce high-purine foods such as red meat, organ meats, and shellfish.
      ii. Limit alcohol and sugar-sweetened foods and drinks.
      iii. Avoid crash diets.

C. Pharmaceutical therapy:

1. Pain medication (narcotic and nonnarcotic) is a priority.
   a. Nonsteroidal anti-inflammatory drugs (NSAIDs) should be discontinued 3 days prior to shock-wave lithotripsy to reduce the risk of bleeding.
   b. Opioids are prescribed for acute pain.
2. Antibiotics should be given for infection.
3. Antiemetics if needed.
4. Both tamsulosin and nifedipine have shown to increase the likelihood of stone passage.
5. Other medical/pharmaceutical management depends on the etiology of the stone:
   a. Clients with calcium stones that cannot be solely managed with dietary modification can be treated with a thiazide diuretic and a low-sodium diet for high urine calcium, potassium citrate for hypocitraturia.
   b. Clients with uric acid stones can be treated with potassium citrate or potassium bicarbonate to alkalinize the urine.
   c. Occasionally allopurinol is used for calcium or uric acid stones.
   d. Struvite stones typically require complete stone removal with percutaneous nephrolithotomy and aggressive prevention and treatment of future UTIs.
6. In clients with ureteral stones >5 and <10 mm in diameter, treatment with the alpha-blocker tamsulosin (0.4 mg once daily) for up to 4 weeks can help to facilitate spontaneous stone passage. Alpha-blockers have higher stone expulsion rate than calcium channel blockers. ▶

D. Surgical options are dependent on stone size and location:

1. Percutaneous nephrolithotomy is the first treatment option for most clients and is considered the treatment of choice for clients with staghorn calculi.

2. Extracorporeal shock-wave lithotripsy is the least invasive of the surgical methods.

3. Percutaneous nephrostomy (should be the last procedure for most clients).

4. Open nephrostomy may also be used.

FOLLOW-UP

A. Reevaluate the client in 24 hours by phone or in the clinic.

B. Evaluate sooner if pain increases due to the potential to progress to complete obstruction.

C. Recurrent stone formation is a manifestation of a systemic disease; evaluate for management of metabolic abnormality.

CONSULTATION/REFERRAL

A. Clients with severe pain, nausea, and vomiting need hospitalization for intravenous (IV) hydration and pain control. Consult with provider.

B. Clients with severe symptoms and persistent obstruction beyond 3 to 4 days should be referred for urologic evaluation.

C. Refer for surgical interventions: Lithotripsy, urethroscope interventions, extracorporeal shock-wave lithotripsy, and percutaneous ultrasonic lithotripsy may be indicated. Treatment varies based on the location and size of the stone. Laparoscopy may be indicated for the removal of a large or **severely impacted ureteral calculi.**

D. Refer to outpatient urology if stone is >10 mm.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Urolithiasis is the most common cause of nonobstetrical abdominal pain that requires hospitalization in pregnancy.

2. Between 80% and 90% are diagnosed in the first trimester.

3. Renal ultrasound is the first-line screening test for pregnant clients. A transvaginal ultrasound may also be performed.

4. Low-dose CT is reserved for complex cases in the second and third trimesters.

5. Conservative treatment is used: bedrest, hydration, and analgesia.

6. Invasive measures include stent placement, ureteroscopy, and percutaneous nephrostomy.

7. Upon presentation, rule out:

a. Ectopic pregnancy.
b. Abruptio placenta.
c. Preterm labor.

B. Geriatrics:

1. Nineteen percent of males and 9% of females have a kidney stone by age 70 years.

2. In the elderly, chronic disease such as diabetes, hypertension, or obesity can increase uric acid levels, placing them at higher risk.

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SEXUAL DYSFUNCTION, MALE: ERECTILE DYSFUNCTION

DEFINITION

A. Erectile dysfunction (ED), also known as impotence, is the persistent inability to achieve or maintain penile erection sufficient for satisfactory sexual performance. ED occurs with reduced blood flow to the penis or nerve damage, as well as psychological triggers. Low self-esteem, performance anxiety, depression, stress, and effects to quality of life occur secondary to ED. ED is noted to be a precursor to symptomatic coronary artery disease (CAD).

B. Age-associated changes in sexual function in males include delay in erection, diminished intensity and duration of orgasm, and decreased force of seminal emission. ED lasting 3 months or longer should have further evaluation and consideration of treatment.

C. Multiple male sexual dysfunction questionnaires are available for order through the Pfizer Patient-Reported Outcomes website located at www.pfizerpcoa.com/disease-area/sexual-health, sponsored by Pfizer, Inc. The questionnaires are available in several translations. Questionnaires include the following:

1. Erectile Dysfunction Inventory of Treatment Satisfaction (EDITS) used in the evaluation of satisfaction with medical treatment modalities for ED.

2. Erectile Hardness Scale (EHS) was developed to assess erection hardness in males with ED.

3. International Index of Erectile Function (IIEF) is available in two versions. Version 1 is applicable to heterosexual males. Version 2 is an updated version so that it is applicable to heterosexual and homosexual males. The IIEF assesses five dimensions relevant to sexual function:

a. Erectile function (six items).
b. Orgasmic function (two items).
c. Sexual desire (two items).
d. Intercourse satisfaction (three items).
e. Overall satisfaction (two items).

4. Index of Premature Ejaculation (IPE) assesses control over ejaculation, sexual satisfaction, and distress.

5. Premature Ejaculation Diagnostic Tool (PEDT) was developed to screen for premature ejaculation (PE), including control, frequency, minimal sexual stimulation, distress, and interpersonal difficulty.

6. Quality of Erection Questionnaire (QEQ) evaluates satisfaction with the quality of erections, including hardness, onset, and duration.

7. Self-Esteem and Relationship (SEAR) questionnaire assesses confidence.
8. Sexual Health Inventory for Men (SHIM) is a five-item abridged version of the 15-item IIEF.
9. Sexual Quality of Life-Men (SQOL-M) was developed to assess sexual confidence, emotional well-being, and relationship issues. This questionnaire has been validated for males with ED and premature ejaculation.

INCIDENCE

A. ED can occur at any age; however, it is more common in those older than 60 years.
B. It is estimated that 18 to 30 million American males have ED.
C. By 2025, it is estimated that 322 million males worldwide will have ED.
D. Males with ED have a 65% to 85% increased risk of subsequent CAD.
E. Reduced libido is estimated as affecting 5% to 15% of males.

PATHOGENESIS

A. Normal pathology: The dorsal nerve of the penis provides innervation, the dorsal somatic nerve provides sensation, and the autonomic nervous system, via the cavernosal nerves, regulates blood flow to the penis, allowing for erection to occur. The ability to maintain an erection relies on the dorsal nerve, the peripheral nerves, penile vasculature, and biochemical releases within the corpora.
B. Multiple factors may contribute to ED:
1. Vascular (most common):
a. This system is responsible for delivering and trapping the blood in the corporal sinusoids. Usually, the blood flowing in is not the problem; rather, the ED is the result of venous leaking from the corporal sinusoids.
b. Arterial insufficiency contributes to decreased blood flow to the cavernosal sinuses. The cavernosal dysfunction causes difficulty with retaining blood in the penis and sustaining an erection.
i. Chronic diseases such as cardiovascular disease, hypertension (HTN), dyslipidemia, diabetes, and obesity affect blood flow.
ii. Certain medications can contribute to this physiologic effect.
iii. Smoking is also a contributing factor, especially in the presence of existing cardiovascular disease due to the vasoconstrictive effects it causes.
2. Psychological:
a. Direct inhibition of the spinal erection center and/or excessive sympathetic nervous system biochemical release occurs, which increases the smooth muscle tone of the penis, preventing erection due to:
i. Age-related decline.
ii. Lack of sexual response.
iii. Personal intimacy-related issues.
iv. Partner-specific intimacy issues.
v. Performance anxiety.
vi. Depression or life stress related.
3. Neurologic:
a. Any disease affecting the brain, spinal cord, and cavernous and penile nerves can impair the ability to achieve erection, such as spinal cord injury, stroke, or diabetes.
b. Surgery in the pelvic region, including prostatectomy, perineal resection, and sphincterotomy, may also be causes.
4. Endocrine:
a. Decreased testosterone level.
b. Increased prolactin level.
c. Hyperthyroidism.
d. Hypothyroidism.

PREDISPOSING FACTORS

A. Cardiovascular disease.
B. Diabetes (neurologic and vascular problems).
C. HTN.
D. Hyperlipidemia.
E. Advanced age (over 60 years).
F. Peripheral neuropathy.
G. Obesity.
H. Neurologic disorders:
1. Spinal cord injuries.
2. Brain injuries.
3. Multiple sclerosis (MS).
4. Parkinson disease.
I. Alcohol abuse.
J. Drug abuse:
1. Heroin.
2. Cocaine.
3. Marijuana.
K. Side effect of medication (e.g., serotonin reuptake inhibitors, antihypertensives, antihistamines, diuretics, nonsteroidal anti-inflammatories, muscle relaxants).
L. Surgical/radiation therapy for cancers of the pelvis.
M. Hypogonadism (hypoandrogenism/hypoestrogenism).
N. Psychological and psychiatric disorders.
O. Peyronie disease (deformity of the penis).
P. Obstructive sleep apnea (OSA).
Q. Physical inactivity.

COMMON COMPLAINTS

A. Inability to achieve an erection.
B. Erection is not firm enough for penetration.
C. Inability to maintain an erection.
D. Inability to control the timing of ejaculation.
E. Lack of interest or desire (most common).
F. Pain with intercourse.

OTHER SIGNS AND SYMPTOMS

A. Diminished self-esteem.
B. Depression.
C. Anxiety.
D. Reduced libido.
E. Relationship difficulties.
F. Premature ejaculation.

SUBJECTIVE DATA

History taking for ED includes sexual, medical, surgical, emotional, and medication evaluations.

A. Sexual history:
1. Did the onset of ED coincide with a specific event?
2. How long has the client had trouble attaining or maintaining an erection?
3. Are they able to obtain an erection in order to penetrate? On a scale of 0 to 10, how hard is the erection? ▶

4. Is the ED getting worse?
5. Do they come close to achieving orgasm and ejaculating?
6. How long is the client able to have intercourse prior to ejaculation?
7. Is there pain or discomfort with ejaculation?
8. Does the client have nocturnal or morning erections?
9. How frequently does the client have sexual activity?
   a. Is the activity planned, or does it occur spontaneously?
   b. How much foreplay occurs?
   c. Do the client and partner agree on the frequency of intercourse?
   d. Is the client's partner satisfied?
10. Has the client tried any treatment(s) for ED?
   a. What treatments have been tried?
   b. Inquire about their desire to try any particular therapy. Are they opposed to try any particular therapy?

B. Medical history:
1. Does the client have HTN? When was HTN? What is their usual blood pressure?
2. Does the client have diabetes?
   a. Are they insulin-dependent?
   b. Do they have any peripheral neuropathy?
3. Does the client have heart disease? When was their heart disease diagnosed?
4. Has the client ever had cancer, including any surgery, chemotherapy, and radiation?
5. Does the client have dyslipidemia? What were the results of their last laboratory tests?
6. Does the client have a sleep disorder?
7. Does the client smoke? How much, including the number of pack-years?
8. Does the client drink? How much, how often?
9. Does the client have penile curvature (Peyronie disease)?
10. Does the client have any neurologic disorders?

C. Surgical history:
1. Has the client had any prior surgeries, including pelvic or prostate, or experienced trauma?
2. Has the client had any invasive cardiac procedures or surgery?

D. Emotional history:
1. Has the client ever had any traumatic sexual experience?
2. Has the client had a loss of libido?
3. Does the client have a history of depression or mood disorders?
4. Is the client experiencing any problems related to work and/or family?
5. Does the client have any intrapartner problems such as separation or divorce?

E. Medication history: Ask the client to list all medications currently being taken, particularly substances not prescribed, including herbal products and illicit drugs. Multiple drug classifications include medications that contribute to ED. Review medications from these drug classes:
1. Nitrates.
2. Antihypertensives (particularly alpha-blockers).
3. Antiulcer medications.
4. Lipid-lowering medications.
5. 5-Alpha reductase inhibitors (e.g., finasteride or dutasteride).
6. Antidepressants.
7. Herbal products.
8. Illicit drugs.
9. Caffeine.

PHYSICAL EXAMINATION

A. Check blood pressure, height, and weight. Calculate body mass index (BMI).

B. Inspect:
1. Inspect general appearance, noting dyspnea and weakness.
2. Inspect skin for jaundice, pallor, and diaphoresis.
3. Inspect legs for edema, cyanosis, and venous stasis.
4. Perform a funduscopic examination.
5. Evaluate visual field defects (present in hypogonadal males with pituitary tumors).
6. Inspect for penile plaques (indicates Peyronie disease).
7. Inspect the testicles:
   a. Check for presence of atrophy.
   b. Assess asymmetry.
   c. Evaluate the cremasteric reflex by stroking the inner thighs and observe ipsilateral contraction of the scrotum.

C. Palpate:
1. Palpate abdomen for masses, tenderness, bounding pulses, and organomegaly.
2. Palpate peripheral pulses in legs.
3. Palpate femoral pulses.
4. Examine breasts to detect gynecomastia.
5. Palpate the testicles for masses.
6. Perform a rectal exam to evaluate the prostate.

D. Auscultate:
1. Auscultate carotid arteries for bruits.
2. Auscultate abdomen for bruits and bowel sounds.
3. Auscultate heart for murmurs, rubs, clicks, irregularities, or extra sounds.
4. Auscultate for femoral bruits (possible pelvic blood occlusion).
5. Auscultate all lung fields.

E. Observe ipsilateral contraction of the scrotum.

F. Mental status:
1. Assess for depression.

G. Neurologic exam:
1. Perform a neurologic examination if neurologic etiology is suspected.

DIAGNOSTIC TESTS

A. Hormonal testing and treatment of ED should be individualized based on clinical presentation, including libido, premature ejaculation, fatigue, testicular atrophy, and muscle atrophy that suggests a hormonal abnormality.

B. Laboratory tests:
1. Lipid profile.
2. Triglycerides.
3. Glucose or hemoglobin (Hgb) A1C.
4. Prostate-specific antigen (PSA) testing (if on testosterone replacement).
5. Hematocrit (if on testosterone replacement).
6. Testosterone (performed in the morning) and other hormone levels, such as luteinizing hormone, or prolactin level, if high index of suspicion for prolactinoma including visual disturbance or headache.
7. Urinalysis for protein and glucose.

C. Duplex ultrasound of the cavernous arteries and other vascular testing as indicated. Penile perfusion ultrasound may be done to evaluate arterial perfusion of the penis (not commonly used).

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D. Nocturnal penile tumescence (NPT) as indicated. This determines the quality and number of nighttime erections. This test is not routinely used and is typically used in younger, more complicated clients.

E. Other tests as indicated for abnormal findings on physical examination.

DIFFERENTIAL DIAGNOSES

A. ED.

B. Testosterone deficiency.

C. Decreased libido.

D. Anorgasmia.

E. Peyronie disease.

PLAN

A. General interventions:

1. Lifestyle modification to include weight loss, increased physical activity, limited alcohol consumption, and smoking cessation.

2. Control of comorbidities, including cardiovascular (CV) disease, diabetes, and HTN, is desired.

B. Client teaching:

1. Educate client about modifying controllable risk factors such as keeping diabetes and HTN under control, diet, exercise, and stopping smoking.

2. Failure to respond to phosphodiesterase-5 (PDE5) inhibitor treatment may be from improper instructions or an inadequate dosage of medication. Table 15.6 details dosing and side effects.

3. The initial administration of an alprostadil intraurethral suppository should be done in the office in order to demonstrate correct administration.

4. Stepwise therapy for ED includes pharmaceuticals and surgery.

C. Stepwise therapy for ED includes the following treatment modalities:

1. First-line therapy: Pharmaceutical treatment with PDE5 inhibitors is the first-line therapy for treatment of ED. PDE5 inhibitors are not initiators of erection and require sexual stimulation for an erection to occur. The evidence shows that the PDE5 inhibitors improve erections and successful intercourse with an approximately 80% success rate. The use of PDE5 inhibitors has been extensively studied; however, they are not without side effects (see Table 15.6 for dosing and side effects).

a. **Contraindications to PDE5 inhibitors include high-risk conditions and the concomitant use of nitrites. If the client develops angina while using a PDE5 inhibitor, other antianginal agents should be used instead of nitroglycerin.**

b. High-risk clients/conditions are defined as:

i. Unstable or refractory angina.
ii. Refractory angina.
iii. Uncontrolled HTN.
iv. Congestive heart failure (CHF; New York Heart Association classes III and IV).
v. Myocardial infarction (MI) or a CV accident within the previous 2 weeks.
vi. High-risk arrhythmias.
vii. Hypertrophic obstructive and other cardiomyopathies.

c. Before proceeding to other ED therapies, clients reporting failure of PDE5 inhibitors should be evaluated to determine whether the medication trial was adequate:

i. Evaluate food/drug interactions.
ii. Timing and frequency of dosing.
iii. Lack of adequate sexual stimulation.
iv. Heavy alcohol use.
v. Relationship issues.
vi. Using a licensed PDE5 inhibitor medication.

d. After evaluation, reeducation, and counseling on the medications, and partner–partner expectations, a different PDE5 inhibitor may be needed, or titrate the client's current dosing for a change if needed.

e. Discuss other options for ED if the client has a contraindication to, or an unsuccessful trial of, PDE5 inhibitors.

f. Testosterone therapy is not indicated in the treatment of ED if the client has a normal serum testosterone level.

2. Second-line therapy: Pharmaceutical therapy with intracavernous injection is the second-line therapy for treatment of ED. Penile injection therapy involves injection of alprostadil, a vasoactive drug, into the corpora cavernosa of the penis to expand the blood vessels and increase the blood flow to produce an erection. The most common side effects of alprostadil are burning and a prolonged erection lasting more than 4 hours. Prolonged erections require medical intervention to reverse the erection.

3. Third-line therapy: A penile implant is the third-line therapy for treatment of ED. Penile prostheses (implants) are surgically implanted, semirigid rods or a hydraulic device to ensure a rigid erection. The prosthesis does not usually affect urination, sex drive, orgasm, or ejaculation. Pain and/or reduced sensation, infection, or mechanical failure may occur from the prosthesis.

4. Vacuum erection devices are external cylinders used to pump the penis into the cylinder and produce an erection by drawing blood into the penis. An occluding band is then placed at the base of the penis in order to prevent the blood from leaving with subsequent loss of the erection. **Only vacuum constrictor devices containing a vacuum limiter should be used. The occluding band to maintain the erection should be limited to 30 minutes.**

5. Penile arterial revascularization is indicated for young males (younger than 45 years) with no known risk factors for atherosclerosis. The goal of the surgery is to correct injury by rerouting the blood vessel around a blockage or injured blood vessel. Those with insulin-dependent diabetes or widespread atherosclerosis are not candidates for this surgery.

6. Venous ligation surgery is rarely used. Those with insulin-dependent diabetes or widespread atherosclerosis are also not candidates for venous surgery.

FOLLOW-UP

A. Follow up in 1 to 3 weeks after treatments are initiated to monitor client satisfaction and quality of erections.

B. At the time of prescription renewal, clients prescribed PDE5 inhibitors should have a review of the effectiveness, side effects, and any significant change in health status, including a review of all medications.

TABLE 15.6 DOSING AND SIDE EFFECTS OF ORAL PDE5 INHIBITORS

Common Side Effects	Sildenafil Citrate (Viagra)	Vardenafil HCl (Levitra)	Tadalafil (Cialis)	Stendra (Avanafil)
Doses	25, 50, and 100 mg doses	5, 10, and 20 mg doses	2.5 and 5 mg: available for continuous daily use 10 and 20 mg doses	50, 100, and 200 mg doses
Instructions	Recommended starting dose is 50 mg. Take on an empty stomach. Maximum dosing is once a day. Titrate according to client response/side effects. It is effective 30–60 minutes from administration. A heavy fatty meal may reduce or prolong absorption.	Recommended starting dose is 10 mg (5 mg initial dose for the elderly). Take on an empty stomach. Titrate according to client response/side effects. It is effective 60 minutes from administration. A fatty meal reduces its effect.	Recommended starting dose is 10 mg. Titrate according to client response. Maximum dosing is once a day. It is effective from 30 to 60 minutes from administration. Peak efficacy occurs after 2 hours. Efficacy is maintained for up to 36 hours. It is not affected by food. It has been approved for continuous, daily use in 2.5 and 5 mg doses. It is also prescribed for BPH.	Recommended starting dose is 100 mg. Titrate according to client response/side effects. It may be taken with or without food. It is effective 15 to 30 minutes from administration. Maximum dosing is once a day.
Common Side Effects	**Sildenafil Citrate (Viagra)**	**Vardenafil HCl (Levitra)**	**Tadalafil (Cialis)**	**Stendra (Avanafil)**
Headache	X	X	X	X
Flushing	X	X	X	X
Nasal congestion/rhinitis	X	X	X	X
Dyspepsia	X	X	X	
Priapism	X	Rare	Rare	X
Myalgia			X	
Sinusitis		X		X
Backache			X	X
Limb pain			X	
Prolonged erection	X			X
Tachycardia		X		
Visual disturbance	Blue-green color tinge to vision, light sensitivity and blurred vision (lasts 2–3 hours).			
Hypotension with alpha-blockers	Should be stable on alpha-blocker prior to initiating a PDE5 inhibitor. Use lowest recommended PDE5 inhibitor dose.	Should be stable on alpha-blocker prior to initiating a PDE5 inhibitor. Use lowest recommended PDE5 inhibitor dose.	Should be stable on alpha-blocker prior to initiating a PDE5 inhibitor. Use lowest recommended PDE5 inhibitor dose.	Should be stable on alpha-blocker prior to initiating a PDE5 inhibitor. Use lowest recommended PDE5 inhibitor dose.
Sudden vision loss	Discontinue if vision loss occurs.	Discontinue if vision loss occurs.	Discontinue if vision loss occurs.	Discontinue if vision loss occurs.
Sudden hearing loss	Discontinue if hearing loss occurs.	Discontinue if hearing loss occurs.	Discontinue if hearing loss occurs.	Discontinue if hearing loss occurs.

(*continued*)

TABLE 15.6 DOSING AND SIDE EFFECTS OF ORAL PDE-5 INHIBITORS (*CONTINUED*)

Common Side Effects	Sildenafil Citrate (Viagra)	Vardenafil HCl (Levitra)	Tadalafil (Cialis)	Stendra (Avanafil)
Use with nitrates (includes nitroglycerin, isosorbide dinitrate, amyl nitrate, and sodium nitroprusside).	Contraindicated due to hypotension	Contraindicated due to hypotension	Contraindicated due to hypotension	Contraindicated due to hypotension
		Not to be used in clients with congenital QT prolongation		

BPH, benign prostatic hyperplasia; HCl, hydrochloride; PDE5, phosphodiesterase-5.

EMERGENT ISSUES/INSTRUCTIONS

A. A prolonged erection (priapism) lasting more than 4 hours is a medical emergency often requiring immediate urologic attention.

CONSULTATION/REFERRAL

A. Clients whose risk is undetermined for PDE5 inhibitors should undergo further evaluation by a cardiologist before receiving therapies for sexual dysfunction.

B. Surgical consultation: Clients with penile deformities may require surgical correction.

C. Urologist consultation.

D. Endocrinology consultation for complex endocrine disorders.

E. Psychosexual counseling may be considered for the client and/or couple.

F. Psychotherapy is recommended when ED is related to anxiety and/or depression.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. A mild prolongation of the QT interval has been observed with vardenafil. The product labeling for vardenafil recommends that caution be used in clients with known history of QT prolongation or in clients who are on current medications that prolong the QT interval.

2. Testosterone therapy is not indicated in the treatment of ED if the client has a normal serum testosterone level.

3. Clients who present with sleep disorders should also be questioned about the presence of ED.

4. In clients with diabetes, PDE5 inhibitors may fail.

5. PDE5 inhibitors are contraindicated in clients with high CV risk and for clients on nitrates.

6. Clients with ED are associated with a higher incidence of CV events.

B. Geriatrics:

1. After age 70, ED prevalence is nearly 70% and is strongly related to poor health.

a. Dose medications on the lower end for elderly clients:

i. Sildenafil: Dose at 25 mg for age greater than 65 years. Renal and hepatic dosing required. Caution in those with CHF, or MI within the past 6 months.

ii. Vardenafil: Dose at 5 mg for age greater than 65 years. Renal and hepatic dosing required.

iii. Tadalafil: Renal and hepatic dosing required.

iv. Avanafil: Renal and hepatic dosing required. Not to be used if cerebrovascular accident or MI within the past 6 months.

b. Atherosclerosis in feeder vessels to the penis and vessel damage from diabetes mellitus (DM) are commonly associated with the development of ED.

c. Polypharmacy is associated with ED as many medications interfere with libido or are linked with ED, including thiazide diuretics, clonidine, ketoconazole, and cimetidine. Others are linked with loss of libido (clonidine selective serotonin reuptake inhibitors [SSRIs], opioids, antiandrogens, and 5-alpha reductase inhibitors).

d. Geriatric syndrome assessment: Falls. Discuss the risk of dizziness or light-headedness when standing up because of orthostatic hypotension for males receiving treatment for ED. Older clients taking PDE5 inhibitors have an increased risk for falls related to orthostatic hypotension, especially with concomitant use of alpha-blockers.

e. Encourage client and caregiver or spouse to discuss with provider side effects they may be experiencing from these medications to ensure client safety.

RESOURCES

American Association of Sex Educators, Counselors, and Therapists

35 E Wacker Drive
Suite 850
Chicago IL 60601
(202) 449–1099
Fax (202) 216-9646
info@aasect.org
www.aasect.org

American Society for Reproductive Medicine
1209 Montgomery Highway
Birmingham, AL 35216
205-978-5000
https://connect.asrm.org/home?ssopc=1

American Urological Association
1000 Corporate Boulevard
Linthicum, MD 21090
866-746-4282
aua@AUAnet.org
https://www.auanet.org/#

International Society for Sexual Medicine
North American Office
14305 Southcross Dr.
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SEXUAL DYSFUNCTION, MALE: PREMATURE EJACULATION

DEFINITION

A. Premature ejaculation (PE) is defined as the inability to control or delay ejaculation, causing personal distress in males. Two subtypes exist, based on intravaginal ejaculation latency time (IELT):

1. Lifelong PE is an IELT of less than 1 minute. Onset is typically from the first sexual encounter. Males have no ability to control the ejaculation.

2. Acquired PE is an IELT of less than 3 minutes. Onset is at any point in sexual history. The ability to delay ejaculation is decreased or absent.

3. A third subtype, ejaculation before penetration, is ejaculation that occurs before vaginal penetration. This is considered the most severe form of PE.

B. Two subtypes of PE exist based on personal experience; neither is a true sexual dysfunction:

1. Variable PE is irregular and not the result of psychogenic cause. Onset is at any point in sexual history. The ability to delay ejaculation is decreased or absent.

2. Subjective PE is self-reported rapid ejaculation despite normal ejaculation time. Onset is at any point in sexual history. The ability to delay ejaculation is decreased or absent.

INCIDENCE

A. PE is reported in approximately 30% to 70% of males. It is the most common type of sexual dysfunction in those younger than 40 years of age. Erectile dysfunction (ED) commonly coexists.

B. PE most commonly occurs between the ages of 18 and 30 years.

C. PE can coexist with secondary impotence in those over 45 years of age.

PATHOGENESIS

A. The exact cause is unknown.

B. Organic: chronic prostatitis, hyperthyroidism, genetic disorders.

C. Psychogenic: relationship problems, psychological preoccupation with performance.

D. Iatrogenic: nerve damage from surgery or trauma.

PREDISPOSING FACTORS

A. Depression.
B. Consumption of alcohol.
C. Obesity.
D. High blood pressure (BP).
E. High cholesterol.
F. Diabetes.
G. Smoking.
H. Depression and anxiety medication use.

COMMON COMPLAINTS

A. Males:
1. Inability to achieve or sustain erection.
2. Absent or delayed ejaculation.
3. Inability to control the timing of ejaculation.

B. Both sexes:
1. Lack of interest or desire (most common).
2. Inability to become aroused.
3. Pain with intercourse.

OTHER SIGNS AND SYMPTOMS

A. Diminished self-esteem.
B. Depression.
C. Anxiety.
D. ED.
E. Reduced libido.
F. Relationship difficulties.

SUBJECTIVE DATA

A. Include full medical history, sexual history, and psychological, social, and drug history using open-ended questions.

B. Ask:
1. Personal and partner(s)' sexual history.
2. Perceived ejaculatory control.
3. Estimated IELT.
4. Previous interventions used to correct the issue.
5. Impact on personal life and relationships.
6. If the issue has been lifelong or acquired.
7. If a loss of erection occurs before ejaculation, to distinguish ED from PE.

C. Screening tools:
1. Premature Ejaculation Diagnostic Tool available at www.pfizerpcoa.com/sites/default/files/ped_us_enreview_only_3_1_1.pdf. The PEDT is a five-item screen for PE in males. ▶

2. The Index of PE available at www.pfizerpcoa.com/index-premature-ejaculation-ipe. The IPE is a 10-item questionnaire to assess changes in control over ejaculation, sexual satisfaction, and distress in males with PE. The questionnaire is available in several translations. Charges may apply for commercial use.

PHYSICAL EXAMINATION

A. Vital signs: Check BP, pulse, and respiration.

B. Inspect:

1. Thyroid for nodules.

2. Abdomen for surgical scars.

3. Neurologic examination for deficiencies.

4. Lower extremities for hair distribution pattern, lesions, or trauma.

5. Genitalia for abnormalities.

6. Any additional examination should be based on history and previous examination findings.

C. Palpate:

1. Thyroid for nodules.

2. Abdomen for masses or tenderness.

3. Lower extremities for pain and pulses.

4. Neurologic examination of lower extremities.

5. Genitalia for lesions, masses, or pain.

DIAGNOSTIC TESTS

A. Should be based on individual history and physical findings.

DIFFERENTIAL DIAGNOSES

A. ED.

B. Testosterone deficiency.

PLAN

A. General interventions:

1. The treatment plan should include the client and sexual partner.

2. The client and partner should be educated about the possible interventions outlined as follows:

a. Both should be educated in treatment options.

b. Client and partner satisfaction is the desired outcome.

B. Client teaching:

1. Consider the impact of the severity of the issue on the client, any treatable causes, and client wishes.

2. Effectiveness of psychotherapy may diminish over time.

3. Behavioral therapy: "Stop-and-start" method of ceasing genital stimulation until arousal sensation diminishes, or the "squeeze" method of squeezing the glans prepuce at heightened arousal.

C. Psychotherapy:

1. Recommended for lifelong, acquired, or variable PE, and is the first-line treatment for subjective PE.

D. Pharmaceutical therapy:

1. Off-label medications include serotonergic tricyclic antidepressants and selective serotonin reuptake inhibitors (SSRIs).

a. Serotonergic tricyclic antidepressants:

i. Clomipramine 25 to 50 mg/d.

b. SSRIs:

i. Paroxetine 10, 20, or 40 mg/d *or* 20 mg 3 to 4 hours preintercourse is the strongest among SSRIs in delaying ejaculation.

ii. Fluoxetine 5 to 20 mg/d.

iii. Sertraline 25 to 200 mg/d *or* 50 mg 4 to 8 hours preintercourse.

iv. Citalopram 20 to 40 mg/d.

2. Off-label medications also include eutectic mixture of local anesthetics (EMLA).

a. Lidocaine 2.5%/prilocaine 2.5% cream: Apply to glans penis 20 to 30 minutes preintercourse.

FOLLOW-UP

A. The client should be seen in 2 to 4 weeks if medication was started to assess for efficacy. SSRIs typically achieve maximum effect after 1 to 2 weeks.

B. Taper to discontinue any SSRIs is required. The SSRI withdrawal syndrome is characterized by dizziness, headache, nausea, vomiting, and diarrhea, and occasionally agitation, impaired concentration, vivid dreams, depersonalization, irritability, and suicidal ideation.

CONSULTATION/REFERRAL

A. May refer to a urologist or sexual health specialist/counselor, particularly if there is provider discomfort with this topic.

B. Psychotherapy is recommended for lifelong, acquired, or variable PE, and is the first-line treatment for subjective PE. Referral to a practitioner who specializes in sexual therapy is recommended.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. Off-label SSRIs should be used with caution for the increased risk of serotonin syndrome.

2. Off-label EMLA cream used without a condom may result in vaginal wall numbness in the sexual partner.

3. Prolonged application of EMLA may result in loss of erection if used for 30 to 45 minutes.

B. Geriatrics:

1. Geriatric syndrome assessments:

a. Delirium/behavioral disturbance: SSRI-related neurocognitive side effects include changes in mood, anxiety, and akathisia.

b. Falls: Consistently assess for falls. Antidepressants, alone or in combination, are highly anticholinergic, sedating, and cause orthostatic hypotension. Safety measures to reduce falls in the home include removing small area carpets, keeping night lights along the path to the bathroom, and installing a toilet seat with grab bars or nearby grab bars.

2. Pharmacologic caution in geriatrics:

a. Renal and hepatitis dosing of paroxetine is required. Hepatic dosing of fluoxetine and sertraline is required.

3. Beers list caution: SSRIs should be used with caution in older adults. Avoid total of more than three central nervous system (CNS)-active drugs; minimize number of CNS-active drugs. (Refer to Appendix C, Table C.4.)

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SEXUAL HEALTH ISSUES IN THE AGING POPULATION

DEFINITION

A. The World Health Organization has declared that sexual health is an integral part of overall health. Sexuality is an aspect of the human species that begins at birth and ends at death, despite socially accepted stereotypes. Sexuality is not strictly a component of procreation, but also an element of the entire individual, intertwined with religious, cultural, and spiritual beliefs. It frequently binds individuals into romantic relationships.

B. According to a survey conducted by AARP and the University of Michigan Institute for Healthcare policy and Innovation, 40% of respondents between the ages of 65 and 80 are sexually active. Seventy three percent of the respondents reported satisfaction with their sex life. Results of the survey found that 18% of older males and 3% of older females report taking medications of supplements to improve sexual function.

C. In 2013, the *Diagnostic and Statistical Manual of Mental Disorders (DSM)* released the fifth edition with significant changes to sexual dysfunction diagnoses:

1. One of the biggest changes was the combination of sexual interest and arousal disorders into one category with gender specificity.
2. Sexual aversion disorder was eliminated during the transition from the *DSM-IV-TR* to the *DSM-5.*
3. Genital arousal disorder has yet to be classified in the *DSM*.

INCIDENCE

A. The prevalence of sexual problems in the geriatric population is also frequently underestimated. According to the Global Study of Sexual Attitudes and Behaviors (GSSAB), nearly half of all sexually active respondents aged 40 to 80 reported at least one sexual complaint, including erectile dysfunction, premature ejaculation, lack of sexual interest, lack of sexual pleasure, inability to reach orgasm, and lubrication difficulties. Despite this, less than 19% sought medical attention for such issues.

B. Medical providers frequently underestimate the commonality of sexual activity in the older population.

C. Although sexual complaints are frequently considered specific to certain population genres, most of these diagnoses are actually age bimodal due to the variety of causative etiologies.

D. Although many older adults remain sexually active, they rarely bring up the topic of sexual health with their healthcare providers:

1. Although 20% of individuals aged 60 to 94 report having had intercourse within the previous 3 months, only 38% of males and 22% of females have discussed sexual behavior with their healthcare provider after the age of 50.
2. Barriers to pursuing medical care for sexual complaints include the following:
   a. Believing that the problem is not serious.
   b. Not being bothered by the problem.
   c. Belief that menopause-related changes were irreversible.
   d. Lack of awareness of available treatments.
   e. Lack of access or affordability of medical care.
   f. Embarrassment.

E. Across the globe, only 9% of males and females are asked about their sexual health by their healthcare providers, yet more than half of males and females consistently report sexual activity in the previous year. Barriers to sexual health history taking were the following:

1. Lack of time.
2. Interruptions.
3. Limited communication skills.
4. Inability to cope with issues that arise with sexual history response.
5. Embarrassment.
6. Feeling that taking such a history in the older population is not appropriate.

PATHOGENESIS

A. Although sexuality transforms throughout the life span and varies among individuals, we are typically born with similar constituents: reproductive organs, sex hormones, and a brain wired to receive nerve impulses from erogenous parts of the body.

B. At a very young age, individuals often identify areas of the body that induce pleasure with physical stimulation. Over the course of the first few decades of life, sexual structures and hormones mature, sexual functioning develops, and integration of partnered sexuality and intimacy often occurs.

C. After childbearing years, the aging process can complicate sexual functioning for both males and females. Unfortunately, this frequently terminates sexual activity despite medical capabilities to prevent and treat sexual disorders. If these changes are acknowledged and embraced, individuals can find themselves in just as satisfying a sexual point in their lives as ever. Some even report improvement from past years.

PREDISPOSING FACTORS

A. Chronic health conditions:

1. Cardiovascular disease.
2. Diabetes.
3. Obstructive sleep apnea (OSA).
4. Dementia/Alzheimer disease.
5. Parkinson disease.
6. Multiple sclerosis.
7. Cancer.
8. Arthritis.
9. Incontinence.

B. Depression.

C. Intrapartner violence.

D. Postsurgical condition/complication.

E. Physical disabilities.

F. Loss of a spouse/partner.

G. Side effects from medications:

1. Angiotensin-converting enzyme (ACE) inhibitors.
2. Beta-blockers.
3. Calcium channel blockers.
4. Nitrates.
5. Diuretics.
6. Cholesterol-lowering drugs.

▶

7. Antidepressants.
8. Tranquilizers.

COMMON COMPLAINTS

A. Sexual dysfunction in males encompasses the following problems:
1. Lack of desire/arousal.
2. Erectile dysfunction:
a. Inability to achieve or maintain an erection sufficient for satisfactory sexual performance.
b. Curvature of the penis with erection (Peyronie disease).
c. Problems with penetration.
d. Orgasmic disorder.
3. Ejaculation.
B. Alternately, in females, sexual complaints occur in the realms of:
1. Lack of desire/arousal.
2. Lack of orgasm.
3. Sexual/genital pain: lack of vaginal lubrication.
4. Urinary:
a. Dysuria.
b. Urinary frequency/urgency.

SUBJECTIVE DATA

A. Sexuality integrates into many layers of the individual and nearly always requires collaboration with a multidisciplinary team. Functioning as a sort of "gatekeeper" in this realm of medicine can help manage treatment recommendations from multiple providers for complicated, multifaceted, and sensitive health matters.
B. When addressing clients with potential sexual issues, a variety of questionnaires and assessment tools can help facilitate efficient communication in a busy medical setting. They can also aid in "breaking the ice" with potential uncomfortable topics. A study conducted in the United Kingdom identified that general practitioners do not discuss sexual health with their older clients because they feel it is not appropriate to discuss such issues with this age group and because they feel sexual health equates with younger people. Interestingly, these beliefs were found to be based on stereotypes versus actual client experience. In this circumstance, questionnaires would not only identify the presence of sexual complaints irrespective of age, but also foster client–provider communication. See Table 17.10 in Chapter 17, Gynecologic Guidelines, for a list of questionnaires that can help to identify sexual dysfunction in clients.
C. Mailing the questionnaire prior to the appointment: Not all questions are pertinent to each client and it is important to identify this in the cover letter.
2. Mailed questionnaires offer the opportunities for the following:
a. Answering sensitive questions in a private setting.
b. Some clients like to include their partners' input in the answering of questions, which is also helpful when paperwork is mailed prior to the appointment.
3. Allowing clients to leave questions blank indicates the following:
a. The question does not pertain to their situation.
b. They do not understand the question.
c. They would rather discuss them in the office.
D. In your interview, it is important to be considerate of the client's comfort level when addressing sensitive topics such as this, and potentially modify assessment techniques and terminology.

TABLE 15.7 VALIDATED SEXUAL HEALTH QUESTIONNAIRES FOR MALES AND FEMALES

Male	Female
EDITS	FSFI
EHS	FSDS-R
IIEF	DSDS
IPE	HSDD
PEDT	SFQ28
QEQ	ASFQ
SEAR	SQOL-F
SHIM	
SQOL-M	
AMS Scale	

AMS, Ageing Male Symptoms; ASFQ, Abbreviated Sexual Function Questionnaire; DSDS, Decreased Sexual Desire Screener; EDITS, Erectile Dysfunction Inventory of Treatment Satisfaction; EHS, Erection Hardness Scale; FSDS-R, Female Sexual Distress Scale-Revised; FSFI, Female Sexual Function Index; HSDD, Hypoactive Sexual Desire Disorder Screener; IIEF, International Index of Erectile Function; IPE, Index of Premature Ejaculation; PEDT, Premature Ejaculation Diagnostic Tool; QEQ, Quality of Erection Questionnaire; SEAR, Self-Esteem and Relationship Questionnaire; SHIM, Sexual Health Inventory for Men; SFQ28, Sexual Function Questionnaire; SQOL-F, Sexual Quality of Life-Female; SQOL-M, Sexual Quality of Life-Men.

E. During the client interview, consider factors that play a role in the client's potential belief structure regarding sexuality, such as age, culture, ethnicity, religious affiliation, and sexual orientation.

PHYSICAL EXAMINATION

A. Physical examination is dependent on the type of sexual dysfunction and other chronic medical conditions.

DIAGNOSTIC TESTS

A. Diagnostic testing is dependent on the type of sexual dysfunction and other chronic medical conditions.

PLAN

A. It is incredibly important to consider the entire individual when evaluating sexual health, utilizing a holistic approach inclusive of the mind, body, and spirit when addressing concerns and implementing treatment plans. An individualized approach includes consideration of age-related factors. It is also critical to involve partner(s) when appropriate and agreed upon by the client.
B. LGBT elderly:
1. More than two million older adults identify as LGBT. In addition, the National Social Life, Health and Aging Project (NSHAP) indicated approximately 4% of the 3,005 respondents reported at least one same-sex sexual relationship (SSSR). Although the age of the respondents fell between 57 and 85 years of age, those reporting at least one SSSR tended to be younger, more educated, in better health, and more likely to be actively working.
2. Rates of the LGBT population are generally considered to be underestimated, especially in the elderly population, due to fears of social stigmas and discrimination. Keeping ▶

in mind the age of this population, it is important to remember that up until the early 1970s homosexuality was considered a mental disorder in the *DSM* of the American Psychiatric Association (APA). In addition, many individuals may identify as heterosexual but still engage in some same-sex sexual contact.

3. Use of nonspecific and nonoffensive terms on paperwork and in an office encounter can help to facilitate rapport between a medical provider and client. Considering that a client experience begins in the waiting room, medical providers should make sure to include posters, handouts, books, magazines, and television shows that do not convey derogatory opinions of any client population. Also, use of client questionnaires that can be filled out privately prior to a face-to-face encounter allows clients to share personal details in a safe manner.

4. In the older LGBT community, most respondents identify as gay men (61%), followed by lesbian (33%), transgender (7%), bisexual men (3%), bisexual women (2%), and "queer" (1%).

5. As previously stated, homosexual contact is the most commonly reported sexually transmitted infection (STI) risk behavior in individuals older than 50. Although age differences in gay men do not seem to make a significant difference in likelihood of engaging in sexual or risk behavior, those with significantly older partners tend to take the receptive position more during anal intercourse.

C. Use of aids/toys/products:

1. Use of sexual aids and toys may be a taboo subject for many older persons. At the same time, changes that occur to sexual functioning with age often warrant modification and inclusion of products into sexual activity that may not have been necessary in the past. The conversation of such products with clients should be approached with sensitivity.

2. It can be helpful to compare use of sexual aids to other more socially acceptable therapies used on the body, such as weights to strengthen weak muscles. This analogy can normalize a previously uncomfortable concept.

3. As previously reviewed, changes occur to vaginal mucosa with age, including tissue thinning, dryness, and inelasticity:

a. Vaginal moisturizers and lubricants can drastically improve the quality of sexual encounters and potentially alleviate vaginal discomfort.

b. Clients should be instructed to avoid products with flavoring, dyes, and perfumes because many of these ingredients can irritate sensitive vulvovaginal mucosa.

4. Clients should be aware of personal allergies and sensitivities. Available lubricant and moisturizing products contain a variety of potential irritants that should be reviewed by the client before purchasing and using.

5. For clients with problems getting and maintaining erections, there are over-the-counter (OTC) options such as constrictive rings that fit at the base of the penis and help to hold blood in the erect penis. There are also masturbation sleeves that assist in enhancing sensation during self-stimulation and partnered manual stimulation. OTC penis pumps enhance blood flow in the penis, although the prescription versions are of better quality and sometimes covered by insurance.

6. Vibrators are a popular choice for enhancing vulvovaginal sexual response for clients reporting arousal and orgasm difficulties. There are many different shapes, sizes, speeds, colors, and compositions, which can be daunting for a client looking for their first vibrator. If possible, it can be helpful to have some examples to show clients in the office or even a safe website to view together so that an explanation of each type can be reviewed.

7. For clients in heterosexual relationships, there are constrictive penis rings with small vibrators attached to provide clitoral stimulation during penetration. There are also a variety of arousal creams, gels, oils, and so forth available, although, once again, they should be used with caution as there is always a potential for contact dermatitis.

8. Many elderly individuals are either not going to have transportation to get to a store that sells sexual aids and toys or they are not going to feel comfortable making that trip. Providing clients with resources such as safe websites or a local product representative can help facilitate ease of purchase. Another option that some clinics utilize is selling these products on site. This is a mutually beneficial endeavor, as it offers an additional revenue stream while providing clients the opportunity to purchase sensitive items in a safe and private manner.

9. Older clients may not be familiar with use of sexual aids and toys. Drawings and demonstrations on anatomic models in the medical office setting can help with proper use at home.

DIFFERENTIAL DIAGNOSES

A. Sexual dysfunction in the elderly.

B. Sexual interest/arousal disorders in males and females:

1. Consider a variety of etiologies in clients reporting bothersome sexual dysfunction, including physical health, relationship status, and psychosocial factors to best individualize the treatment regimen. Treating underlying cause(s) will frequently improve sexual functioning.

2. Depression consistently coincides with interest/arousal disorders. Approximately 40% of individuals with a sexual disorder have depression. Unfortunately, the most common treatment for depression, serotonin-specific reuptake inhibitors (SSRIs), frequently result in sexual side effects in both males and females. Although males report higher rates of sexual dysfunction related to SSRIs, females tend to experience more severe dysfunction.

3. Consider collaboration with a multidisciplinary team such as sex therapists, sexuality educators, medical specialists and subspecialists, physical therapists, and alternative health providers to capture all aspects of these conditions.

C. Erectile dysfunction (ED):

1. ED is the inability to achieve or maintain an erection sufficient for satisfactory sexual performance.

2. Frequency of ED increases with age and comorbidities.

3. ED can be a symptom of more concerning etiologies, including diabetes and cardiovascular disease, and should be considered when evaluating clients.

4. ED is a known comorbidity of testosterone deficiency. Additional comorbidities of testosterone deficiency include increased body weight, adiposity and increased waist circumference, insulin resistance, type 2 diabetes mellitus (DM), hypertension, inflammation, atherosclerosis/cardiovascular disease, and increased incidence of mortality. More severe testosterone deficiency-related symptoms correlate with higher cardiovascular risk in males.

5. Common prescriptive treatment for ED includes the following:

a. Phosphodiesterase-5 inhibitors (PDE5i):

i. Despite available options, only about 25% of individuals with ED are treated and PDE5is are the most frequently utilized medication. Interestingly, clients older than the age of 60 are much less likely to be treated than those age 40 to 59.

b. Injectable or urethral prostaglandins.

c. Exogenous testosterone replacement when applicable.

d. Additional treatment options include constrictive penis rings, vacuum erection devices (VED), and surgical placement of penile prostheses.

e. Some literature even suggests the benefit of pelvic floor muscles (PFMs) physical therapy for clients experiencing ED.

D. Orgasmic disorder:

1. Of females between 57 and 85 years of age, 35% to 38.2% were unable to achieve orgasm, comparable to 16.1% to 33% of males in the same age category. Both males and females experienced more of a prevalence with advancing age. In females, this complaint ranked second only to low desire and trouble lubricating. In males, inability to achieve orgasm ranked fifth to lack of interest in sex, anxiety about performance, trouble maintaining or achieving an erection, and climaxing too early.

2. For individuals to be diagnosed with orgasmic dysfunction, a delay in or absence of orgasm must happen following sufficient sexual stimulation.

3. Simple explanation of sexual physiology and average time to orgasm can help some clients identify a misunderstanding of expectations.

4. In cases of true orgasmic dysfunction, especially acute onset, clients should be worked up for underlying causes such as cardiovascular and/or neurologic etiologies.

5. Visual examination of the genitals can help to rule out skin changes that can impede sensation.

E. Premature ejaculation:

1. Premature ejaculation (PE) occurs in approximately 21% to 30% of males, with less prevalence in older males. This data coincide with worsening incidences of ED with advancing age.

2. The consensus on the definition of premature ejaculation as stated by the International Society for Sexual Medicine (ISSM) is "ejaculation which always or nearly always occurs prior to or within about 1 minute of penetration." The typical ejaculatory latency in males is between 4 and 8 minutes.

3. Although there is no clearly defined cause for PE, some research suggests a correlation with multiple causes, including genetic predisposition, serotonin receptor activity, elevated penile sensitivity, and nerve conduction atypias. More recent evidence has shown a connection with PE, ED, and folate deficiency, possibly due to the effect of folic acid on the metabolism of nitric oxide.

4. Clients with PE often report embarrassment over their condition and frequently avoid sexual relationships because of it. Pharmaceutical treatment options utilized over the years include topical anesthetics, PDE5is, and antidepressant medications.

5. Combining pharmaceutical intervention with behavioral, cognitive, and sex therapy techniques is the most likely regimen to prove efficacious for premature ejaculation.

F. Genito-pelvic pain/penetration disorder:

1. In the general female population, anywhere from 7% to 58% of females report pain with intercourse. Specifically in the older population, 12.7% of females between the ages of 40 to 80 report pain with sex.

2. There are many potential causes for painful intercourse in older females, including the following:

a. Genitourinary symptoms of menopause (GSM).

b. Disuse atrophy.

c. Pelvic floor dysfunction.

d. Changes to vaginal anatomy due to surgery, such as vaginal hysterectomy.

e. Vulvovaginal skin conditions and infections.

f. In addition, physical disabilities, chronic pain, and cognitive changes can negatively impact the sexual response cycle, leading to pain during vaginal penetration.

3. Estrogen plays a direct role in vulvovaginal epithelial cell maturation, which subsequently maintains healthy vulvovaginal pH and appropriate vaginal flora. During the perimenopausal period and following both natural and surgical menopause, significant changes occur to the vaginal microbiota in response to loss of endogenous estrogen. Symptoms of vulvovaginal atrophy (VVA) occur in approximately 50% of postmenopausal individuals, including burning with urination, painful intercourse, bleeding with intercourse, vaginal discharge, vulvovaginal soreness, itching, and burning. Given the prevalence of this condition in older individuals, this population should be screened for signs and symptoms of VVA.

4. There are many Food and Drug Administration-approved treatment options for VVA including local estrogen replacement and an oral selective estrogen receptor modulator (SERM):

a. Not all clients are candidates for treatment with hormones and/or estrogen agonists. Clients should be counseled and treated on a case-by-case basis, taking into consideration their medical history.

b. In certain situations, collaboration with the client's other healthcare providers can help weigh risk versus benefit of traditional hormonal treatment for VVA.

5. Vaginal surgeries such as hysterectomies and repairs can lead to scarring, strictures, and shortening of the vaginal canal. These surgical complications combined with VVA can result in tremendous pain with vaginal penetration, including intercourse, use of sexual aids and toys, and even pelvic examinations. If appropriate, 6 weeks of preoperative local vaginal estrogen can improve tissue integrity, including synthesis of mature collagen, decreased degradative enzyme activity, and increased thickness of the vaginal wall. This suggests improved tolerance for suturing and maintenance of connective tissue integrity for prolapse repair without significant changes to circulating estrogen levels.

6. Pain with intercourse for any reason can result in avoidance of sexual contact:

a. Avoidance of sexual contact can lead to disuse atrophy, further worsening painful sex.

b. Fear of pain further compounds the problem and frequently the PFMs become involved. Muscle tension is a natural guarding response that occurs as a protective mechanism in the presence of pain. PFM ▶

dysfunction frequently accompanies pelvic pain and painful intercourse.

c. Incorporation of PFM rehabilitation with a trained pelvic floor physical therapist is crucial to properly addressing these issues.

d. Although sexual pain for males is not classified in the *DSM*, pelvic floor physical therapy (PFPT) can benefit males with pelvic floor dysfunction as well.

7. Given recent changes to the recommendations for annual Pap testing in the older female population, many females are receiving less frequent routine pelvic medical care:

a. Because the vulva and vagina are not overtly visible without intentional inspection, this area of the body can easily be missed, especially if conditions occur that are asymptomatic. For example, vulvar skin changes, vaginal pH shifts, and asymptomatic vaginal infections will not be identified without medical evaluation.

b. It is important to maintain vulvovaginal inspection and pelvic examination in older females at least on an annual basis to identify changes from baseline:

i. Use of colposcopy for the vulva and vagina can be helpful to better visualize mild skin changes.

ii. If consent is obtained, photography is a great way to document anatomic variances for future reference.

iii. There should be a low threshold for performing a biopsy with any concerning and grossly unidentifiable tissue findings.

G. Peyronie disease:

1. Although male sexual pain is not classified in the *DSM*, Peyronie disease is an incurable condition involving fibrotic plaques of the penis that can result in a palpable plaque, curvature of the penis with erection, and pain with sexual activity. Penetrative intercourse can also result in partner discomfort due to the curvature.

2. In addition to the physical sequelae, males report embarrassment and psychological distress over the aesthetic changes and sexual implications. Younger males are prone to this condition due to the frequency and intensity of intercourse, although there is a peak of incidence in older males likely due to weaker erections; this causes more opportunity for tunical trauma secondary to erectile dysfunction.

3. Treatment is available, including injectable therapies, interferon alpha-2b, and collagenase clostridium histolyticum, as well as surgery, although efficacy varies based on the severity of symptoms.

H. STIs:

1. The medical provider should never assume potential for STIs based on client age. It has been stated that sexual activity does not terminate at a certain age. Therefore, it is important to consider the fact that most sexually active individuals will contract an STI at some point in their lives.

2. Many cases of STIs remain undiagnosed and many STIs can have long-term sequelae especially if left undiagnosed and untreated. The most common risk behaviors in individuals older than 50 include the following:

a. Homosexual contact.

b. Intravenous drug use.

3. According to the Centers for Disease Control and Prevention (CDC), sexually transmitted diseases in the United States reached an all-time high for the sixth year in a row. Although STIs predominately occur in the younger population, rates of STI episodes in the older population have been on the rise.

a. Human papillomavirus (HPV):

i. Although the prevalence rates of new HPV infection decline with age, there is still a significant prevalence in the older population.

ii. The rate of progression from new infection to cervical intraepithelial neoplasia (CIN) 2+ or CIN 3+ was not higher for females aged 34 years and older comparable to younger women after 3 years of follow-up. Newly detected HPV infection does not typically persist or progress onto CIN 2 or worse disease regardless of age.

iii. *See Client Teaching Guide for Chapter 18, "Human Papilloma Virus."*

b. Trichomoniasis:

i. Approximately 50% to 60% of *Trichomonas* infections are asymptomatic in females.

ii. Whereas some of the adverse health outcomes from infection with *Trichomonas vaginalis* (TV) are not a concern in the older client population, such as preterm birth and lowbirth weight infants, trichomoniasis has been shown to increase the risk of HIV infection.

iii. *See Client Teaching Guide for Chapter 18, "Trichomoniasis."*

c. HIV:

i. Despite comparable rates of reported sexual risk factors in the younger versus older population, late middle-aged (50–59 years old) and old adults (60–75 years old) are less likely to use condoms and to have ever had an HIV test. In 2018, one in six new HIV diagnoses were among people who were aged 50 and older. HIV/AIDS knowledge and risk perception, perceived behavioral control, and behavioral intentions toward condom use are lower among older adults compared with their younger counterparts.

ii. According to the CDC, at the end of 2018 an estimated 379,000 people aged 55 and older have HIV.

1) People with HIV are living longer.

2) Older adults are more likely to be diagnosed with HIV later in the course of their disease, usually because they are unaware of risk factors.

iii. Adults older than 60 are less likely to discuss HIV/AIDS with family, friends, and medical providers.

iv. *See Client Teaching Guide for Chapter 20, "Reference Resources Regarding HIV/AIDS."*

d. Herpes simplex virus (HSV):

i. Painful ulcers result from HSV presentation; asymptomatic HSV type 2 shedding can contribute to chronic inflammation in the genital tract and painful intercourse.

ii. The social stigma associated with HSV infection complicates those entering into new sexual relationships. Older clients with clinically diagnosed genital herpes should be counseled on preventive measures as well as ways to approach the topic with potential partners. Informing clients of the prevalence of this STI can help to lessen the fear and shame often associated with HSV.

iii. *See Client Teaching Guide for Chapter 18, "Herpes Simplex Virus."*

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SEXUALITY AND CHRONIC HEALTH CONDITIONS

A. Lifestyle and genetics play a role in the aging process of each individual. Medical intervention for age-related ailments varies widely across different client populations. With advancing medical technology and research, humans are living longer; with that, a larger proportion of the life span is spent with chronic health ailments and conditions. Medications, surgeries, and other medical interventions can further complicate sexuality in the aging population.

1. Cancers:

a. In the United States, the five leading cancer sites in females by prevalence are breast, lung, colon/rectum, uterine, and thyroid. In males, the five leading cancers occur in the prostate, lung, colon/rectum, bladder, and melanoma of the skin.

b. Many treatments for cancer can have a significant impact on sexual functioning. These effects can persist throughout the remainder of the individual's life.

c. The psychological burden of a potential life-threatening condition impacts every facet of life, including sexuality and intimacy. This is relevant for both the client as well as their partner.

d. Although there has been a tremendous amount of research in the area of nerve-sparing surgeries for prostate cancer, urologic and sexual side effects remain a significant concern. Common side effects of radical prostatectomy include orgasmic dysfunction, urinary incontinence (UI) during sexual activity, penile sensory changes, penile shortening, and penile deformity.

e. Among females following mastectomy, significantly more report problems with sexual desire, arousal, the ability to achieve an orgasm, and intensity of the orgasm.

f. Both prostate and breast cancer treatment regimens frequently involve endocrine therapy that suppresses or eliminates circulating estrogen and/or testosterone:

i. Although these individuals may have already a decline in hormone levels depending upon age at initiation of suppressive treatment, the abrupt shift can prove quite detrimental to overall quality of life.

ii. With androgen deprivation therapy for prostate cancer, males frequently experience symptoms similar to females in menopause, including hot flashes, cognitive dysfunction, and sexual complaints such as erectile dysfunction, orgasmic difficulty, and loss of sexual desire.

iii. In females, aromatase inhibitor-treated breast cancer clients report more sexual complaints than tamoxifen-treated clients and controls. Although only 31% of tamoxifen-treated clients reported painful intercourse, 57% of aromatase-inhibitor-treated clients reported the same. Aromatase inhibitor-treated clients also reported a 74% incidence of insufficient lubrication.

2. Diabetes:

a. Diabetes mellitus (DM) is on the rise in the United States. In males with recently diagnosed type 2 DM, there is a high prevalence of sexual dysfunction. In this population, erectile dysfunction (ED) was classified as either mild, mild-moderate, moderate, or severe, with a cumulative prevalence of nearly 67%. More than 21% described their ED as severe.

b. Additional sexual comorbidities include premature ejaculation, delayed ejaculation, and hypoactive sexual desire. Considering the progressive nature of diabetes, early diagnosis, counseling, and management can aid in preservation of sexual functioning.

c. Sexual dysfunction is more prevalent in females with all types of diabetes, although more so in those with type 2 DM. In females with diagnosed DM, nearly one in four experience sexual dysfunction, although degree of severity varies based on type of diabetes, diabetic complications, hemoglobin A1C value, and presence of depression.

d. Low Female Sexual Function Index (FSFI) values correlate with high body mass index (BMI). This is of note with type 2 DM clients given the comorbidity of obesity.

e. Among studies of sexual functioning in both males and females with DM, clients consistently have a higher prevalence of depression, which further negatively impacts sexual functioning.

f. *See Client Teaching Guide for Chapter 24, "Diabetes."*

3. Sleep apnea:

a. Sleep apnea is a chronic breathing disorder that disrupts sleep. In addition to the physical impact on the individual, partners are often bothered by the sound of heavy breathing and loud snoring. Because of this, sleep dynamics may change and result in the couple sleeping in separate beds and/or rooms.

b. Approximately 60% of males with diagnosed obstructive sleep apnea (OSA) have concomitant ED. Although the use of continuous positive airway pressure (CPAP) is effective at managing OSA, only about 29% of males report improvement in ED. Use of phosphodiesterase-5 inhibitors (PDE5i) has been helpful in this client population, even when CPAP is not included in the treatment regimen.

c. There is limited understanding of sexual health in females with OSA, although multiple studies have shown an increase in sexual dysfunction concurrent with OSA. Some evidence suggests that sexual dysfunction correlates with OSA in obese females only if nocturnal hypoxia is present. Other than this, severity of OSA has not consistently been linked with degree of female sexual distress (FDS).

d. Refer to Chapter 12, "Respiratory Guidelines," for further detailed information on OSA.

4. Cardiovascular disease:
   a. After a cardiac event, clients are frequently left with uncertainty of their ability to resume sexual activity. Although clients turn to their healthcare professionals for guidance, most medical providers do not address the topic of sexuality after a cardiovascular event, specifically myocardial infarctions (MIs).
   b. Many individuals with a history of cardiovascular event(s) are fearful of resuming regular physical activity, including sexual activity, due to fear of recurrence or even death:
      i. A study intended to observe changes in blood pressure (BP) and heart rate during sexual activity showed that these measurements increase just slightly for a short time and recover their baseline level soon after sexual activity for healthy adults.
      ii. Given this knowledge, many medical providers are comfortable allowing clients to resume some form of sexual activity within a reasonable amount of time following a cardiovascular event. This decision needs to be made on a case-by-case basis.
   c. Although cholesterol-lowering medications can benefit erectile function in men with concomitant ED and hypercholesterolemia, many of the medications used to treat and manage cardiovascular health conditions can have a negative effect on sexuality.
   d. There have been some data to suggest a correlation with 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitor medications, otherwise known as statins, and low libido in males. Sex-specific research regarding the effect of statins on the sexual health of females is lacking.
   e. Hypertension in females can lead to sexual dysfunction in the areas of libido, arousal, and orgasm more so than males. Whereas beta-blockers are known to produce sexual side effects, treatment with alternative medications, and lifestyle modifications can potentially reduce sexual dysfunction in this population.
   f. Refer to Chapter 13, "Cardiovascular Guidelines," for further detailed information on cardiovascular conditions.
5. Dementia/Alzheimer disease:
   a. Sexuality can be significantly impacted by dementia and Alzheimer. The most commonly reported sexual dysfunction is disinterest due to apathy and blunted affect. Spouses can develop disinterest and poor sexual functioning in response to worsening of partner's cognitive decline.
   b. In severe cases, hypersexual tendencies occur as a manifestation of disinhibition in clients with dementia and Alzheimer. Examples of this include sexual comments, public exposure masturbation, and unwarranted sexual proposals.
   c. Refer to Chapter 23, "Neurologic Guidelines," for further detailed information on dementia/Alzheimer.

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SEXUALITY AND END OF LIFE

A. Many couples maintain sexual activity even in the presence of terminal illness. Sexual functioning should be acknowledged and included in end-of-life planning with the medical provider with the understanding that sexuality and intimacy extend beyond penetrative intercourse.

B. Physical limitations warrant modification, not termination, of sexual activity if both partners desire continued sexual contact.

C. By initiating the conversation with the client and their partner, the medical provider normalizes this concept and establishes an open communication forum. Subsequently, clients may be more open to appropriate sexual health recommendations and referrals.

SEXUALITY AND PHYSICAL DISABILITIES

A. Physical health problems are one of the most commonly reported reasons for sexual inactivity. With the increasing incidence of physical ailments that occur with aging, most elderly individuals will experience an acute or chronic disability at some point that impacts sexual functioning.

B. When a couple in a sexual relationship encounters independent changes in their physical health, sexual functioning becomes complicated twofold. Acknowledging and addressing these changes can help alleviate stress surrounding individual and partnered sexual functioning.

C. In many cases, certain sexual positions are not possible to maintain with physical disabilities. At the same time, sexually active individuals may not be familiar with alternative sexual positions and practices. Older clients may not be comfortable with certain aspects of sexuality. According to the 2009 AARP survey, 20% of males and 12% of females have oral sex at least once a week. Whereas cunnilingus and fellatio may not have been a form of sexual activity included in the practices of older couples prior to the onset of a disability, inclusion of this practice can help to maintain or even resurrect sexual activity in the cases of erectile dysfunction (ED), premature ejaculation, diminished arousal/orgasm, painful intercourse, physical disabilities, and so forth.

D. In addition to oral sex, clients can frequently benefit from education on alternative sexual practices, positions, and use of aids and toys. Referral to a certified sex therapist and/or a sexuality educator can benefit clients in this situation.

E. Because neurologic functioning is so critical to the sexual response cycle, neurologic conditions can significantly impede sexual functioning. Lower urinary tract symptoms are common in clients with multiple sclerosis, and detrusor overactivity is an independent predictor of ED and female sexual dysfunction. This same study further supported the connection with depression and sexual dysfunction in both male and female clients.

F. In males with multiple sclerosis, ED is the most common sexual complaint, with prevalence over 50%, followed by decreased sexual desire, then difficulty reaching orgasm and ejaculation. Despite this prevalence and negative impact on ▶

sexual quality of life, few males with multiple sclerosis report sexual complaints to their healthcare providers. The length of disease does not seem to play a role in male sexual complaints, although it does with females.

G. Parkinson disease is another progressive disease of the neurologic system that can impact sexual functioning. Over 60% of individuals with Parkinson report fatigue. Other commonly reported symptoms include changes to bowel and bladder habits, nervousness, feelings of sadness, restless legs, pain, dribbling saliva, and excessive sweating:

1. In males with Parkinson disease, sexual dysfunction is a common symptom that also correlates with depression.

2. In females with Parkinson, the most influential factors on sexuality are depression as well as anxiety. Females with this disease report more problems with mood and apathy, whereas males report more sexual dysfunction.

CONSULTATION/REFERRAL

A. Despite the prevalence of sexual activity in the older population and the quality of life impact of sexual dysfunction, few medical programs incorporate sexual health training into their curriculum outside of sexually transmitted infections (STIs) and reproduction. Even fewer address specific considerations for the geriatric population. Because of this, it is up to the medical provider to seek the latest information on caring for sexual health issues:

1. Providers should acknowledge sexuality as an important component of overall health when caring for our older clients. It is also necessary for providers to either manage sexual dysfunction or refer out when appropriate.

2. Collaboration with outside healthcare providers fosters a multidisciplinary approach to a complex medical entity. Sexual dysfunction, especially in the elderly population, is nearly nonexistent in medical training.

3. Although few medical programs incorporate sexual health training into their curriculum, especially in relation to the older client population, there are a variety of organizations available to help foster medical research and clinical practice in the realm of sexual medicine.

4. It is helpful to establish a network of referral sources in the local area with which to collaborate, including pelvic floor physical therapists, sex therapist, sexuality educators, and medical specialists such as urologists, gynecologists, and oncologists.

5. Some key organizations to consider becoming involved in include the International Society for Sexual Medicine (ISSM), International Society for the Study of Women's Sexual Health (ISSWSH), Sexual Medicine Society of North America (SMSNA), American Association of Sexuality Educators, Counselors and Therapists (AASECT), and the Society for Sex Therapy and Research (STAR).

INDIVIDUAL CONSIDERATIONS

A. Divorcees/widows/widowers:

1. The United States has the highest rate of divorce in the world, with approximately 45% of marriages expected to end in divorce. According to the U.S. Census Bureau, 17.5% of people age 50 and older were either divorced or separated as of 2011. This age group is the only population where divorce rates are rising, which further supports the need for sexuality education with older clients.

2. Infidelity is a fairly common occurrence in U.S. marriages, from which older couples most certainly are not immune. From the AARP Sex, Romance, and Relationships survey, 21% of male respondents and 11% of female respondents admitted that they cheated during a current or recent long-term relationship, yet only 12% of both sexes say that their partner cheated on them. This identifies a discrepancy between true infidelity in this population and partner awareness:

a. Social media allows individuals to connect who previously may not have had contact. Interactions via technology allow for private communication that facilitates loss of inhibitions that may play more of a role in face-to-face encounters. As younger generations age, social media will likely become even more commonplace with advancing technology.

b. Younger couples are adhering less to some of the strict beliefs, values, and morals that prevented many older couples from having premarital sexual activity. As this younger population ages, individuals are more likely to have had multiple sexual partners prior to their marriage, potentially leading to difficulty with long-term monogamy.

c. Physical changes including disabilities, chronic health conditions, and aesthetic transitions can impact sexual attraction, leading partners to possibly seek out something "better."

d. Despite the potential increase in infidelity in coming years within the older population, only about 6% of respondents from the AARP survey reported that infidelity was the cause of their breakup. Some respondents actually reported that infidelity enhanced their relationship; 25% of cheaters reported that their infidelity improved their sex lives and 11% of the partners agreed.

e. Marital status does not seem to play a role in the prevalence of male ED, although having never been married and widowhood are predictors of low sexual desire in males.

f. Older widowers are more likely to remarry than older widowed females. Younger age correlates with increased likelihood of becoming involved in a new relationship for females, although higher monthly income and level of education were better predictors for males.

3. There is minimal research in the realm of sexuality after spousal loss. Widows report a lower incidence of sexual intercourse than widowers and they are more likely to report lack of sexual desire:

a. Recent partner loss has been shown to be a significant risk factor for depression in the elderly, which is a known correlating factor for sexual dysfunction.

b. For sexually active widows and widowers, four key needs reported to maintain sexual desire are the following:

i. Good health.
ii. Good sexual functioning.
iii. Positive sexual self-esteem.
iv. Sexually skillful partner.

B. Survivors:

1. The emotional burden of losing a spouse or partner is frequently compounded by survivor's guilt. It is not uncommon for the surviving partner to feel uncomfortable or uneasy about developing feelings toward new partner(s). There is the guilt of disrespecting the memory of the deceased, the social stigma of being perceived as "moving on too fast," and the fear of the unknown. ▶

2. Many older widows and widowers have been in a monogamous relationship for an extended period of time and are unfamiliar with modern-day dating practices. In addition, it is completely normal for a widow/widower to inadvertently compare new intimate and sexual partners with the deceased spouse. This makes for potential interpersonal conflict as well as partner discomfort.

3. Many spouses take on the role of caregiver in the situation of a chronic illness. Caretaker guilt can develop during this period of time, which hinders the caregiver's psychosocial and somatic adjustment of this role. The abrupt change in role from caregiver to grieving widow following spousal death has the potential to be extremely psychologically taxing and should be acknowledged by the medical provider. Referral to a grief counselor familiar with such situations can be very helpful in this circumstance.

4. For spousal loss related to suicide, there is added sensitivity. Bereavement group postvention (BGP) therapy has been shown to reduce anger, guilt, and despair. Because of the stigma attached to suicide, surviving partners may find it difficult to bring up their experience with future partners.

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TESTICULAR TORSION

DEFINITION

A. Testicular torsion is twisting of the testicle around the vas deferens, with compromise in the blood supply and possible necrosis to the testicles. Testicular torsion is a urologic emergency and is the most frequent cause of testicle loss in the adolescent male population. Approximately 40% of all cases of acute scrotal pain and swelling are diagnosed with testicular torsion. There is often a history of recurrent episodes of testicular pain before torsion.

B. Testicular torsion is most commonly misdiagnosed as epididymitis:

1. Epididymitis usually presents with gradual onset of pain that is localized posterior to the testis that gradually radiates to the lower abdomen.

2. These symptoms are rare with torsion.

INCIDENCE

A. Incidence in males younger than 25 years old is approximately 1 in 4,000. Although torsion can occur at any age, the largest number of cases occurs during adolescence.

B. The bell clapper congenital anomaly is present in approximately 12% of males, and 40% have the abnormality in the contralateral testicle.

PATHOGENESIS

A. Testicular torsion and torsion of the spermatic cord are caused by abnormal fixation of the testicle to the scrotum, allowing free rotation. The bell clapper deformity allows the testicle to twist spontaneously on the spermatic cord. Venous occlusion and engorgement cause arterial ischemia and infarction of the testicle.

PREDISPOSING FACTORS

A. Age: more common in adolescence.
B. Trauma to testicle.
C. Spontaneous occurrence.
D. Congenital bell clapper anomaly.
E. Exercise.
F. Undescended testicle.
G. Active cremasteric reflex.

COMMON COMPLAINTS

A. Sudden onset of severe unilateral scrotal pain (less than 24 hours).
B. Swelling of scrotal sac.
C. High position of the testicle.
D. Abnormal cremasteric reflex.

OTHER SIGNS AND SYMPTOMS

A. Sudden onset of testicular pain, which may radiate to groin.
B. Possible edema.
C. Abdominal pain (20%–30%).
D. Nausea and vomiting (50% of cases).
E. Fever (16%).
F. Urinary frequency (4%).

SUBJECTIVE DATA

A. Review the onset, duration, and course of symptoms.
B. Review for a history of episodes of intermittent testicular pain that resolved spontaneously.
C. Review abdominal symptoms such as pain, nausea and vomiting, and fever.
D. Review urethral discharge (possible sexually transmitted infection [STI]) and dysuria.
E. Review the client's history for trauma to the scrotum or testicle.

PHYSICAL EXAMINATION

A. Check temperature (if infection is suspected), blood pressure (BP), pulse, and respirations.
B. Inspect:
1. Observe the client generally for pain before and during examination.
2. Visualize the scrotal sac for edema, symmetry, lesions, discharge, and color (especially for blue dot superior to the affected testicle). Testis is located high in the scrotum as a result of shortening of the cord by twisting.
3. Check the inguinal and femoral areas for bulges and hernias.
C. Auscultate:
1. Auscultate all four quadrants of the abdomen; note bowel sounds.
2. Assess the scrotum for bowel noise.
D. Palpate:
1. Palpate the abdomen for masses, rebound, and tenderness or guarding.
2. Palpate the groin; check the lymph nodes.
3. Examine for an inguinal hernia. ▶

4. Genital exam:
   a. Check warmth, tenderness, swelling, and any nodularity; if a mass is present, check if it is solid or cystic. The testes should be sensitive to gentle compression but not tender. They should feel smooth, rubbery, and free of nodules.
   b. Elicit a cremasteric reflex by stroking the inner thigh with a blunt object (reflex hammer or ink pen). The testicle and scrotum should rise on the stroked side. **Cremasteric reflex is usually absent in testicular torsion.**
   c. Elevate the scrotum; there is usually no relief in pain with torsion. Elevation of the scrotum may improve the pain of epididymitis (Prehn sign).

DIAGNOSTIC TESTS

A. Urinalysis: normal in 90% of cases of testicular torsion.
B. Doppler ultrasonography for blood flow and scrotal ultrasonography.

DIFFERENTIAL DIAGNOSES

A. Testicular torsion: Firm, tender mass of acute onset in an afebrile young male with a history of prior episodes must be considered to represent torsion until proven otherwise.
B. Epididymitis.
C. Orchitis.
D. Hydrocele.
E. Testicular tumor: usually a hard, enlarged, painless testicle.
F. Acute appendicitis.
G. Scrotal/testicular trauma.
H. Varicocele.

PLAN

A. General interventions:
   1. Immediately refer the client to a urologist and/or ED.
   2. Symptoms lasting *more than 6 hours* can indicate testicular necrosis.
B. Client teaching:
   1. Educate client/family regarding risks involved with testicular torsion and emergent need of treatment.
   2. Support client and family.
C. Pharmaceutical therapy:
   1. None.

FOLLOW-UP

A. Client should follow up with the urologist as directed.

EMERGENT ISSUES/INSTRUCTIONS

A. Testicular torsion is a urologic emergency requiring surgery. Instruct the client to go to the ED.

CONSULTATION/REFERRAL

A. All clients with suspected testicular torsion need to be immediately evaluated by a physician and referred to a urologist. Testicular torsion may result in an infarction within 6 hours.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
   1. In older males, rule out epididymitis, especially with new sexual partners or with symptoms of dysuria and urethral discharge. (Refer to section "Epididymitis.")

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URINARY INCONTINENCE

DEFINITION

A. Urinary incontinence (UI) is the complaint of involuntary loss (leakage) of urine. The condition occurs in both males and females but is much more frequent in females. UI can be severe enough to have unpleasant social or hygienic consequences. UI is diagnosed primarily on history; inquire about UI at every interview. UI is a symptom of an underlying disease process in most cases; some cases are reversible with appropriate treatment. The International Consultation on Incontinence Questionnaire (ICIQ) or the ICIQ Short Form Questionnaire for UI survey can be requested from www.iciq.net/background.htm.
B. Voiding up to seven times per day in the waking hours is considered normal with a micturition volume of 250 to 300 mL per void (although the volume is typically higher with the first morning void). A healthy adult bladder can have a comfortable tolerance of approximately 500 mL and can accommodate this relatively large volume of urine with little discomfort.
C. Incontinence is not considered part of normal aging. Morbidity related to incontinence includes urinary tract infections (UTIs), indwelling catheters, falls/fractures, sleep interruption, social withdrawal, and depression.
D. Successful toileting depends on ready access to facilities, motivation to remain dry, mobility and manual dexterity, and the cognitive ability to recognize/react to the urge to void.
E. UI can be divided into the following categories: functional, urge, overflow, stress, mixed, and postvoid-dribbling. Each category has a unique etiology, pathophysiology, symptoms, and management.

INCIDENCE

A. The exact incidence of UI is unknown. The prevalence of incontinence increases with age.
B. The incidence of females identified with the definition of any leakage at least once in the past year ranges from 25% to 51%. The incidence is reported to be 10% when identified with weekly urinary leakage. The 2014 American College of Physicians guideline notes the following incidence of UI in females:
   1. In young females (aged 14–21 years), the incidence is approximately 25%.
   2. Incidence of middle-aged and postmenopausal females (aged 40–60 years) is approximately 44% to 57%.
   3. The elderly (≥75 years of age) have approximately a 75% incidence of UI.
C. The prevalence of UI in males is approximately half that of females. The incidence of UI is affected by treatment of prostate disease. Males with incontinence have a higher risk of institutionalization compared with males without UI. males are sometimes reluctant to bring up the topic to their clinician. Only one in five males seek treatment for UI.
D. Elderly clients are more frequently affected with UI; 6% to 10% of admissions to long-term care facilities are related to incontinence.
E. Clients with mild cognitive impairment are 30% more likely to have UI.

PATHOGENESIS

A. UI can be caused by pathologic, anatomic, or physiologic factors and differs by type of incontinence:

1. Functional incontinence: Loss of urine because of the inability to get to the bathroom, either due to problems of mobility or cognition.

2. Urge incontinence/overactive bladder (OAB): inability to delay voiding after the sensation of fullness is perceived. Common causes are detrusor hyperactivity or hyperreflexia associated with disorders of the lower urinary tract, tumors, stones, uterine prolapse, cystitis, urethritis, or impaired bladder contractility. Central nervous system (CNS) disorders such as stroke, dementia, parkinsonism, or spinal cord injury also can be causative factors.

3. Overflow incontinence: Urine is retained in the bladder after voiding due to incomplete emptying after an attempt to urinate. Common causes are anatomic obstruction by an enlarged prostate, a prolapsed cystocele, a contractile bladder caused by diabetes, spinal cord injury, multiple sclerosis (MS), or suprasacral cord lesions.

4. Stress incontinence: involuntary loss of urine during coughing, sneezing, laughing, bending over, or other physical activity that increases intra-abdominal pressure. Prostate surgery is the most common cause of stress incontinence in males.

5. Mixed incontinence: combination of stress and urge incontinence in which the bladder outlet is weak and the detrusor is overactive.

6. Postvoid dribble (PVD): Urine is retained in the urethra after the bladder has emptied. It causes wetness soon after the client has completed urinating but not at other times. PVD is most commonly idiopathic. It may occur along with other lower urinary tract symptom (LUTS) or independently of LUTS. As an isolated symptom, PVD is most commonly idiopathic, although rarely it is due to a urethral stricture or diverticulum or to hypospadias.

PREDISPOSING FACTORS

A. Age for both males and females.
B. Female: 85% of cases are in females.
C. Increased parity.
D. Previous genitourinary (GU) surgeries (e.g., prostate surgery and hysterectomy).
E. Restricted mobility.
F. Menopause.
G. Infections.
H. Chronic illnesses (e.g., diabetes).
I. Fecal impactions.
J. Excessive urinary output.
K. Delirium.
L. Dementia.
M. Neurologic disorders (e.g., stroke, spinal cord injury).
N. Variety of medications (e.g., antihypertensive medicines, diuretics, sedatives).
O. Pelvic trauma (e.g., episiotomy, forceps delivery).
P. Obesity.
Q. Sleep apnea.
R. Depression.
S. High-impact exercise.

COMMON COMPLAINTS

A. Urgency: sudden and compelling desire to pass urine.
B. Urge incontinence: involuntary leakage accompanied by urgency with the following precipitating factors:

1. Hearing running water.
2. Placing hands in water.
3. Trying to unlock the door when returning home.
4. Exposure to a cold environment.

C. Stress incontinence: involuntary leakage with the following precipitating factors:

1. Exertion.
2. Sneezing/coughing.
3. May experience leakage with little or no activity.

D. OAB: Symptoms may occur with or without urge incontinence.

1. Urgency.
2. Frequency.
3. Nocturia.

OTHER SIGNS AND SYMPTOMS

A. Mixed incontinence: urge and stress leakage.
B. Experiencing leakage with little or no activity.
C. Continuous leakage (i.e., dribbling).
D. Daytime frequency.
E. Nocturia: up one or more times a night to void.
F. Slow urine stream, intermittent stream, or hesitancy.
G. Need to strain to start, maintain, or improve voiding.
H. Incomplete emptying sensation.

SUBJECTIVE DATA

A. Question the client regarding onset, duration, and severity of the incontinence:

1. Do you ever leak urine when you do not want to?
2. Do you ever leak urine when you cough, laugh, or exercise?
3. Do you ever leak urine on the way to the bathroom (urgency)?
4. Do you ever use pads, tissue, or cloth in your underwear to catch urine?

B. Elicit situations when UI is worse, when it is improved, and what stimuli are associated with increasing UI (high fluid intake, high caffeine intake, agitation).
C. Review whether the client is pre- or postmenopausal.
D. Review other LUTS such as nocturia.
E. Review the client's history of bowel function (e.g., fecal incontinence). If constipation is a problem, abdominal pressure from a large, retained stool can cause symptoms, including retention.
F. Review medications, including over-the-counter (OTC) drugs and herbals:

1. Review if the presence of medications, such as angiotensin-converting enzyme (ACE) inhibitors, can trigger a chronic cough.
2. Review medications prescribed for UI:
a. Evaluate their effectiveness.
b. Evaluate if the medications were discontinued due to side effects:
i. Dry mouth (oxybutynin).
ii. Constipation.
iii. Dizziness (trospium).
iv. Blurred vision.
v. Insomnia (oxybutynin).
vi. Hallucinations (tolterodine).

G. Review previous continence therapy and its effectiveness:

1. Pelvic floor muscle training (PFMT), first-line therapy with stress incontinence.
2. Bladder training, including timed voiding for urge incontinence. ▶

3. PFMT + bladder training for mixed UI.
4. Weight loss and exercise in obese females.
5. Lifestyle changes, including timed urination, dietary changes, and fluid limitation.
6. Abobotulinumtoxin A injection therapy (BXT).
7. Percutaneous nerve, magnetic, or electrical stimulation.
8. Previous surgical treatments.

H. Review the impact of incontinence on quality of life, including work impairment, sexual dysfunction, activities of daily living, sleep, recreational activity, social interaction, and depression.

I. Assess whether the elderly client has incontinence despite toileting.

J. Review sexual function.

K. Review history and previous treatment for prostate disease.

L. Review other comorbid medical diagnoses such as neurologic disabilities, narrow-angle glaucoma, and diabetes:

1. Do not prescribe a muscarinic medication in a client with narrow-angle glaucoma unless approved by the treating ophthalmologist.
2. Use caution when prescribing antimuscarinic for OAB with a frail client because anticholinergic side effects can result is increased risks that outweigh the benefits.

M. Review the diabetic client's blood glucose averages (polyuria is increased with uncontrolled diabetes).

PHYSICAL EXAMINATION

A. Both sexes:

1. Check temperature (if an infection is suspected), pulse, blood pressure (BP), and respirations.
2. Inspect:
   a. Inspect for evidence of cardiac overload: pedal edema.
3. Auscultate:
   a. Auscultate the lungs for evidence of fluid overload: rales.
4. Palpate:
   a. Palpate the abdomen for masses, fullness over bladder, and tenderness.
5. Neurologic examination:
   a. Assess to determine the presence of sensation of the perineal area:
      i. Ask the client if they feel pressure in the bladder or rectal area when they need to urinate or defecate.
      ii. Ask the client if they can feel when wiping.
      iii. During the physical exam, assess for perineal sensation.
   b. Assess cognitive and functional status, including mobility, transfers, manual dexterity, and ability to toilet in the elderly.
   c. Screen for depression.

B. Female:

1. Inspect:
   a. Assess perineal skin for irritation, thinning, vaginal atrophy, and vaginal discharge.
2. Palpate:
   a. Perform a bimanual pelvic exam for prolapse, masses, or tenderness.
   b. Perform a rectal exam for sphincter tone, masses, and fecal impaction.
   c. Perform a pelvic examination:
      i. Remove the top blade of the speculum and evaluate the vaginal wall support.
      ii. Ask the client to cough to reevaluate the vaginal wall support.
      iii. Ask the client to perform a Kegel to evaluate the pelvic floor muscles.

C. Male:

1. Inspect:
   a. Inspect the glans penis for abnormalities in urethral meatus. (Hypospadias may cause postvoid dribbling.)
   b. Uncircumcised clients should be evaluated for phimosis and balanitis.
2. Palpate:
   a. Perform a rectal exam for sphincter tone, masses, fecal impaction, prostate size, and contour.
   b. Palpate the scrotum to evaluate masses.
   c. Evaluate the presence of an inguinal hernia since straining with a partial urinary obstruction can worsen an inguinal hernia.

DIAGNOSTIC TESTS

A. The history, physical examination, and urinalysis are sufficient to guide initial therapy. Other tests include the following:

1. Urine culture and sensitivity if infection is suspected.
2. Urine cytology if hematuria or pelvic pain is present.
3. Cystometry. See Section II: Procedures, "Cystometry."
4. Postvoid residual (PVR) by catheterization or ultrasound.
   a. A PVR of less than 50 mL is considered adequate emptying, and greater than 200 mL is considered inadequate, suggesting either detrusor weakness or obstruction. Indications for PVR:
      i. Males with mild to moderate lower urinary symptoms.
      ii. Males with OAB (urgency).
      iii. Persons with spinal cord injury or Parkinson disease.
      iv. Persons with prior episodes of urinary retention.
      v. Persons with severe constipation.
5. A serum prostate-specific antigen (PSA) should be considered.
6. Routine urodynamic testing is not recommended.
7. Cystoscopy is not required for incontinence; however, it is indicated for hematuria.

DIFFERENTIAL DIAGNOSES

A. Eight reversible causes of transient incontinence can be remembered by using the mnemonic DIAPPERS:

1. **D**elirium.
2. **I**nfection (urinary).
3. **A**trophic urethritis and vaginitis.
4. **P**harmaceuticals.
5. **P**sychological disorders, especially depression.
6. **E**xcessive urine output.
7. **R**estricted mobility.
8. **S**tool impaction.

PLAN

A. General interventions

1. Functional UI: Therapy is directed at the cause of the condition, such as overdiuresis, inability to go to the toilet, or poor access to toilet facilities.
2. Behavioral interventions should be reviewed.
3. Other alternatives:
   a. Surgical treatment is based on the cause of incontinence. ▶

b. Continence pessaries may benefit females with stress UI.
c. Electrical stimulation may be prescribed to inhibit bladder instability and improve striated and levator contractility and efficiency.
d. Treatment of concomitant constipation is an important step in the treatment of incontinence.
e. Treatment of vulvovaginal atrophy is another important step in the treatment of incontinence. (Refer to "Genitourinary Syndrome of Menopause" in Chapter 17, "Gynecologic Guidelines.")
f. Injection of BTX (BOTOX) is an option for treatment of urge incontinence in selected clients.
g. Transurethral bulking agents injected into the submucosal tissues of the urethra or bladder neck is an option for clients with UI caused by benign prostatic hypertrophy (BPH).
h. Indwelling catheters are not recommended as a management strategy for OAB.

B. Client teaching: *See Client Teaching Guide for this chapter, "Urinary Incontinence: Females."*

1. A bladder diary (Table 15.8) may provide information on usual timing and circumstances of UI.
2. Timed scheduled voiding and/or prompted caregiver-scheduled toileting.
3. *See Client Teaching Guide for this chapter, "Urinary Incontinence: Females,"* for a copy of Table 15.8 to hand out to clients.
4. Bladder training by systematic ability to delay voiding through the use of urge of inhibition.
5. Stress incontinence: Kegel perineal exercises may improve UI by 30% to 90%. Exercises using graduated vaginal cones or weights induce pelvic muscle tone and strength, reducing UI. A pelvic floor physical therapist can train client this technique.
6. Overflow UI: Crede method is used for expressing the bladder, by applying pressure with the hands placed in the suprapubic area after voiding to assist in emptying.
7. Intermittent catheterization is an option frequently utilized after other measures have failed or overflow UI occurs.
8. Weight loss.
9. Dietary changes including elimination of bladder irritants: caffeine, citrus/acidic foods, alcohol, and carbonated drinks.
10. Avoid constipation. (Refer to the section "Constipation" in Chapter 14, "Gastrointestinal Guidelines.")
11. Reduce excessive fluid intake, with no fluid intake 3 to 4 hours before bedtime if nocturia is a problem.

C. Pharmaceutical therapy:

1. Do not prescribe a muscarinic medication with clients when they are currently on other medications with anticholinergic properties.
2. Use anticholinergic and antispasmodic drugs to decrease reflex bladder contractions and increase bladder capacity (contraindicated in uncontrolled narrow-angle glaucoma, urinary retention, gastric retention, and frail clients).
3. The choice of pharmacologic therapy should be based on the client's age, comorbidities, concurrent medications, adverse effect profile, tolerability, ease of use, and cost of the medication.
4. The Food and Drug Administration (FDA) has not approved any medications for stress incontinence.
5. The American College of Physicians (ACP) recommends against *systemic* pharmacologic therapy for stress UI:
a. Oxybutynin chloride (Ditropan): tablets and syrup formulation:
i. Adults: 5 mg orally two to three times per day (maximum 20 mg/d).
ii. A lower starting dose of 2.5 mg two or three times a day is recommended for the frail elderly.
b. Oxybutynin chloride (Ditropan XL): extended-release (ER) tablets:
i. Adults: initially 5 to 10-mg once a day; may increase the dosage weekly in 5-mg increments (maximum 20 mg/d).
c. Oxybutynin chloride (Gelnique):
i. Ten percent topical gel is available in a 1-g gel sachet or a metered-dose pump that dispenses 1 g unit dose (30 metered dose/metered-dose pump).
ii. Apply 1-g gel sachet or one actuation of the metered-dose pump once a day to intact skin abdomen, upper arms/shoulders, or thighs. Application sites should be rotated.
d. Oxybutynin (Anturol) 3% gel:
i. 84 mg/d (three pumps) applied daily to intact skin of the abdomen, upper arms/shoulders, or thigh. Application sites should be rotated.
e. Oxybutynin transdermal (Oxytrol), the first available OTC medication:
i. 3.9 mg/d patch applied twice weekly (every 3–4 days). Application sites should be rotated with each new system to avoid reapplication to the same site within 7 days.
f. Tolterodine tartrate (Detrol):
i. Initial recommended dose is 2 mg BID. The dose may be lowered to 1 mg BID based on individual response and tolerability.
ii. The ER dose of Detrol LA is 2 to 4 mg orally per day.
g. Fesoterodine (Toviaz): ER capsule:
i. The recommended starting dose is 4 mg once a day (maximum 8 mg/d). The daily dose should not exceed 4 mg in the following populations:
1) Clients with severe renal impairment (creatinine clearance [ClCr] <30 mL/min).
2) Clients taking potent CYP3A4 inhibitors, such as ketoconazole, itraconazole, and clarithromycin.
ii. Fesoterodine is not recommended for use in clients with severe hepatic impairment (Child–Pugh C).
h. Darifenacin (Enablex):
i. 7.5 mg oral once a day.
ii. May increase to 15 mg once a day.
i. Solifenacin (VESIcare):
i. Initially 5 mg orally once a day.
ii. May increase to 10 mg daily.

6. Alpha-adrenergic antagonists stimulate urethral smooth muscle contraction.
7. Beta-3 adrenergic agonist: mirabegron (Myrbetriq):
a. Initially 25 mg once a day is effective within 8 weeks.
b. May increase to 50 mg once a day.
c. Daily dose should not exceed 25 mg/d in the following populations: ▶

TABLE 15.8 BLADDER CONTROL DIARY

Your Daily Bladder Diary

This diary will help you and your healthcare team figure out the causes of your bladder-control trouble. The **sample** line shows you how to use the diary. Use this sheet as a master for making copies that you can use as a bladder diary for as many days as you need.

Your name: _____
Date: _____

Time	Drinks		Trips to the bathroom		Accidental leaks		Did you feel a strong urge to go?	What were you doing at the time?
	What kind?	How much?	How many times?	How much urine? (circle one)	How much? (circle one)		Circle one	Sneezing, exercising, having sex, lifting, etc.
Sample	Coffee	8 cups	✓✓	sm med lg	sm med lg		Yes No	Planning
6–7 a.m.				sm med lg	sm med lg		Yes No	
7–8 a.m.				sm med lg	sm med lg		Yes No	
8–9 a.m.				sm med lg	sm med lg		Yes No	
9–10 a.m.				sm med lg	sm med lg		Yes No	
10–11 a.m.				sm med lg	sm med lg		Yes No	
11–12 noon				sm med lg	sm med lg		Yes No	
12–1 p.m.				sm med lg	sm med lg		Yes No	
1–2 p.m.				sm med lg	sm med lg		Yes No	
2–3 p.m.				sm med lg	sm med lg		Yes No	
3–4 p.m.				sm med lg	sm med lg		Yes No	
4–5 p.m.				sm med lg	sm med lg		Yes No	
5–6 p.m.				sm med lg	sm med lg		Yes No	
6–7 p.m.				sm med lg	sm med lg		Yes No	
7–8 p.m.				sm med lg	sm med lg		Yes No	
8–9 p.m.				sm med lg	sm med lg		Yes No	
9–10 p.m.				sm med lg	sm med lg		Yes No	
10–11 p.m.				sm med lg	sm med lg		Yes No	
11–12 midnight				sm med lg	sm med lg		Yes No	
12–1 a.m.				sm med lg	sm med lg		Yes No	
1–2 a.m.				sm med lg	sm med lg		Yes No	
2–3 a.m.				sm med lg	sm med lg		Yes No	
3–4 a.m.				sm med lg	sm med lg		Yes No	
4–5 a.m.				sm med lg	sm med lg		Yes No	
5–6 a.m.				sm med lg	sm med lg		Yes No	

I used ____ pads today. I used ____ diapers today (write number). Questions to ask my healthcare team:

_____

_____

_____

_____

_____

Let's Talk About Bladder Control for Women is a public health awareness campaign conducted by the National Kidney and Urologic Diseases Information Clearinghouse (NKUDIC), an information dissemination service of the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), National Institutes of Health.

Source: Adapted from National Institute of Diabetes and Digestive and Kidney Diseases. (n.d.). Your daily bladder diary. http://www.niddk.nih.gov/-/media/Files/Urologic-Diseases/diary_508.pdf.

i. Clients with severe renal impairment (ClCr <30 mL/min).

ii. Clients with moderate hepatic impairment (Child–Pugh Class B).

d. Mirabegron is not recommended for use in the following populations:

i. Clients with end-stage renal disease (ESRD).

ii. Clients with severe hepatic impairment (Child–Pugh Class C).

8. For postmenopausal clients, vaginal estrogen may restore urethral mucosa; use the same type, dosage, and client selection criteria as with estrogen therapy (ET). Systemic estrogen such as the estrogen patch should not be prescribed for UI.

FOLLOW-UP

A. Follow-up is based on the type and cause of UI.

CONSULTATION/REFERRAL

A. When to consult a physician for UI depends on the type and cause of UI. UI is a problem that can be successfully treated.

B. Refer to a specialist for incontinence with abdominal and/or pelvic pain or hematuria in the absence of a UTI, or when surgical treatment is desired.

C. Refer for urodynamic testing, which is considered the gold standard.

1. Testing requires special equipment and training.

D. Refer to a gynecologist or urology clinic experienced in fitting a continence pessary.

E. Refer to a urologist for hematuria and/or risk factors of bladder cancer.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Stress incontinence is frequently associated with pregnancy and is treated with pelvic exercises.
2. Postpartum: Refer to a pelvic floor physical therapist if UI does not resolve in the first few weeks postpartum and may take up to 6 months to resolve completely.

B. Geriatrics:

1. Geriatric syndrome assessments:
   a. Falls: Incontinence in the elderly is a risk factor for falls. Many falls occur en route to the bathroom, especially at night.
      i. Avoid consumption of diuretics in the hours prior to bedtime (caffeine, alcohol, diuretic medications). Avoid fluids 1 to 2 hours before bedtime.
      ii. Safety measures to reduce falls in the home include removing small area carpets, keeping night lights along the path to the bathroom, and installing a toilet seat with grab bars or nearby grab bars.
      iii. Durable medical equipment such as a bedside commode or a urinal should be considered.
   b. Depression: Incontinence increases social isolation and depression.
   c. Constipation: Fecal impaction is a risk factor for UI. Constipation is a common side effect of anticholinergic and antispasmodic drugs.
   d. Functional incontinence is common in older adults with arthritis or Parkinson or Alzheimer disease. Clients are unable to hold their urine until they reach the bathroom and undress. A toileting program usually assists in this situation. UI is exacerbated with medications such as diuretics. Timing diuretics at least 4 hours prior to bedtime or prior to situations in which a bathroom would not be easily accessible can help, as can a toileting program.
   e. Clients who use pads or adult diapers should be informed of risks associated with infrequent changing. Because of the high cost of absorbent pads and undergarments, infrequent changing is common and contact dermatitis and candidal rashes may develop. If these occur, clients should be encouraged to change pads/garments more frequently, and avoid wearing absorbent garments overnight and instead should use mattress/bedding pads.
2. Pharmacologic caution in geriatrics:
   a. Antimuscarinic medications may increase confusion and should be avoided in frail older adults or adults with dementia and are contraindicated in people with narrow angle-closure glaucoma. Ask about the effectiveness of the medications versus the appearance of side effects such as constipation, dry eye/mouth, blurred near-vision, tachycardia, drowsiness, or reduced cognitive function. Carefully consider benefits versus risks in all older adults.
   b. First-generation alpha-1 blockers are linked with falls in older adults due to orthostatic hypotension. Educate older males prescribed alpha-1 blockers for BPH to use caution, getting up slowly from seated or lying positions. Avoid first-generation alpha-1 blockers (Hytrin, Cardura) in older males with a history of syncope.
3. Beers list caution: Avoid use of desmopressin for nocturia or nocturnal polyuria: There is high risk for hyponatremia in older adults. Desmopressin is a potentially inappropriate medication to use in the elderly; safer alternative treatments are available.

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URINARY RETENTION

DEFINITION

A. Urinary retention is the inability to empty the bladder completely. It can be acute or chronic, with symptoms such as total inability to urinate or simply inability to empty the bladder completely.

B. Acute urinary retention can cause great discomfort or pain; it is sudden in onset and involves inability to urinate at all even when the bladder is full. It is the most common urologic emergency in males. The duration may be short, but it ▶

can also be potentially life-threatening, requiring immediate emergency intervention.

C. Chronic urinary retention presents with the inability to empty the bladder and is long term. Most of the time, chronic urinary retention is diagnosed with other associated genitourinary (GU) conditions such as urinary tract infection (UTI), urinary incontinence (UI), and overactive bladder (OAB).

INCIDENCE

A. Urinary retention occurs less frequently in females than in males.

B. The overall incidence in males ages 40 to 83 years old is less than 7 per 1,000.

C. At age 70, the rate increases to 100 per 1,000.

D. By the age of 80 years, the incidence is up to 300 per 1,000.

PATHOGENESIS

A. Urinary retention can be caused by three different factors:

1. Detrusor underactivity (DU) is defined by the International Continence Society (ICS) as a contraction of reduced strength and/or duration resulting in prolonged or incomplete emptying of the bladder. Both neurogenic and myogenic factors are involved in DU and may be influenced by pharmacotherapy or by primary disorder inhibiting the detrusor and altering urethral sphincter relaxation.

2. Bladder outlet obstruction (BOO) caused by tumors (GU, gastrointestinal [GI], or intestinal), bladder neck stenosis, calculi, structural malformation, or previous surgical interventions are classified as mechanical–anatomic. Little is known of the functional (nonmechanical–nonanatomic) cause but may include dyssynergia of the bladder neck and sphincter detrusor or dysfunction of the ureteral sphincter.

3. Combination of these mentioned.

PREDISPOSING FACTORS

A. There are multiple factors that could cause urinary retention for both males and females, including the following:

1. Neurologic:
- a. Peripheral neuropathy:
  - i. Diabetes mellitus (DM).
  - ii. Infections.
  - iii. Guillain–Barré syndrome.
  - iv. Radiations and postsurgical interventions of the pelvis.
- b. Central nervous system (CNS):
  - i. Costovertebral angle (CVA).
  - ii. Parkinson.
  - iii. Hydrocephalus: normal pressure.
  - iv. Multiple sclerosis (MS).
  - v. Shy–Drager syndrome.
- c. Spinal cord:
  - i. Mass or trauma.
  - ii. Cauda equina syndrome.
  - iii. Spinal dysraphism.

2. Ureteral strictures.

3. Bladder calculi.

4. Medications:
- a. Antihistamines.
- b. Anticholinergics/antispasmodics for bowel and bladder.
- c. Tricyclic antidepressants.
- d. Decongestants.
- e. Nifedipine.
- f. Antiseizure medications (AEDs).
- g. Nonsteroidal anti-inflammatory drugs (NSAIDs).
- h. Opioids.

5. Constipation/fecal impaction.

6. Low nocturnal bladder capacity.

7. Combination of the nocturnal polyuria and low nocturnal bladder capacity.

8. OAB.

9. Ureteral disruption secondary to trauma.

10. Obstructions for males:
- a. Benign prostatic hypertrophy (BPH): most common.
- b. Phimosis.
- c. Cancer of the prostate.
- d. Stenosis of the penile meatal.
- e. UTIs:
  - i. Acute prostatitis.
  - ii. Abscess.
  - iii. Balanitis.

11. Obstructions for females:
- a. Uterine: prolapse, cystocele/rectocele.
- b. Uterine fibroid.
- c. Ovarian cyst.
- d. Ureteral sphincter dysfunction.
- e. Pelvic malignancy.
- f. Infections:
  - i. Genital herpes (local inflammation as well as sacral nerve involvement).
  - ii. Varicella zoster.
  - iii. Vulvovaginitis.
- g. Inflammations or dermatitis.

12. Abdominal mass.

13. Diabetes especially with peripheral neuropathy.

14. Postoperative urinary retention.

15. Postpartum urinary retention.

COMMON COMPLAINTS

A. Acute symptoms:
- **1.** Inability to urinate.
- **2.** Dysuria with pain and urgency.

B. Dysuria with poor or weak stream.

C. Urinary frequency.

D. Urinary urgency.

E. Geriatrics:
- **1.** May not present with classic symptoms.
- **2.** Incontinence.
- **3.** Mental confusion.
- **4.** History of falls.

OTHER SIGNS AND SYMPTOMS

A. Asymptomatic.

B. Bladder spasms.

C. Suprapubic pain or suprapubic discomfort.

D. Depression.

E. Morning fatigue.

F. Frequent wakefulness.

G. Insomnia.

POTENTIAL COMPLICATIONS

A. UTIs.

B. Chronic kidney disease (CKD).

C. Poor quality of life.

SUBJECTIVE DATA

A. Review the onset, course, and duration of symptoms. ▶

B. Review all medications, over-the-counter (OTC) medicines, and herbal products with notation of the medication/classes that cause urinary retention (see the section "Predisposing Factors").
C. Does the client have a fever and chills or back or flank pain (unilateral or bilateral)?
D. Has the client had any recent surgical procedures?
E. Is there any history of UTIs? How often and how were they treated? Were any tests performed in a workup by a urologist?
F. Males: Review the strength of the urinary flow, dribbling, and hesitancy.
G. Females:
1. Postmenopausal: Review whether they have a known prolapse and/or vaginal atrophy.
2. Do they use any systemic or local estrogen medications?
3. Is client postpartum?
H. Is there any history of other medical diseases noted in predisposing factors?
I. Review for the presence of neurologic disorders, including spinal cord injury or MS.
J. Review if there are other active problems or a history of genital problems such as herpes.

PHYSICAL EXAMINATION

A. Both sexes:
1. Check temperature (if an infection is suspected), pulse, blood pressure (BP), and respirations.
2. Inspect for evidence of cardiac overload: pedal edema.
3. Auscultate the lungs for evidence of fluid overload: rales.
4. Perform neurologic examination to determine the presence of sensation and neurogenic bladder. Check anal reflex and sphincter tone.
5. Palpate the abdomen, suprapubic, and back for masses, fullness over bladder, inguinal nodes, and CVA tenderness (bladder is percussible with 150 mL and palpable <200 mL of urine).
6. Assess cognitive and functional status, including mobility, transfers, manual dexterity, and ability to toilet in the elderly.
7. Screen for depression.
8. Assess for falls and possible healing fractures.
9. Evaluate for obesity.
B. Female:
1. Inspect perineal skin for irritation, thinning, vaginal atrophy, herpetic lesion, or vaginal discharge.
2. Remove the top blade of the speculum and evaluate the vaginal wall support.
3. Ask the client to cough to reevaluate the vaginal wall support.
4. Palpate:
a. Perform a bimanual pelvic exam for prolapse, masses, or tenderness.
b. Perform a rectal exam for sphincter tone, masses, and fecal impaction.
C. Male:
1. Inspect:
a. Inspect the glans of the penis for abnormalities in urethral meatus. (Hypospadias may cause postvoid dribbling.)
b. Uncircumcised clients should be evaluated for phimosis and balanitis.
c. Inspect for active herpetic lesions.
2. Palpate:
a. Perform a rectal exam for sphincter tone, masses, fecal impaction, prostate size, and contour.
b. Palpate the scrotum to evaluate masses.
c. Evaluate the presence of an inguinal hernia since straining with a partial urinary obstruction can worsen an inguinal hernia.

DIAGNOSTIC TESTS

A. The history, physical examination, and urinalysis may be sufficient to guide initial therapy and to rule out other causative factors. Other tests include the following:
1. Urine culture and sensitivity if infection is suspected.
2. Urine cytology if hematuria or pelvic pain is present.
3. A prostate-specific antigen (PSA) should be considered; however, it is expected to be elevated with acute urinary retention.
4. Cystoscopy/ureteroscopy.
5. Urodynamic testing is strongly recommended.
6. Postvoid residual (PVR) measurement.
7. CT scans.
8. Electromyography.
9. Depression screen.
10. Neurologic evaluation may be needed to include, but is not limited to, CT studies.
11. Chemistry study with hemoglobin (Hgb) A1C.
12. Nocturnal pulse oximetry study.
13. Sleep study may be indicated, especially in those positive for nocturnal hypoxemia.
14. Ultrasound of the kidneys with Doppler.

DIFFERENTIAL DIAGNOSES

A. Urinary retention.
B. Urinary obstruction:
1. Bladder calculi.
2. Ureteral stricture.
C. Medication(s) side effect.
D. Neurologic etiology.
E. Male-related etiologies as noted in the "Predisposing Factors."
F. Female-related etiologies as noted in the "Predisposing Factors."
G. Fecal impaction.
H. Abdominal mass.
I. CKD.

PLAN

A. General interventions
1. Initial management of acute urinary retention is prompt bladder decompression with a urethral catheter.
2. Urethral catheterization is contraindicated in clients who have had recent urologic surgery (radical prostatectomy or urethral reconstruction).
3. A suprapubic catheter may be necessary if a urethral catheter cannot be passed due to obstruction.
B. Client teaching:
1. Behavior training includes the following:
a. Doing a "double void" by emptying the bladder, then waiting and trying again to empty the bladder.
b. Biofeedback.
c. Bladder and pelvic muscle retraining.
2. Train for self-catheterization if indicated. ▶

3. Keep a 24-hour voiding diary. *See Client Teaching Guides for this chapter "Urinary Incontinence: Females," and "Urinary Retention."*

4. Avoid constipation/fecal impaction.

5. Evaluate the need for an indwelling catheter (e.g., postsurgical).

6. Implantable neuromodulation therapy to the sacral nerves (InterStim) has been helpful.

C. Pharmaceutical therapy:

1. Review all medications and herbals to evaluate what may be discontinued.

2. Medications may need to be started to treat an enlarged prostate:

a. Dutasteride (Avodart): 0.5 mg daily.
b. Finasteride (Proscar): 5 mg daily.

3. Evaluate the need to prescribe medications to relax the bladder outlet and prostate to help relieve blockage:

a. Doxazosin (Cardura): 1 to 4 mg daily.
b. Silodosin (Rapaflo): 8 mg daily (give with food).
c. Tadalafil (Cialis): 5 mg daily.
d. Tamsulosin (Flomax): 0.4 mg daily.
e. Terazosin (Hytrin): 1 to 10 mg daily at bedtime.

FOLLOW-UP

A. Follow-up is determined by client's needs, severity of symptoms, and whether complications are present. Adequate time should be allowed for treatments involving nonpharmacologic and pharmacologic approaches.

EMERGENT ISSUES/INSTRUCTIONS

A. Acute urinary retention is considered a medical emergency that should be managed by immediate catheterization. Instruct the client to go to the ED immediately.

CONSULTATION/REFERRAL

A. Refer to a urologist for consideration of:

1. Treatment with a urethral dilation, urethral stents.

2. Surgical intervention.

3. Suprapubic catheter.

4. Urogynecologist: for females with structural problems and pregnancy.

C. Neurology.

D. Endocrinology.

INDIVIDUAL CONSIDERATIONS

A. Adults/Geriatrics:

1. Pharmacologic caution in geriatrics:

a. Review all medications to identify medications that can lead to urinary retention, including antidepressants, antipsychotics, antiarrhythmics, antiparkinsonian agents, and antihistamines.

b. First-generation alpha-1 blockers are linked with falls in older adults due to orthostatic hypotension. Educate older males to use caution, getting up slowly from seated or lying positions. Avoid first-generation alpha-1 blockers (Hytrin, Cardura) in older males with a history of syncope.

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URINARY TRACT INFECTION (ACUTE CYSTITIS)

DEFINITION

A. Urinary tract infection (UTI) is an infection of the urinary bladder. UTI is defined as the presence of at least 100,000 organisms per milliliter of urine in an asymptomatic client or more than 100 organisms per milliliter of urine with accompanying pyuria (>7 white blood cells [WBC]/mL) in a symptomatic client. Asymptomatic bacteriuria (ASB) when left untreated is a risk factor for acute cystitis (40%) and pyelonephritis in 25% to 30% of pregnant persons.

B. UTIs can be divided anatomically into upper and lower tract (cystitis) infections. For a discussion of upper tract infection, see the section "Pyelonephritis" in this chapter.

C. UTIs may be considered uncomplicated or complicated.

1. An uncomplicated UTI is noted in a healthy person with a normal urinary tract system and may be treated with oral antibiotics.

2. A UTI noted in a person with a structural or functional urinary tract system or in a person who is immunocompromised is considered complicated. It may require parenteral therapy until afebrile.

INCIDENCE

A. Incidence depends on age and sex. The prevalence of UTI in males varies according to age.

1. Young males aged 15 to 50 years rarely develop a UTI.

2. The incidence of UTI in geriatric males may be as high as in geriatric females (up to 15%).

B. Over 50% of females will have one UTI in their lifetime.

1. Prevalence for females increases by 1% per decade and 2% to 4% throughout childbearing years.

2. The incidence of UTIs in pregnancy ranges from 4 % to 7%. In pregnancy, the increased incidence is related to both hormonal influence and anatomic changes that increase the risk of urinary stasis and vesicoureteral reflux.

3. By age 30, approximately 25% of females have experienced symptoms of a UTI.

PATHOGENESIS

A. Bacteria ascend from the perineum through the urethra. The greater susceptibility of younger females and pediatric females is related to a shorter urethra. In older females, it is related to estrogen-mediated dilation of the urethra.

B. The normal male urinary tract has many natural defenses to infection. The greater susceptibility of elderly males is related to problems with the prostate and other urologic diseases and can be linked to the instrumentation required for therapy.

C. Gram-negative bacilli are the most common pathogens; 80% to 90% of cases are related to coliform bacteria (*Escherichia coli*). It originates from fecal floras that colonize the periurethral area.

D. Other gram-negative bacteria include *Klebsiella pneumonia* or *Proteus mirabilis. Staphylococcus saprophyticus* (gram-positive coccus) accounts for about 10% to 15% of UTIs.

E. Other pathogens include *Enterobacter, Pseudomonas, Enterococci*, and *Staphylococci.*

F. The incubation period depends on the pathogen.

PREDISPOSING FACTORS

A. Females (until elderly, then equal frequency in males and females).
B. Pregnancy.
C. Poor hygiene.
D. Trauma.
E. Instrumentation.
F. Sexual intercourse.
G. Oral contraceptive or diaphragm use.
H. Females diagnosed with diabetes (there is no increased risk for diabetic males).
I. Anomalies of the genitourinary (GU) tract.
J. Neurologic factors.
K. Vesicoureteral reflux.
L. Obstruction: stones.
M. Foreign bodies.
N. Bubble baths and hot tubs.
O. Douching.
P. Anal intercourse.
Q. HIV.
R. Uncircumcised penis.
S. Catheterization.
T. Nosocomial infection.
U. Phimosis.

COMMON COMPLAINTS

A. Burning on urination.
B. Frequency.
C. Cloudy or bloody urine.
D. Urgency.
E. Geriatrics:
  1. May not present with classic symptoms.
  2. Fever.
  3. Incontinence.
  4. Mental confusion.

OTHER SIGNS AND SYMPTOMS

A. Asymptomatic.
B. Frequency, dysuria, bladder spasms, suprapubic discomfort, urgency, and nocturia.
C. Suprapubic pain.
D. Fever.
E. Costovertebral angle (CVA) tenderness.
F. Hematuria.

SUBJECTIVE DATA

A. Review the onset, course, and duration of symptoms.
B. Does the client have a fever and chills or back or flank pain (unilateral or bilateral)?
C. Are there any other genital problems such as herpes lesions or vaginal discharge?
D. Review the associated factors: sexual intercourse (specifically review for anal intercourse), douching, or bubble bath.
E. Ask female clients if they use appropriate hygiene practices after urination and bowel movements (BMs):
  1. Wiping from front to back.
  2. Frequent changes of hygienic products.
  3. Handwashing.
F. Is the client pregnant? If not, what type of birth control does client use?
G. Is there any history of previous UTIs? How often and how were they treated? Were any tests performed in a workup by a urologist?
H. How much liquid or water does the client drink every day? Note the amount of caffeine.
I. In older males, review the strength of the urinary flow, dribbling, hesitancy, and so forth.
J. In the postmenopausal client, review whether they have a known prolapse and/or vulvovaginal atrophy. Do they use any systemic or local estrogen medications?
K. Is there any history of other medical diseases, including diabetes or sickle cell disease?
L. Review for the presence of neurologic disorders, including spinal cord injury or multiple sclerosis (MS).
M. Does the client require self-catheterization?

PHYSICAL EXAMINATION

A. Check temperature, blood pressure (BP), pulse, and respirations. **The absence of a fever does not exclude the presence of an infectious process.**
B. Inspect:
  1. General observation of general appearance for discomfort before and during examination.
C. Auscultate:
  1. Heart and all lung fields.
D. Palpate:
  1. Palpate the abdomen: kidneys, masses; assess for suprapubic tenderness.
  2. Palpate the back; note CVA tenderness.
  3. Check for inguinal lymph node enlargement.
  4. Palpate the suprapubic area.
E. Percuss:
  1. Percuss over the bladder and the CVA area for tenderness.
F. Females:
  1. Pelvic examination:
    a. Inspect external genitalia for lesions, Bartholin gland cysts, irritation, and discharge.
    b. Milk urethra for discharge.
  2. Assess rectal area.
  3. Speculum exam: Evaluate vaginal vault for discharge, cervicitis, and inflammation; evaluate for atrophic vaginal changes and torn tissue.
  4. Bimanual exam: Check for cervical motion, tenderness, and masses.
G. Males:
  1. Inspect the penis/urinary meatus for phimosis, lesions, signs of inflammation, and discharge. Retract the foreskin (if present) and assess for hygiene and smegma.
  2. Palpate the testes and epididymides for inflammation, tenderness, and masses.
  3. Rectal examination is mandatory in males. Check for swollen and tender prostate. In clients with suspected acute bacterial prostatitis, palpation should be very gentle due to the potential for bacteremia.

DIAGNOSTIC TESTS

A. The diagnosis of UTI can often be made based on a focused history and the presenting symptoms.
  1. Urinalysis: Clean-catch urinalysis may be performed. However, catheterization or suprapubic aspiration may need to be obtained for elderly, obese, microscopic hematuria, or for functionally impaired clients. Catheterization should be reserved for clients with an obstruction or for those who cannot cooperate or collect a clean-catch urine specimen. ▶

2. Urinalysis dipstick findings:
   a. Appearance: should be clear; cloudy urine may indicate the presence of pyuria, pus, blood, cells, phosphate, or lymph fluid.
   b. Odor: usually faint aromatic odor; ammonia odor indicates *Proteus*, which is related to food changes; offensive odor indicates bacterial infection.
   c. pH: normal is around 6 (acid); may normally vary from 4.6 to 7.5; greater than 7.5 may indicate infection.
   d. Specific gravity: reflects the kidney's ability to concentrate urine and the body's hydration or dehydration status; normal is 1.005 to 1.025.
   e. Color: shows concentration; usually yellow or amber.
      i. Straw color: dilute urine.
      ii. Dark color: concentrated (dehydrated).
      iii. Red or red-brown to blood: transfusion reaction, drugs, and bleeding lesions.
      iv. Yellow brown: bile duct disease, jaundice.
      v. Dark brown or black: melanoma or leukemia.
   f. Positive for leukocyte esterase and positive for nitrites. *A negative urine dipstick does not rule out an infection.*
3. Microscopic exam of urine findings: WBCs greater than 2 to 5; WBC/high-power microscope field (HPF); bacteria; positive Gram stain for cocci or rods, yeast, and blood indicate infection.
4. Urine culture and sensitivity:
   a. Positive culture standard 105 colony-forming units: symptomatic females 102; symptomatic males 103.
   b. Screening for asymptomatic bacteria is recommended for clients in pregnancy, for elderly males with documented prostatic or urologic abnormalities, for clients with a recent catheterization, and for clients with known stones or documented structural abnormalities.
5. Culture for sexually transmitted infections (STIs) if suspected.
6. Wet prep for females, if indicated.
7. Imaging:
   a. Ultrasound.
   b. Conventional voiding cystourethrography.
   c. Urodynamic evaluation.

DIFFERENTIAL DIAGNOSES

A. UTI: Watch for systemic symptoms of pyelonephritis.
B. Vaginal or pelvic infection.
C. Prostatitis or epididymitis: tender, enlarged prostate; tender testicle or scrotum.
D. Bladder tumor.
E. Interstitial cystitis (IC).
F. Urinary calculi.
G. Benign prostatic hypertrophy (BPH): changes in urinary stream and nocturia.
H. Overactive bladder (OAB)/urge incontinence.
I. Pelvic organ prolapse.
J. Irritant urethritis.
K. Consider the possibility that chronic, asymptomatic infections are a potential source of disseminated infection, such as endocarditis. This is particularly likely in the male client with prostate disease and infection requiring instrumentation.

PLAN

A. General interventions:
1. Treatment of acute cystitis is aimed at identifying the underlying cause and initiating treatment as soon as possible.
2. If antibiotic therapy is initiated, stress the importance of taking all medications as directed, even if symptoms improve before the end of treatment.

B. Client teaching: *See Client Teaching Guide for this chapter, "Urinary Tract Infection."*
C. Dietary management:
1. Instruct the client to increase fluids and drink at least one large glass of liquid every hour.
2. Instruct the client to avoid foods that irritate the bladder: caffeine, alcohol, tomatoes, citrus, and spicy foods.
3. Encourage the client to drink cranberry juice to help fight bladder infections. If the client dislikes the taste of plain cranberry juice, it can be mixed 1:1 with another juice, such as grape juice.

D. Pharmaceutical therapy:
1. Antibiotics: 3-day course may be efficacious and is less expensive than the traditional 7- to 10-day course of therapy for uncomplicated infections.
2. The antibiotic of choice depends on the specific bacteria found upon culture. Empiric antimicrobial therapy should cover all likely pathogens.
3. First-line therapy:
   a. Nitrofurantoin monohydrate/macrocrystals (Macrobid) 100 mg orally BID for 5 days.
   OR
   b. Trimethoprim-sulfamethoxazole (TMP-SMX) (Septra, Bactrim DS) 160 mg/800 mg, one tablet BID for 3 days (use when bacterial resistance is less than 20% and the client has no allergy):
      i. Prescribe the above-mentioned dosage for 7 to 10 days to clients who are male, are elderly, or have recurrent UTI, or for complicated cases.
4. Second-line therapy:
   a. Ciprofloxacin (Cipro) 250 mg orally BID for 3 days.
   OR
   b. Ciprofloxacin extended-release (Cipro XR) 500 mg orally BID for 3 days. OR
   c. Levofloxacin (Levaquin) 250 mg orally BID for 3 days.
5. Alternative therapy:
   a. Amoxicillin-clavulanate (Augmentin) 875 mg/125 mg orally BID for 7 days. OR
   b. Amoxicillin-clavulanate (Augmentin) 500 mg/125 mg orally TID for 7 days.
6. Urinary analgesic if needed:
   a. Phenazopyridine HCL (Pyridium): 100 to 200 mg TID for 1 to 2 days only when given with an antibiotic. Educate the client that this drug turns urine orange.
   b. Multiple Uro Blue medications are available; take one orally QID. Educate the client that this drug turns the urine blue/green. Methenamine, sodium acid phosphate, and methylene blue (methylthioninium chloride) help to prevent the growth of bacteria in the urine.

Methylene blue, a redox dye in clinical use, has been reported to precipitate serotonin toxicity (ST) in clients using selective serotonin reuptake inhibitor (SSRI) antidepressants. ST, formerly referred to as serotonin syndrome, is one of very few drug–drug interactions involving usual therapeutic doses of commonly used drugs that are capable of developing rapidly and causing death within 24 hours. A mixture of therapeutic doses of any drug with significant monoamine oxidase inhibitor (MAOI) properties with another drug that has potency as a SSRI produces a high risk of precipitating this rapidly worsening interaction. The typical clinical features of ST are (a) neuromuscular hyperactivity: ▶

tremor, clonus, myoclonus, and hyperreflexia, and, in the advanced stage, pyramidal rigidity; (b) autonomic hyperactivity: diaphoresis, fever, tachycardia, tachypnea, and mydriasis; and (c) altered mental status: agitation, excitement, with confusion in the advanced stage.

7. Drugs of choice for UTI or pyelonephritis in pregnancy:
a. Nitrofurantoin (Macrobid) 100 mg: one tablet orally Q12H for 5 days.
b. Amoxicillin: 500 mg orally Q12H for 3 to 7 days.
c. Amoxicillin-clavulanate (Augmentin): 500 mg orally Q12H for 3 to 7 days.
d. Cephalexin (Keflex): 500 mg orally Q12H for 3 to 7 days.
e. Cefpodoxime (Vantin) 100 mg BID for 5 to 7 days.
OR
f. Fosfomycin (Monurol) 3 g orally in a single dose with 3 to 4 oz of water.
g. If dysuria is present: phenazopyridine HCL (Pyridium) 200 mg orally TID for 2 days when given with an antibiotic.

8. Antibiotics that should not be utilized in pregnancy include the following:
a. **Fluoroquinolones (Food and Drug Administration [FDA] class C) are contraindicated during pregnancy due to auditory and vestibular toxicity in the fetus.**
b. **Tetracyclines should not be used in pregnancy (FDA class D) or in children due to tooth staining.**
c. **Nitrofurantoin is contraindicated in pregnant clients at term, during labor, and during delivery.**

9. Consider prophylactic therapy for clients with chronic conditions/recurrent infections:
a. Low-dose antibiotics daily for 3 to 6 months.
b. Self-start antibiotics.
c. Postcoital antibiotics.

10. Quinolones, cephalosporins, and macrolides should be reserved for complicated or resistant infections.

11. Vaginal estrogen should be considered in postmenopausal females with urogenital atrophic changes.

FOLLOW-UP

A. Routine posttreatment urinalysis/culture is not indicated in asymptomatic clients.

B. Have the client return if symptoms do not resolve at the end of treatment.

C. Have the client return if the symptoms reoccur within 2 weeks of treatment for a urine culture. Retreatment with a 7-day course of antibiotics using a different agent should be considered.

CONSULTATION/REFERRAL

A. Young males do not have UTIs very often; a urologic workup may be needed if etiology such as an STI cannot be determined.

B. Clients with bacteriuria are also more likely to have identifiable abnormalities on an intravenous pyelography (IVP), including small kidneys, delayed excretion, caliceal dilation and blunting, ureteral reflux, stones, and obstructive lesions.

C. Consultation with a urologist is essential in all forms of prostatitis or in all but the most clear-cut cases of acute scrotum.

INDIVIDUAL CONSIDERATIONS

A. Females:

1. Recurrent infections are common in females.

2. Repeat urine cultures and sensitivity after an antibiotic course is complete (approximately 2–4 weeks after therapy is completed).

3. If the client is perimenopausal and has two or fewer UTIs a year, consider client-initiated therapy to start when symptomatic. Consider topical estradiol cream for atrophic vaginitis. If the client has three UTIs a year, prescribe a prophylactic single-dose regimen after intercourse. If infections are not related to intercourse, consider urine cultures every 2 months, with extended antibiotic therapy.

B. Pregnancy:

1. The incidence of pyelonephritis in pregnancy is 1% to 2%. Most cases develop as a consequence of undiagnosed or inadequately treated lower UTI.
a. In the presumptive diagnosis of pyelonephritis in pregnancy, ultrasound of the kidneys and urinary tract should be considered.

2. Approximately 75% to 80% of pyelonephritis cases occur on the right side, with a 10% to 15% incidence on the left side. A small percentage of cases are bilateral.

3. Group B strep (GBS) colonization has important implications during pregnancy, contributing to maternal pyelonephritis and preterm birth. Intrapartum transmission may lead to neonatal GBS infection:
a. Clients with documented group B streptococcal bacteriuria in the current pregnancy should be treated at the time of labor or rupture of membranes with appropriate intravenous antibiotics for prevention of early onset neonatal GBS disease.

4. Current guidelines recommend universal vaginal and rectal screening in all pregnant individuals at 35 to 37 weeks' gestation rather than treatment based on risk factors.

5. A urine culture screening is recommended for all pregnant individuals at their first prenatal visit.

6. Suppressive antibiotic therapy should be instituted in pregnant individuals who develop acute cystitis, recurrent or persistent ASB, or pyelonephritis.

7. Clients with sickle cell hemoglobinopathies are at increased risk for UTI and should be screened more aggressively, possibly benefiting from antibiotic prophylaxis.

8. Antibiotics that should not be used during pregnancy include:
a. **Tetracyclines (adverse effects on fetal teeth/bones and congenital defects).**
b. **Quinolones (congenital defects).**
c. **Trimethoprim in the first trimester (facial defects and cardiac abnormalities).**
d. **Sulfonamides in the last trimester (kernicterus).**
e. **Aminoglycosides (permanent ototoxicity in the fetus).**

C. Geriatrics:

Geriatric clients may not have classic symptoms. Consider UTI if the client presents with increased urinary incontinence (UI), fever, and mental confusion.

1. ASB should not be treated in older adults. Both males and females are more likely to have nonspecific signs and symptoms of UTI, but culture results may show fewer than 100,000 organisms related to noninfectious/colonization. Overdiagnosis of UTI is common; research shows greater than 40% of clients hospitalized for UTI did not meet clinical criteria. ▶

2. Empiric treatment for UTI should be avoided in clients unless at least two significant clinical indicators of UTI are present. These can include fever or chills, new or increased dysuria, frequency or urgency, worsening in mental or functional status, new or increased UI, or new flank or suprapubic tenderness. If empiric therapy is considered, it should be changed based on culture and sensitivity, client factors (renal function, allergies), and treatment cost.

3. Prophylaxis is generally not recommended in older adults because it leads to antibiotic resistance.

4. Older females may present with atypical in addition to typical signs and symptoms. These may include lower abdominal pain, back pain, chills, and constipation. Females with atrophic vaginitis, vaginal atrophy from estrogen depletion, are at increased risk for UTI. Use of local or topical estrogens may reduce the risk, but it may take 2 to 3 months for the protective effects of topical/local estrogen therapy to take effect.

5. Frail older adults or nursing home residents may present with nonspecific symptoms including anorexia, confusion, and a decline in functional status. Fever may be absent or diminished in those who are frail. In the setting of atypical symptoms, further testing, such as blood tests, may be required to differentiate a symptomatic UTI from other infections or medical conditions.

6. Beers list caution:

a. Nitrofurantoin should be avoided in older adults with creatinine clearance (ClCr) of less than 30 mL/min or for long-term suppression of bacteria.

b. Nitrofurantoin has the potential for pulmonary toxicity, hepatotoxicity, and peripheral neuropathy, especially with long-term use. (See Appendix C, Table C.1.)

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VARICOCELE

DEFINITION

A. Varicocele is engorgement of the internal spermatic veins above the testes. This vascular abnormality is a cause of decreased testicular function. Some varicoceles are easy to identify and may be surgically corrected. The presence of a varicocele does not mean that surgical correction is a necessity.

INCIDENCE

A. Varicocele may occur in 15% to 20% of normal males; 80% to 90% of cases occur on the left side. Up to 35% to 40% of males with a palpable left-sided varicocele may actually have bilateral varicoceles that are identified upon physical examination.

B. Right-sided varicocele is uncommon and can indicate retroperitoneal malignancy. Varicocele is the leading known cause of male infertility (40%). Decreased sperm counts, infertility, and testicular atrophy occur in 65% to 75% of varicocele cases. There is no correlation between size of the varicocele and the degree of infertility.

PATHOGENESIS

A. The exact pathophysiologic mechanisms for varicoceles are not fully identified. Varicoceles may be caused by valvular incompetence or elevated hydrostatic pressure in the spermatic veins. Testicular temperature elevation also appears to play a role in varicocele-induced dysfunction. **New varicoceles in older males may be secondary to renal tumors.**

B. Varicoceles are the most common cause of male infertility.

PREDISPOSING FACTORS

A. Varicoceles generally manifest at the time of puberty.

B. Tobacco use.

C. Mutations in the gene expressing glutathione S-transferase Mu 1.

COMMON COMPLAINTS

A. Asymptomatic.

B. Infertility.

C. Pain or discomfort in the scrotum.

OTHER SIGNS AND SYMPTOMS

A. Pain or aching and heaviness in the scrotum.

B. Feels like "worms"; scrotum may have bluish discoloration.

SUBJECTIVE DATA

A. Note the onset, course, and duration of symptoms. When was the varicocele first noted?

B. Has the scrotum enlarged? If there is enlargement, over what time span? Does it collapse with lying or sitting down?

C. Is there any pain or discomfort?

D. Has there been any history of infertility?

PHYSICAL EXAMINATION

A. Check vital signs and temperature as indicated.

B. Inspect:

1. The examination should be done when the client is lying or standing in a warm room. Warm temperature promotes relaxation of the scrotum.

2. Examine the general appearance of the penis; note scrotal size, shape, and rugae. Varicocele tends to collapse with the client sitting or supine.

3. Transilluminate the scrotum to visualize the varicocele.

4. Large varicocele can easily be identified by inspection.

C. Palpate:

1. Varicocele classifications:

a. Grade I (small): palpable only on Valsalva maneuver, which increases intra-abdominal pressure and therefore impedes drainage and increases the varicocele size.

b. Grade II (medium): palpable when standing and bearing down (Valsalva maneuver).

c. Grade III (large): visible on inspection alone.

d. Subclinical: not palpable, vein larger than 3 mm on ultrasound; Doppler reflux on Valsalva maneuver.

2. Palpate each side of the scrotum for testicular size, presence of varicocele (Valsalva maneuver performed while the client stands helps to reveal a small varicocele), and absence of vas deferens:

a. A moderate-sized varicocele can be identified by palpation without having the client perform the Valsalva maneuver.

3. Palpate the spermatic cord between the thumb and forefingers while the client performs a Valsalva maneuver:

a. A small varicocele is identified only when the client bears down, increasing the intra-abdominal pressure. ▶

b. Varicocele should significantly diminish in size when the client assumes the supine position.

4. Evaluate whether the varicocele can be reduced while the client is supine.

5. Palpate the abdomen for hernias, masses, and tenderness.

6. Rectal exam: Palpate the prostate and seminal vesicles for tenderness and other signs of infection.

D. Auscultate:

1. Listen over the scrotum to assess bowel sounds to rule out hernia.

DIAGNOSTIC TESTS

A. Scrotal ultrasound with a high-resolution color-flow Doppler is the diagnostic method of choice when clinical exams are equivocal but are not indicated for standard evaluation.

B. Semen analysis times two, if indicated.

C. CT to evaluate retroperitoneal pathology (e.g., renal cell carcinoma) for:

1. Sudden onset of varicocele.

2. Single right-sided varicocele.

3. Any varicocele that is not reducible in the supine position.

DIFFERENTIAL DIAGNOSES

A. Trauma

B. Hernia.

C. Epididymitis.

D. Hydrocele.

E. Testicular tumor: Consider retroperitoneal tumor, especially if presenting symptoms have a sudden onset.

F. Spermatocele.

PLAN

A. General interventions:

1. Urologic consultation for diagnosis and possible surgery. Surgery should be considered when all of the following conditions are met:

a. Palpable varicocele found upon physical examination.

b. Couple with known infertility.

c. Female with normal fertility or potential treatable cause of infertility.

d. Male partner with abnormal semen parameters or abnormal results from sperm function tests.

B. Client teaching: *See Client Teaching Guide for this chapter, "Testicular Self-Examination."*

1. If discomfort is present, an athletic supporter should be tried.

2. If surgery is not indicated, no interventions are needed.

C. Pharmaceutical therapy:

1. None recommended.

FOLLOW-UP

A. After surgery, no follow-up is necessary if the client is taught scrotal self-examination.

B. If no surgery is performed, the client should be taught self-examination and instructed to return for pain or change in size and shape.

C. Adolescents with varicoceles should be followed with annual objective measurements of testis size and/or semen analyses in order to detect the earliest sign of varicocele related testicular injury.

CONSULTATION/REFERRAL

A. Refer the client to a urologist for surgical evaluation. Varicocelectomy is recommended in cases of pain and infertility, and it may be offered in the preadolescent to ensure proper testicular development.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. New onset varicocele in older males may indicate a renal tumor.

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CLIENT TEACHING GUIDE

BENIGN PROSTATIC HYPERPLASIA

PROBLEM Enlargement of the prostate causes bothersome problems with urinating. Symptoms of an enlarged prostate include having to go to the bathroom more often, especially at night; trouble starting or stopping your urine; decreased urine stream; strong and sudden urge to urinate; occasional leaking urine; and feeling that you do not empty.

CAUSE The cause is not known, but it may be due to change in your hormones as you get older.

PREVENTION None.

TREATMENT PLAN Treatment depends on how bad the symptoms are. Medications can help, but an operation to fix the problem may be needed.

A. Empty your bladder on a schedule every 2 to 3 hours to prevent overfilling of the bladder. Do not hold it in. Empty your bladder when you get the first urge.
B. Avoid drinking liquids before bedtime or before driving, traveling, or attending events where finding a bathroom quickly could be difficult.
C. Avoid drinking caffeinated beverages or alcohol after dinner or within 2 hours of bedtime.
D. Double-void to empty your bladder more completely. After each time you urinate, try again right away.
E. In some cases, you may have to insert an intermittent urinary catheter to drain your urine and relieve the symptoms until you can follow up with your provider (see websites listed under "Resources").

Activity: There are no restrictions. You may need to plan schedules with access to bathrooms in mind.

Diet: Avoid spicy foods that irritate the bladder. Caffeine and alcohol act as diuretics and increase your need to urinate.

Medications:

A. Medications that help relieve the blockage may block hormones or relax the muscles that control urination.
B. Do not take over-the-counter remedies such as cold medications, decongestants, antihistamines (for allergies), and diarrhea medicines; they make symptoms worse.
C. Always read labels to check for advice: "Do not take if you have prostate enlargement," for example.
D. Antibiotics are used if there is also an infection in your bladder or prostate.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:

A. You cannot urinate.
B. Your symptoms worsen.
C. You have a fever.
D. Other: _____

Phone: _____

RESOURCES Urinary Catheterization Female: www.nlm.nih.gov/medlineplus/ency/patientinstructions/000144.htm

Urinary Catheterization Male: www.nlm.nih.gov/medlineplus/ency/patientinstructions/000143.htm

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CLIENT TEACHING GUIDE

CHRONIC KIDNEY DISEASE

PROBLEM Chronic kidney disease (CKD) means the kidney is having trouble performing its normal function to maintain health.

FUNCTIONS OF A NORMAL KIDNEY The kidney has several functions:

A. Removal and absorption of fluids to maintain balance.
B. Filtration of blood to remove waste products.
C. Regulation of blood pressure.
D. Hormone regulation for blood production in the bone marrow.
E. Regulation of hormones and minerals.
F. Maintenance of healthy bones.

CAUSES CKD has many causes:

A. Diabetes is the leading cause of CKD in the United States.
B. High blood pressure is the second leading cause of CKD in the United States.
C. Other causes of CKD:
  1. Kidney/bladder infections.
  2. Genetic (inherited) disease: polycystic kidney disease and Alport syndrome.
  3. Congenital diseases.
  4. Autoimmune disease.
  5. Drugs (legal and illegal) and toxic substances.

DETECTION CKD can be detected by:

A. Blood tests: blood urea nitrogen (BUN) and creatinine.
B. Urine tests: protein in urine and creatinine clearance.
C. Other types of tests: ultrasound, immunoassays, CT scan, biopsies, and so forth.
D. The most important test is the blood test for glomerular filtration rate (GFR):
  1. Normal GFR rate: 90 or higher without protein in the urine.
  2. Gets lower with age.
  3. An indicator on the function of the kidney.
  4. Used to determine CKD stages.
  5. Is lower as the kidney function worsens.

STAGES OF CKD

TABLE 1 STAGES OF CHRONIC KIDNEY DISEASE

Stage I (with protein in the urine)	GFR: 90 or higher
Stage II (mild)	60–89
Stage III (moderate)	30–59
Stage IV (severe)	15–29
Stage V (kidney failure)	14 or less

GFR, glomerular filtration rate.

SYMPTOMS OF CKD Symptoms can vary:

A. Usually unnoticed in early stages. A urine test at a routine office visit may be the first time you learn there are problems with your kidneys.
B. More noticeable symptoms appear as CKD worsens (usually around stage III).

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CLIENT TEACHING GUIDE

CHRONIC KIDNEY DISEASE (*CONTINUED*)

C. Those symptoms can include the following:
  1. Nausea.
  2. Fatigue/weakness; no energy.
  3. Decreased or lack of appetite.
  4. Weight loss or rapid weight gain.
  5. Shortness of breath, which may worsen with activity or at rest (when awakening in the morning).
  6. Swelling of the legs and feet and around the eyes.
  7. Cloudy mind or difficulty with concentration.
  8. Muscle cramps.
  9. Frequent nighttime urination.
  10. Difficulty sleeping or staying asleep.

PROBLEMS CAUSED BY CKD CKD can cause multiple problems:

A. Heart, including heart failure and other blood vessels:
  1. High blood pressure.
  2. Cholesterol abnormalities.
  3. Heart attack.
  4. Stroke.
B. Poor nutritional status.
C. Weak and unhealthy bones.
D. Anemia.
E. Water retention (swelling).
F. Progression to kidney failure.

LIFESTYLE CHANGES TO PREVENT PROGRESSION OF CKD Ways you may slow down the progression of CKD and deal with its other effects:

A. Take charge and be proactive:
  1. Be familiar with your healthcare team.
  2. Provide information on beliefs and practices relating to health.
  3. Inform your healthcare team about herbs and other alternative medicine you are using.
  4. Be involved in the treatment plan.
B. Diet:
  1. Meet with a registered dietitian.
  2. Read food labels.
  3. Eat well-balanced meals.
  4. Some dietary restrictions may be necessary based on kidney function and stages of CKD.
  5. Your dietitian or provider will help you understand the restrictions of proteins, fat, salt, and minerals in the food you eat. Follow dietary instructions:
    a. Dietary intake of protein is usually restricted to 0.8 to 1.0 g/kg/d.
    b. Dietary sodium (salt) should be restricted to no more than 2 g/d.
    c. Potassium should be restricted to 40 to 70 mEq/d.
    d. Calories should be restricted to 35 kcal/kg/d; for your height and weight, a_____-calorie diet is your recommended goal.
    e. Fat intake should be about 30% to 40% of total daily caloric intake.
    f. Phosphorus should be restricted to 600 to 800 mg/d.
    g. Calcium should be restricted to 1,400 to 1,600 mg/d.
    h. Magnesium should be restricted to 200 to 300 mg/d.
    i. Carbohydrates: The recommended normal intake is 225 to 358 g/d.
C. Exercise:
  1. Avoid being inactive. Some physical activity is better than none.
  2. Moderate exercise is recommended for at least 150 minutes (2 hours and 30 minutes) a week:

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CLIENT TEACHING GUIDE

CHRONIC KIDNEY DISEASE (*CONTINUED*)

a. Aerobic exercise that increases your heart rate should be done in episodes of at least 10 minutes and should be spread throughout the week, at least 3 days a week.
b. Muscle-strengthening activities should be done at least 2 days a week.

3. The benefits of exercise:
a. Lowers blood pressure.
b. Improves cholesterol.
c. Lowers hemoglobin A1C in diabetes.
d. Strengthens bones.
e. Leads to weight loss.
f. Improves signs/symptoms of depression.
g. Boosts the immune system.
h. Reduces stress.
i. Provides an overall better feeling.

D. Target heart and blood vessels:
1. Control blood pressure: Goal is less than 130/80 mmHg.
2. Lower your cholesterol, especially low-density lipoprotein (LDL).
3. Lose weight. Every pound counts.
4. Lower hemoglobin A1C level with the goal less than 6.9% if you have diabetes:
a. Exercise.
b. Diet: no restrictions, low-saturated-fat, low-carbohydrate diet.
c. Smoking cessation is essential.
d. Reduce or eliminate alcohol intake.
e. Medications may be necessary for the following:
i. Hypertension: blood pressure medications.
ii. Hyperlipidemia: cholesterol medications.
iii. Diabetes: medications to control blood sugar.

E. Target bones:
1. Become familiar with your lab results (vitamin D level, calcium level, phosphorous level).
2. Supplements: Vitamin D, calcium supplements, and phosphate binders may be necessary.
3. Low phosphorous diet may be necessary.
4. Exercise: Weight-bearing exercise will help strengthen bones (walking, dancing, lifting weights, etc.).

F. Target anemia:
1. Goal of the hemoglobin should be 11 g/dL or better.
2. Diet rich in iron.
3. Medication may be necessary (depends on the type of anemia):
a. Erythropoietin-stimulating agents.
b. Vitamin B_{12}.
c. Folic acid.

G. Target water retention:
1. Limit salt intake.
2. Diet (avoid processed foods/fast foods).
3. Medications may be needed (diuretics).

FOODS HIGH IN PHOSPHORUS Amount of phosphorus in foods. Limit portions (registered dietitian consult is recommended).

POTASSIUM-FRIENDLY FOODS Limit portion to 1/2 cup unless otherwise noted (registered dietitian consult is recommended).

RESOURCES American Diabetes Association: www.diabetes.org

Food and Fitness: www.diabetes.org/food-and-fitness/?loc=GlobalNavFF

BC Renal Agency: www.bcrenalagency.ca

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CLIENT TEACHING GUIDE

CHRONIC KIDNEY DISEASE (*CONTINUED*)

TABLE 2 LIST OF FOODS HIGH IN PHOSPHORUS

Biscuits (mix) 1 (1 oz) 133 mg	Cheese (1 oz) 161 mg
Chocolate 1 bar (2 oz) 137 mg	Cola 1 can (12 oz) 60 mg
Cream soups (1 cup) 151 mg	Dried beans and peas (1 cup cooked) 206 mg
Hot dogs and sausage (2 each) 162/220 mg	Ice cream (1 cup) 157 mg
Liver and organ meats (3.5 oz) 400 mg	Macaroni and cheese (1 oz) 265 mg
Pancake mix 3 (4-in. pancakes) 368 mg	Peanut butter (3 tbsp) 172 mg
Pizza (1 slice) 259 mg	Pork and beans (1 cup) 266 mg
Yogurt (8 fl. oz) and pudding (1/2 cup) 292/280 mg	

TABLE 3 LIST OF POTASSIUM-FRIENDLY FOODS

Fruits	
Apple	Berries (strawberries, blueberries, blackberries, cranberries)
Cherries	Grapes
Juices (apple, cranberry, grape, lemon, nectar, pear, peach, and pineapple)	Lemon
Peach (canned or 1 fresh)	Pear (canned or fresh)
Pineapple	Plum (1/2 cup canned or fresh)
Strawberries	Watermelon
Vegetables	
Cabbage	Carrots (cooked or raw)
Celery	Corn (canned or fresh)
Cucumbers	Eggplant
Green (or wax) beans	Green peas
Lettuce	Onion
Radishes	Squash (crookneck, summer, and zucchini)

Shopping Guide for Kidney Health: http://www.bcrenalagency.ca/resource-gallery/Documents/Kidney%20Friendly%20Shopping%20Guide%20Final.pdf

Diabetes Kidney-Friendly Grocery List: http://www.bcrenalagency.ca/resource-gallery/Documents/Diabetes%20Kidney-Friendly%20Shopping%20Guide.pdf

National Kidney Foundation: www.kidney.org

Kidney Cooking, A Family Recipe Book for kidney Patients: https://www.kidney.org/sites/default/files/docs/kidney_cookbook_lr.pdf

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CLIENT TEACHING GUIDE

EPIDIDYMITIS

PROBLEM Epididymitis is infection of the gland behind the testicle that stores and carries sperm. This may be a sudden or chronic problem. Discomfort should go away in 13 days. Swelling of the scrotum may be present for up to 8 weeks.

CAUSE It is usually caused by a bacterial infection. The *Escherichia coli* bacteria is a common cause of infection; epididymitis is often caused by a urinary tract infection (UTI), or possibly from a sexually transmitted infection.

PREVENTION

A. Prevent UTIs with good hygiene:
  1. Keep your penis and scrotum clean.
  2. Clean under foreskin if uncircumcised.
  3. Wash your hands after each time you go to the bathroom (both urine and bowel movements).
B. Limit your sexual partners and use a condom to prevent all types of infection.

TREATMENT PLAN

A. Try one of the following comfort measures:
  1. Intermittent cold compress for acute swelling and pain relief. Do not leave an ice pack on the skin for longer than 20 minutes, and do not use it more often than once every hour.
  2. Local heat or sitting in warm bath water also help relieve the initial discomfort.
B. Use an athletic supporter or elevate scrotum on a small rolled washcloth.
C. When the pain is better, wearing brief underwear will support the scrotum and can help with the pain.
D. Your sexual partner(s) need(s) to be treated for sexually transmitted disease.

Activity:
A. Rest; do not engage in strenuous activity or heavy lifting.
B. Avoid sex until you finish the antibiotic.

Diet:
A. It is important that you drink plenty of fluids.
B. Avoid foods that irritate the bladder: caffeine, alcohol, and spicy foods.

Medications:
A. Antibiotics are used to kill bacteria causing infection.
B. Take all of the antibiotics prescribed for you; do not stop taking drugs after symptoms are gone.
C. Do not share your antibiotics with your sex partner(s); a full prescription of antibiotics is needed for both of you to get better.
D. Take anti-inflammatory drugs such as acetaminophen (Tylenol) or ibuprofen as needed for discomfort.
E. You may be prescribed a pain medication only for a short period of time.

You Have Been Prescribed the Following Antibiotic: _____

You Need to Take: _____

Finish all of the antibiotics, even if you feel better.

You Have Been Prescribed the Following for Pain: _____

You Need to Take: _____

You Need to Notify the Office If:
A. You have a great increase in pain or swelling in the scrotum after going home.
B. You have worsening symptoms after starting the antibiotics.
C. You have discharge from your penis.
D. You have pain during ejaculation.
E. You have fever over 100.4°F.
F. Other: _____

Phone: _____

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NOCTURIA

PROBLEM Nocturia is waking up to urinate more than once or twice a night. Symptoms include having to go to the bathroom more often, especially at night; trouble starting or stopping your urine; decreased stream; and feeling that you do not empty.

CAUSES There are many causes of nocturia, including drinking liquids too close to bedtime, certain medicines, chronic health problems, hormone changes, pregnancy, and aging.

PREVENTION Prevention of nocturia depends on the cause.

TREATMENT PLAN Treatment depends on how bad the symptoms are and what is the cause.

A. Empty your bladder on a schedule every 2 to 3 hours to prevent overfilling of the bladder.
B. Do not drink liquids within 2 hours of your bedtime.
C. You may be asked to keep a 24-hour bladder diary.
D. Your provider will review all of your medications. You may need to change your schedule of when you take certain medicines. You also may need other medications prescribed.
E. Males with prostate enlargement may need an operation if there is an obstruction or other causes that need to be fixed by surgery.
F. Double-void to empty your bladder more completely. Once you have finished passing urine, count to 10 slowly and try to pass urine again.

ACTIVITY:

A. There are no activity restrictions.
B. You may need to plan schedules with access to bathrooms in mind.
C. Getting up during the night increases your risk for falls. Safety measures include the following:
    1. Remove small area carpets and rugs.
    2. Keep night lights along the path to the bathroom or use a flashlight.
    3. Install grab bars.

Diet: Avoid spicy foods that irritate the bladder. Caffeine and alcohol act as diuretics and increase your need to urinate.

Medications:

A. Medications that help relieve the blockage may block hormones or relax the muscles that control urination.
B. Antibiotics are used if there is an infection in your bladder or prostate.
C. Do not take over-the-counter medications such as cold medications, decongestants, antihistamines (for allergies), and diarrhea medicines. They make symptoms worse.
D. Always read labels to check for advice: for example, "Do not take if you have prostate enlargement."

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:

A. You cannot urinate.
B. Your symptoms worsen.
C. You have a fever.
D. Other: _____

Phone: _____

RESOURCES American Urogynecologic Society: www.augs.org

American Urological Association: www.UrologyHealth.org

National Association for Continence: www.nafc.org

The Simon Foundation for Continence: www.simonfoundation.org

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CLIENT TEACHING GUIDE

PROSTATITIS

PROBLEM Prostatitis is infection and/or inflammation of the prostate. Symptoms can be problems with urinating, increased frequency or painful urination, fever, or chills. You may have pain in the scrotum and buttocks and blood in the urine or semen. Prostatitis can be treated and does not cause impotence.

CAUSE An infection may be caused by bacteria from the bladder or reflux of urine or it may have started from a rectal infection.

PREVENTION

A. Prevent prostate infection with good hygiene:
  1. Wash your hands after each time you go to the bathroom (urine and bowel movements).
  2. Clean under foreskin if uncircumcised.
  3. Urinate when you have the urge. Do not hold your urine for long periods of time.
  4. Limit your sexual partners and use a condom to prevent all types of infection.
B. Staying sexually active with ejaculation may reduce the incidence of prostatitis.

TREATMENT PLAN

A. You may have to be in the hospital for severe infections.
B. At home, try the following comfort measures:
  1. Use a sitz bath or sit in warm bath water or a whirlpool three times a day to provide some relief.
  2. You may find you feel more comfortable when you empty your bladder in a warm water bath when your pelvic muscles relax.
C. Your sexual partner(s) may need to be evaluated and treated, too (ask your healthcare provider).

Activity: Rest; do not engage in strenuous physical activity or heavy lifting.

Diet: Increase fluid intake. ***Fluid Exception:*** **Decrease caffeine and alcohol, which can irritate the urethra.**

Medications: _____

You Have Been Prescribed the Following Antibiotic: _____

You Need to Take: _____

Finish all of the antibiotics even if you feel better.
A. Antibiotics cure the infection by killing bacteria. You need to continue taking these drugs until the infection is completely cured (usually about 1 month). You need to take all the antibiotics even if your symptoms are gone.
B. Pain medications combined with anti-inflammatory drugs to reduce pain are usually taken for 3 to 7 days. Take acetaminophen (Tylenol) to bring down fever.

You Have Been Prescribed the Following for Pain: _____

You Need to Take: _____

You Have Been Prescribed the Following for Fever: _____

You Need to Take: _____

You Have Been Prescribed the Following for Chronic Prostatitis/Chronic Pelvic Pain Syndrome:

You Need to Take:

_____

(*continued*)

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CLIENT TEACHING GUIDE

PROSTATITIS (*CONTINUED*)

You Need to Notify the Office If You Have:

A. Symptoms that get worse or do not improve during treatment.
B. An inability to urinate; you may need to go to the hospital.
C. Symptoms that recur after treatment.
D. Fever over 100.4°F.
E. Other: _____

Phone: _____

RESOURCES American Urological Association Foundation: www.UrologyHealth.org

National Kidney and Urologic Disease Information Clearinghouse: www.kidney.niddk.nih.gov

National Urology Health Hotline Toll Free 1-800-828-7866

The Prostatitis Foundation: www.prostatitis.org

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TESTICULAR SELF-EXAMINATION

The testicular self-examination is easy to do and does not take very much time to perform. Checking every month is a good way to become familiar with this area of your body and will help detect testicular cancer.

Set a date for the same day every month. An easy way to remember is choosing the first day of the month, the last day of the month, or your birth date. The best time to check your testicles is during or after a hot bath or shower. (Heat makes the testicles relax.)

Tumors can be felt. Boys and men from 15 to 35 years are at the highest risk due to hormonal activity.

PROCEDURE

A. If possible, do the self-examination in front of a mirror after a hot bath or shower.
B. Check for any swelling of the skin.
C. Support each testicle with one hand and examine it with the other hand.
D. Use both hands to feel all of the scrotal bag:
  1. With one hand, lift your penis, and check your sac with the other hand. Feel any change in shape or size.
  2. Look for red or blood veins that are bigger than they used to be.
  3. The left side may hang slightly lower than the right (this is normal).
E. Check each testicle:
  1. Place your left thumb on the front of your left testicle and your index and middle fingers behind it.
  2. Gently but firmly roll the testicle between your thumb and fingers.
  3. Then use your right hand to examine the right testicle the same way.
  4. The testicles should feel smooth, rubbery, oval-shaped, and slightly tender. They should move freely.
  5. Locate the epididymis and spermatic cord. The epididymis is the irregular, cord-like structure on the top and the back of the testicle:
    a. Gently squeeze the spermatic cord above your left testicle between your thumb and the first two fingers of your left hand.
    b. Check for lumps and masses along the entire length of the cords.
    c. Repeat on the right side, using your right hand.
F. Call your healthcare provider if you notice the following:
  1. Any lumps, even small, pea-sized ones.
  2. Any masses; sometimes feeling “like a bag of worms.”
  3. A dull ache in the lower abdomen or in the groin.
  4. A feeling of heaviness in the scrotum.
  5. A significant loss of size in one of the testicles.
  6. Pain or discomfort in a testicle or in the scrotum.
  7. Any other changes you noticed since the last time you felt yourself for your examination.
G. Your healthcare provider may refer you to a urologist for further evaluation.

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CLIENT TEACHING GUIDE

URINARY INCONTINENCE: FEMALES

PROBLEM Urinary incontinence is when you "leak" or are unable to hold your urine. **Incontinence is not considered a normal part of aging.**

CAUSE The cause depends on the type of incontinence you have. Incontinence is common in conditions that cause pressure on the bladder, such as pregnancy, chronic coughing, obesity, menopause, pelvic organ prolapse, and certain neurologic problems:

A. Functional incontinence: lose urine because you are unable to get to the bathroom.

B. Urge: unable to delay emptying your bladder after the sensation of fullness is felt; may leak urine when hearing running water, placing hands in water, or trying to get into the bathroom.

C. Overflow: loss of urine when the bladder is "too full."

D. Stress: unable to hold your urine during coughing, sneezing, laughing, bending over, or other physical activity.

E. Overactive bladder: stress incontinence with or without urge incontinence; symptoms include urgency, frequency, and getting up to empty your bladder one or more times a night.

PREVENTION Exercise regularly and practice pelvic floor exercises, commonly called Kegel exercises. Do not become constipated, so you do not strain to have a bowel movement. Stop smoking. If you have a cough, you may need to see your healthcare provider to help treat it.

TREATMENT PLAN Treatment depends on the cause and type of incontinence. Fill out the bladder diary (see Table 1) and return it to your provider to help figure out what kind of problem you have.

A. Pelvic floor exercises should be done every morning, afternoon, and evening; repeat the exercise five times for each set, and gradually increase to 10 times each set. To perform these exercises:

1. Start by doing your pelvic muscles exercises lying down. When your muscles get stronger, do your exercises sitting or standing.
2. Do not tighten your tummy, leg, or butt muscles, just squeeze the muscles you use to start and stop the flow of urine.
3. Do not hold your breath or practice while you are on the toilet urinating.
4. Pull in pelvic muscles and hold them tight for a count of five.
5. Repeat five times.
6. Work up to doing three sets of 10 repeats.
7. Kegel exercises take just a few minutes a day, and most females notice an improvement after a few weeks of daily exercise.

B. Empty your bladder frequently. As soon as you feel the urge to urinate, go to the bathroom. You may need to go to the bathroom on a timed schedule.

C. You may be taught relaxation techniques to control the feeling of having to go quickly.

D. Avoid becoming constipated and having to strain down to have a bowel movement.

Activity: Try to get daily exercise. Use absorbent undergarments until your bladder leaking is under control.

Diet: Eat a well-balanced diet. If you are overweight, consider a weight-loss program. Avoid drinking lots of liquids, especially caffeinated beverages and alcohol.

Medications: What you take depends on the type of bladder leakage you have.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have: _____

Phone: _____

RESOURCES American Urogynecologic Society: www.augs.org

American Urological Association: www.UrologyHealth.org

National Association for Continence: www.nafc.org

The Simon Foundation for Continence: www.simonfoundation.org

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CLIENT TEACHING GUIDE

URINARY INCONTINENCE: FEMALES (*CONTINUED*)

YOUR DAILY BLADDER DIARY This diary will help you and your healthcare team figure out the causes of your bladder-control trouble. The **sample** line shows you how to use the diary. Use this sheet as a master for making copies that you can use as a bladder diary for as many days as you need.

Your Name: _____

Date: _____

TABLE 1 BLADDER CONTROL DIARY

Time	Drinks		Trips to the bathroom		Accidental leaks		Did you feel a strong urge to go?	What were you doing at the time?
	What kind?	How much?	How many times?	How much urine? Circle one.	How much? Circle one.		Circle one.	Sneezing, exercising, having sex, lifting, etc.
Sample	Coffee	2 cups	✓✓	○ sm ○ med ○ lg	○ sm ○ med ○ lg		Yes (No)	Running
6–7 a.m.				○ ○ ○	○ ○ ○		Yes No	
7–8 a.m.				○ ○ ○	○ ○ ○		Yes No	
8–9 a.m.				○ ○ ○	○ ○ ○		Yes No	
9–10 a.m.				○ ○ ○	○ ○ ○		Yes No	
10–11 a.m.				○ ○ ○	○ ○ ○		Yes No	
11–12 noon				○ ○ ○	○ ○ ○		Yes No	
12–1 p.m.				○ ○ ○	○ ○ ○		Yes No	
1–2 p.m.				○ ○ ○	○ ○ ○		Yes No	
2–3 p.m.				○ ○ ○	○ ○ ○		Yes No	
3–4 p.m.				○ ○ ○	○ ○ ○		Yes No	
4–5 p.m.				○ ○ ○	○ ○ ○		Yes No	
5–6 p.m.				○ ○ ○	○ ○ ○		Yes No	
6–7 p.m.				○ ○ ○	○ ○ ○		Yes No	
7–8 p.m.				○ ○ ○	○ ○ ○		Yes No	
8–9 p.m.				○ ○ ○	○ ○ ○		Yes No	
9–10 p.m.				○ ○ ○	○ ○ ○		Yes No	
10–11 p.m.				○ ○ ○	○ ○ ○		Yes No	
11–12 midnight				○ ○ ○	○ ○ ○		Yes No	
12–1 a.m.				○ ○ ○	○ ○ ○		Yes No	
1–2 a.m.				○ ○ ○	○ ○ ○		Yes No	
2–3 a.m.				○ ○ ○	○ ○ ○		Yes No	
3–4 a.m.				○ ○ ○	○ ○ ○		Yes No	
4–5 a.m.				○ ○ ○	○ ○ ○		Yes No	
5–6 a.m.				○ ○ ○	○ ○ ○		Yes No	

I used _____ pads today. I used _____ diapers today (write number).

Questions to ask my healthcare team:

_____

_____

Note: Let's Talk About Bladder Control for Women is a public health awareness campaign conducted by the National Kidney and Urologic Diseases Information Clearinghouse (NKUDIC), an information dissemination service of the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), National Institutes of Health.

Source: From NKUDIC, National Institutes of Health (NIH).

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CLIENT TEACHING GUIDE

URINARY RETENTION

PROBLEM Urinary retention is the inability to empty your bladder. Symptoms include not being able to urinate or the inability to empty your bladder completely. **Acute urinary retention can cause a great deal of discomfort or pain when you cannot empty your bladder even though it is full, and is considered a medical emergency.**

CAUSES There are many causes of urinary retention, including from side effects of medications or a blockage of the bladder such as from a kidney stone. Other causes include a urinary tract infection (UTI), not emptying your bladder after surgery, stroke, multiple sclerosis, or trauma. The most common cause for males is related to the prostate. One cause of urinary obstruction is a prolapse of the uterus or cystocele/rectocele. Females may experience urinary retention after delivery of a baby.

PREVENTION None.

TREATMENT PLAN Treatment depends on the cause and how bad the symptoms are.

A. Medication side effects can cause urinary retention but there are others that can help.
B. An operation to fix the obstruction may be needed.
C. Do not take over-the-counter medications such as cold medications, decongestants, antihistamines (for allergies), and diarrhea medicines. They make symptoms worse.
D. Always read labels to check for advice: for example, "Do not take if you have prostate enlargement."
E. Avoid drinking liquids before bedtime or before going out.
F. Double-void to empty your bladder more completely.
G. You may be given a bladder diary to fill out and bring to your next visit.
H. In some cases you may be taught to insert a catheter into your bladder to empty it and relieve the symptoms.
I. Empty your bladder on a schedule every 2 to 3 hours to prevent overfilling of the bladder.

Activity: There are no restrictions. You may need to plan schedules with access to bathrooms in mind. Kegel exercise may help for some females.

Diet: Avoid spicy foods that irritate the bladder. Caffeine and alcohol act as diuretics and increase your need to urinate.

Medications:

A. Medications may be prescribed to help relieve urinary retention by relaxing the muscles that control urination.
B. Antibiotics are used if there is also an infection in your bladder or prostate.

You Have Been Prescribed: _____

You Need to Take: _____

EMERGENT ISSUES/INSTRUCTIONS **Call your health provider or go to the ED immediately if you are unable to urinate. This is considered a medical emergency.**

A. Other: _____

Phone: _____

RESOURCES American Urogynecologic Society: www.augs.org

American Urological Association: www.UrologyHealth.org

National Association for Continence: www.nafc.org

The Simon Foundation for Continence: www.simonfoundation.org

Urinary Catheterization Female: www.nlm.nih.gov/medlineplus/ency/patientinstructions/000144.htm

Urinary Catheterization Male: www.nlm.nih.gov/medlineplus/ency/patientinstructions/000143.htm

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CLIENT TEACHING GUIDE

URINARY TRACT INFECTION

PROBLEM You have a bladder infection. The symptoms include painful, frequent urination, and pain over your bladder. Your symptoms may be mild, moderate, or painful.

CAUSE Bacteria caused the infection of the bladder. A bladder infection is more common in females and in males who have prostate problems.

PREVENTION

A. Empty your bladder often:
  1. As soon as you feel the urge to go, empty your bladder at that time.
  2. Do not hold your urine.
  3. You may need to urinate on a schedule during the day, at least every 2 to 3 hours.
B. Wash your hands after going to the bathroom (both urine and bowel movements).
C. Good hygiene for females:
  1. Wipe front to back every time you empty your bladder and especially after bowel movements.
  2. Take showers instead of baths; do not take bubble baths.
  3. Empty your bladder before and after sex.
  4. Avoid feminine hygiene sprays and douches.
D. Wear cotton underwear. Do not wear tight underwear and clothes. Take off your underwear at night while sleeping.

TREATMENT PLAN Treatment depends on how bad (severity) the infection is. Antibiotics are used to kill bacteria that cause infections. **The most important thing is to finish all of your medications even if you feel better.**

Activity: Rest; avoid strenuous activity. Avoid sexual activity until you finish the antibiotics.

Diet:

A. Increase fluids; drink at least one large glass of liquid every hour.
B. Avoid foods that irritate the bladder: caffeine, alcohol, and spicy foods.
C. Drink cranberry juice to help fight bladder infections. If you do not like plain cranberry juice, mix it with another juice, such as orange juice.

Medications:

A. Antibiotics kill bacteria that cause infection. Make sure you take all of your medications, not just until you feel better.
B. Take acetaminophen (Tylenol) for fever.
C. You may be prescribed a medication to prevent bladder spasms and pain while urinating. This changes the color of your urine to orange or blue.

You Have Been Prescribed the Following Antibiotics:

_____

You Need to Take: _____

Take all of your antibiotics, even if you feel better.

You Have Been Prescribed the Following for Discomfort: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Worsening symptoms or symptoms not improving during treatment.
B. Fever higher than 100.4°F.
C. Blood in your urine.
D. Symptoms that come back after you finish all of your medications: painful urination, back pain, fever, chills, or nausea.
E. Other: _____

Phone: _____

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CHAPTER 16

OBSTETRICS GUIDELINES

Penny Wortman

PREPREGNANCY COUNSELING: IDENTIFYING CLIENTS AT RISK

A. A person's health before pregnancy influences their ability not only to conceive, but also to maintain pregnancy and to achieve a healthy outcome. Some people are unaware that their medical conditions, medications, occupational exposure, or social practices may have negative consequences in the earliest weeks of pregnancy before the pregnancy test is positive. They do not realize that organogenesis begins around 17 days after fertilization. Steps to provide the ideal environment for the developing fetus are most likely to be effective if they precede the traditional initiation of prenatal care.

B. The goal of prepregnancy care is to reduce perinatal mortality and morbidity. Targeting only self-referred clients who are planning their next pregnancy, or those referred with risk factors can result in a significant number of missed opportunities for primary prevention. The American College of Obstetricians and Gynecologists (ACOG) recommends counseling to all nonpregnant females and males with reproductive potential at each visit in an effort to improve health outcomes during the reproductive period. Nurses working with all those of childbearing age and their families have a responsibility to promote reproductive health during every health encounter. Even among married females in the United States, the unintended pregnancy rate is 45%.

C. Identifying clients at risk begins with an individual's intention for pregnancy. Prepregnancy counseling must include timing and spacing of pregnancies. Optimizing maternal, fetal, and neonatal health outcomes requires a thorough health and social history. Beginning with a review of the client's health history will help to identify specific needs for screening and counseling. Reviewing the social history will also help with identifying clients at risk. Along with a review of the health and social history, it is important to obtain accurate pregnancy dating which is a vital part of a healthy outcome.

D. When discussing preconception plans, the client's history, as well as that of their partner, should be evaluated for poor health habits (alcohol, smoking, drug use), exposure to toxic substances (radiation and chemicals), multiple sexual partners (risk of HIV, hepatitis, and sexually transmitted infections [STIs]), and racial or ethnic origin. Prepregnancy evaluation should identify any current health issues as well as barriers to healthcare access. Pregnancy and reproductive health history are also important parts of the review.

E. Health needs can be identified with a review of the following:

1. Maternal age: Pregnancy-induced hypertension (PIH) occurs at the extreme of ages, insulin-dependent diabetes increases with maternal age, and the risks of Down syndrome and other chromosomal abnormalities increase with maternal age. Advanced maternal age (AMA) is defined as 35 years of age at delivery. The ACOG requires counseling on genetic screening/testing options for females of AMA. Paternal age is also a factor in need for consideration of genetic screening.

2. Universal carrier screening is available for females who desire to know if they are carriers for an autosomal recessive disorder such as Tay–Sachs disease, sickle cell anemia, alpha and beta thalassemias, cystic fibrosis, and spinal muscular atrophy. Ideally, this blood test should be done preconceptionally, but it can be performed at any time during the pregnancy. If the screening test is positive, the client's partner can then be tested.

3. Social issues: Screen every client for intimate partner violence. Research indicates that most abused individuals continue to be victimized during pregnancy and that the violence may escalate. Child abuse is also common in homes where there is abuse of adults. This assessment should be done only if the partner is not present. Information should be given to the client concerning available community, social, and legal resources, and their immediate safety, the safety of their children, and an escape plan should be assessed. Report child abuse to the state's mandatory reporting agency.

4. Financial: Discuss insurance, maternity benefits, work-leave policy, and contingency plans for lost wages due to pregnancy complications with the client.

5. Environmental and occupational considerations: Routine assessment of hobbies and home environment along with employment environment of both partners may identify exposures that have been associated with adverse reproductive consequences that can be minimized in the prepregnancy period.

6. Adverse fetal effects are dose-dependent and gestational age at exposure related to the following:

a. Radiation: Fetal effects include microcephaly, mental retardation, eye anomalies, intrauterine growth retardation (IUGR), and visceral malformations. Lead aprons should be used to protect the client from any radiation exposures.

b. Heavy metals: Mercury exposure is related to brain damage and neuromuscular defects. Lead exposure is ▶

The contributions of Christina Reed, Susan Drummond, and Merita O'Sullivan to this chapter in prior editions are acknowledged here.

related to increased spontaneous abortion, low birth weight, brain damage, and increased premature rupture of membranes (PROM). Cadmium is retained by the fetal liver and kidney and is also associated with fetal craniofacial defects. Nickel is associated with neonatal deaths.

c. Pesticides: Occupations at risk for pesticide exposure include, but are not limited to, the following: ranch and farmworkers (including migrant workers); gardeners (home and professional); groundskeepers; florists; structural pest control workers; hunting and fishing guides; healthcare workers who deal with contamination; and people employed in pesticide production, mixing, and application. Dioxin is associated with an increased rate of spontaneous abortion, myelomeningocele, and limb defects. The pesticides dichlorodiphenyltrichloroethane (DDT) and dichlorodiphenyldichloroethylene (DDE) are associated with increased abortion, prematurity, low birth weight, PIH, and early weaning.

d. Other: Carbon monoxide is associated with increased stillbirths, neurologic deficits, seizures, spasticity, and retarded psychomotor development. Ozone is associated with increased spontaneous abortion and increased structural defects. Anesthetic gases are associated with increased abortion, birth defects, low birth weight, and infertility.

7. Infectious diseases: See Chapter 18, "Sexually Transmitted Infections Guidelines," and Chapter 19, "Infectious Disease Guidelines."

8. Medications: Assess and minimize the risk of exposure to medications by reviewing the client's use of prescription and nonprescription drugs, including herbal supplements. Provide the client with information on the safest choices and avoiding drugs associated with fetal risks. Identify all prescription and nonprescription medications taken by the mother and partner to assess for risks to the fetus associated with current medications. Teratogenic defects linked to certain medications may include cleft lip and palate, congenital heart disease, microcephaly, caudal dysplasia, and caudal regression syndrome.

9. Medical problems: Health assessment of potential risk not only to the fetus but also to the mother, should they become pregnant, should be discussed. Care must be taken to identify and counsel all clients whose life expectancy could be markedly reduced by pregnancy or whose fetus would have a high likelihood of complications. For example, individuals with known cardiac problems, epilepsy, transplanted organs, or uncontrolled diabetes and hypertension should be told of the risks associated with pregnancy:

a. Diabetes: Researchers have demonstrated a dose-related response between glycosylated hemoglobin (Hgb A1C) during the first trimester of pregnancy and the incidence of congenital defects: the better the glycemic control, the lower the risk for birth defects. The prepregnancy plan for diabetes includes the following:

i. Change all clients on oral agents to insulin therapy before pregnancy is attempted.

ii. Achieve strict plasma glucose control. ACOG recommends self blood glucose monitoring during pregnancy with the following glucose levels to be met:

1) Fasting: ≤95 mg/dL.

2) Preprandial glucose values less than 100 mg/dL.

3) 1-hour postprandial glucose levels less than 140 mg/dL.

4) 2-hour postprandial glucose levels less than 120 mg/dL.

5) Nighttime glucose levels should not drop below 60 mg/dL. Care should be taken to avoid hypoglycemia during pregnancy.

iii. Reduce the Hgb A1C to 6% or less.

iv. Assess the client for vasculopathy, neuropathy, nephropathy, and retinopathy.

v. Refer the client for genetic and nutritional counseling.

vi. Enhance the client's knowledge of diabetes during pregnancy.

vii. Nutrition: Dietary evaluation and recommendations of alternatives that may benefit the fetus' development are important components of prepregnancy counseling. Evaluation of nutritional status should include assessment of the appropriate weight for the client's height as well as a discussion of eating habits such as vegetarianism, fasting for religious or personal reasons, eating disorders, the use of megavitamins, and herbal supplements: Dietary restrictions due to allergies and food sensitivities are important to discuss.

b. Neural tube defects (NTDs): In about 90% of the cases, NTDs are not expected on the basis of history. NTDs are associated with multifactorial causes, including environmental factors, undernutrition (lack of folic acid), chromosomal defects, maternal hyperthermia, diabetes, clomiphene citrate (Clomid) induction, and maternal obesity. In 1996, the Food and Drug Administration (FDA) approved a population-based strategy, effective January 1998, to fortify grain food sources with folic acid. The recurrence risk of NTD is 2% to 3% without the use of preconceptional doses of folic acid. Even with the FDA strategy, folic acid supplement of 0.4 mg (400 mcg) per day at least 1 month before conception and during the first trimester of pregnancy is recommended. Those who have had a child with an NTD and those with seizure disorders require higher doses of folic acid, 4 mg daily.

10. Obstetric considerations: Prepregnancy reproductive history is an important tool for identifying factors that may be amenable to intervention. Review term, preterm, aborted (elective, spontaneous, and therapeutic) pregnancies, as well as a short history on living children. Review the gestational age at delivery of each neonate and any pregnancy and delivery complications. Preterm labor (PTL) has a 30% recurrence risk, and preeclampsia has a 5% to 70% recurrence rate in subsequent pregnancies. Clients who developed severe features of preeclampsia and delivered before 30 weeks' gestation have the highest risk of preeclampsia in future pregnancies. In some instances, after prepregnancy and genetic counseling, the couple may decide to forego pregnancy or to use assisted reproductive technologies such as donor eggs and/or sperm.

11. Genetic screening: Recommended for all individuals wishing to become pregnant. Genetic counseling for discussion of testing options is recommended if the mother is ▶

35 years or older at the time of delivery, or if they have a family history of any abnormal genetic disorders, such as Down syndrome. The parents choose whether they would like to have genetic testing performed to evaluate the fetus for abnormal chromosomes. The following tests can be performed for genetic screening:

a. Noninvasive prenatal screening: Cell-free DNA from the fetus is found in maternal serum and can lead to prenatal identification of pregnancies at high risk for trisomy 13, 18, and 21, as well as detection of sex. This screen is a maternal blood test and can be done as early as 10 weeks. A positive screen should be confirmed with a diagnostic test such as chorionic villus sampling (CVS) or amniocentesis.

b. CVS: performed at 10 to 12 weeks' gestation.

c. Amniocentesis: performed at 15 to 18 weeks' gestation, ideally, but can be performed later in pregnancy.

i. Amniocentesis can also be performed to assess for spinal cord defects. Amniocentesis can detect elevated protein levels (alpha-fetoprotein and the presence of acetyl cholinesterase) in the amniotic fluid that is present in the event of a spinal cord defect. Therefore, if performing an amniocentesis, information regarding both genetic makeup and spinal cord defects can be determined during the single procedure of the amniocentesis.

12. Recurrent loss: The workup and counseling for recurrent losses include evaluation for a uterine defect (septal or bicornuate uterus or uterus didelphys), endocrine problems (luteal phase defect or hypothyroidism), chromosomal defects, or presence of antiphospholipid syndrome. Antiphospholipid syndrome is defined as the presence of maternal anticardiolipin antibodies and/or lupus anticoagulant in association with recurrent pregnancy loss, thrombotic events, and/or thrombocytopenia. Testing for anticardiolipin antibodies and lupus anticoagulant should be considered for clients who have a history of three or more unexplained consecutive pregnancy losses before 10 weeks or have a history of one or more unexplained pregnancy losses after 10 weeks with a morphologically normal fetus. In the nonpregnant client, thrombosis of a single vessel is the most common complication associated with antiphospholipid syndrome. Those with pregnancy loss should be asked of family history of autoimmune disorders and prior screening for celiac disease.

13. Lifestyle: Queries regarding a client's social lifestyle history should seek to identify behaviors and exposures that may compromise reproductive outcome. While environmental exposures are a frequent concern of couples considering pregnancy, clients should be informed that, in general, use of alcohol, tobacco, and other mood-altering drugs is more hazardous for a fetus than most other lifestyle choices:

a. Alcohol: Alcohol is a known teratogen. There is no safe limit of alcohol use during pregnancy. Clients should be informed that prenatal alcohol consumption is a preventable cause of birth defects including neurodevelopmental deficits and intellectual disability. A recent study reported increasing rates of alcohol consumption during pregnancy suggesting that prepregnancy counseling as well as counseling in all three trimesters may be beneficial. Although statistics vary widely, research indicates that as many as 50% of mothers expose their fetuses to alcohol at some time during pregnancy.

b. Smoking: Fetal effects of smoking are also related to the dose–response effect. Smoking is associated with an increase in bleeding in pregnancy (abruption and placenta previa), IUGR, preterm birth (PTB), low birth weight, stillbirth, respiratory distress in the neonate, and sudden infant death syndrome. Counsel the client on smoking cessation. Suggestions of ways to quit include tapering the use of nicotine (tapering and brand switching to lower tar and nicotine), monitoring smoking behavior, setting a contract to quit smoking, identifying social support or a buddy, and restricting area(s) such as a no smoking zone. Give the client positive reinforcement for behavior change and cessation of the use of tobacco. Nicotine replacement therapy may be considered but only under close supervision with careful discussions with the client, as the U.S. Preventive Services Task Force has concluded that nicotine replacement products during pregnancy have not been sufficiently evaluated for safety or efficacy. Those exposed to secondhand smoke while pregnant are more likely to have lower birthweight babies.

c. Substance use: If substance exposure is complicated by addiction, structured recovery programs are usually needed to effect behavioral change. Substance use/abuse is teratogenic to the fetus, and cessation of all substances is imperative before, during, and after pregnancy.

14. Exercise: Exercise and recreational activities should be reviewed and discussed relative to safety, including the use of bike helmets. If the client is not currently exercising, the prepregnancy period is an excellent time to begin exercising. Additionally, starting an exercise routine during pregnancy is also recommended if there are no complications during the pregnancy. Both aerobic and strength training exercises appear to have no adverse effects to the fetus. Exercise during pregnancy should be under the supervision of the obstetrical provider. Use of saunas or hot tubs and high fevers in the first trimester have been associated with an increased risk of NTDs; however, increased temperature as a result of exercise has not been shown to have the increased risk.

15. Prepregnancy counseling helps to identify high-risk clients who need intensive care during pregnancy and delivery, and it identifies those who need referral for medical management, nutritional counseling, genetic counseling, or behavior modification. Prescribe a prenatal vitamin with the recommended 800 to 1,000 mcg daily of folic acid for any individual less than 35 years of age and 4,000 mcg daily of folic acid for any individual 35 years and older considering pregnancy.

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PRENATAL CARE: INITIAL PRENATAL VISIT

A. The initial prenatal visit is a very important visit. Ideally, all clients should start prenatal care in the first trimester; however, clients may not initiate or have access to prenatal care until the second or third trimester. Therefore, each client's plan of care needs to be adaptive to gestational age on entry of care. During the initial visit, a comprehensive health history is obtained; blood is drawn, and urine collected for baseline prenatal laboratory values to be established; and depending on the time, the physical examination may also be performed. Prescribe a prenatal vitamin if the client is not currently taking them. For clients less than 35 years old, prescribe a prenatal vitamin with the recommended 800 to 1,000 mcg daily of folic acid. For those 35 years or older, prescribe a prenatal vitamin and increase their folic acid to 4,000 mcg daily. Many practitioners have the client return in 1 to 2 weeks to perform the physical exam and to review the blood and urine results from the initial visit.

B. The initial visit is also a time for teaching the pregnant client. Literature and brochures on health promotion (e.g., breast self-exam, dietary recommendation, exercise, and smoking cessation) and information regarding the normal changes, discomforts, and concerns during pregnancy should be provided. The after-hours contact information should also be provided, along with contact information for the labor and delivery unit at the hospital or birth center. Reassure the client that as the pregnancy progresses you will answer questions they may have; however, outside resources may also be beneficial for the client and their family. Encourage the client to enroll in childbirth education, sibling classes (if applicable), breastfeeding classes, and any other classes of interest to them and their partner.

C. Review the client's medical and surgical history (including obstetric history), family genetic history, psychiatric disorders, contraception history, medications taken since the past menstrual period, menstrual history, social habits (smoking, substance abuse, alcohol), environmental exposures (job, hobbies, pets in the home, travel history, etc.), exposure to abuse (mental, physical, sexual), and sources of social support and health promotion (immunizations up to date, enrolled in Women, Infants, and Children [WIC], etc.).

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PRENATAL CARE: FIRST TRIMESTER OVERVIEW

PHYSICAL EXAMINATION

A. General assessment:
- **1.** Vital signs.
- **2.** Weight and height.
- **3.** Fetal heart tones (120–160 beats per minute [bpm]), usually heard at 13 weeks or higher.
- **4.** Fetal movement, usually first felt between 18 and 22 weeks.
- **5.** Mood and attitude toward pregnancy: excitement planned, unplanned ambivalence, or concern based on previous childbirth experience.

B. General appearance.

C. Thyroid:
- **1.** Enlarged/goiter: thyroid-stimulating hormone (TSH) and free T4 and T3 labs, thyroid ultrasound (US) needed.
- **2.** Nodules palpated: TSH and free T4 and T3 labs, thyroid US needed.

D. Breasts:
- **1.** Symmetry.
- **2.** Pain: breast US needed.
- **3.** Masses: breast US needed.
- **4.** Nipple discharge:
  - a. Bloody discharge: breast US needed.

E. Heart:
- **1.** Normal auscultatory changes start in the first trimester and end 1 week after delivery:
  - a. Higher basal heart rate.
  - b. Louder heart sounds.
  - c. Wide splitting of S1.
  - d. Splitting of S2 in third trimester.
  - e. Systolic ejection murmur: pulmonary and tricuspid.
  - f. Third heart sound: most females.
  - g. Fourth heart sound: rare.
  - h. Venous hum.
  - i. Mammary souffle (systolic or continuous) in third trimester and postpartum in breastfeeding females.
  - j. Diastolic murmurs are not normal in pregnant females: consult cardiology.

F. Lungs:
- **1.** Clients with history of asthma need high-risk consultation.

G. Abdomen:
- **1.** Measure the fundal height after 12 weeks' gestation.

H. Skin:
- **1.** Dermatitis is common.
- **2.** Scarring.

I. Extremities.

J. External genitalia:
- **1.** Herpes outbreak present.
- **2.** Bartholin gland enlargement.
- **3.** Condyloma present.

K. Adnexa:
- **1.** Enlarged: transvaginal US needed.
- **2.** Pain: transvaginal US needed.

L. Cervix:
- **1.** Chadwick sign: cervix with a bluish hue from 6 to 8 weeks.

M. Vagina:
- **1.** Increased vaginal discharge: white and clear normal.

N. Rectum:
- **1.** Hemorrhoid present.

COMMON DIAGNOSTIC TESTS

A. Complete blood count (CBC) with platelets.

B. Rubella titer.

C. HIV fourth generation (with the client's consent).

▶

D. Syphilis with reflex to *Treponema pallidium* particle agglutination (TP-PA) or fluorescent treponemal antibody absorption (FTA-ABS) or *T. pallidum* enzyme immunoassay (TP-EIA) (treponemal test; rapid plasma reagin [RPR] or Venereal Disease Research Laboratory [VDRL]—nontreponemal tests).
E. Hepatitis B surface antigen.
F. Blood type and Rh factor.
G. Antibody screen.
H. Urine culture and sensitivity.
I. Hemoglobin (Hgb) fractionation.
J. Hgb A1C.
K. Cystic fibrosis and spinal muscular atrophy genetic carrier screening (with client's consent).
L. Tuberculosis testing, if client has exposure to tuberculosis or symptomatic.
M. Chest x-ray, if the client has a history of tuberculosis and/or positive purified protein derivative (PPD) or QuantiFERON TB Gold blood test.
N. Early 1–hour glucose challenge test (GCT), if client has a history of gestational diabetes, family history of diabetes, obese (body mass index [BMI] 30 or higher), history of fetal macrosomia, or advanced maternal age (AMA).
O. Thyroid panel including TSH and free T4, if client has a thyroid disorder or family history of a thyroid disorder.
P. Vaginal infection testing including chlamydia, gonorrhea, *Trichomonas*, bacterial vaginosis, and *Candida*.
Q. Pap smear and human papillomavirus testing if indicated per guidelines.
R. Dating US before 14 weeks' gestation.

PLAN

A. Pharmaceutical therapy:
1. Prescribe a prenatal vitamin if the client is not currently taking them:
a. Individuals less than 35 years old, prescribe a prenatal vitamin with the recommended 800 to 1,000 mcg daily of folic acid.
b. Individuals 35 years or older, prescribe a prenatal vitamin and increase their folic acid to 4,000 mcg daily.
2. Iron supplement:
a. Prophylaxis: Prenatal vitamins contain the recommended dose of iron.
b. Anemia: Increase elemental iron by 30 to 120 mg/d and instruct the client to take the iron with orange juice to increase absorption in the stomach.
B. Client teaching:
1. Nutrition.
2. Recommended weight gain based on prepregnancy BMI.
3. Prenatal classes.
4. Substance abuse (alcohol, smoking, drugs).
5. Environmental exposures.
6. Domestic violence (history, type of injuries).
7. Over-the-counter (OTC) and herbal supplement medication use.
8. Activity (work, exercise).
9. Travel.
10. Personal hygiene.
11. Sexual activity.
12. Common discomforts/treatments in pregnancy.
13. Symptoms to report immediately.

COMPLICATIONS

A. Ectopic pregnancy.
B. Spontaneous abortion.
C. Missed abortion.
D. Blighted ovum.
E. Threatened abortion.
F. Urinary tract infection (UTI).
G. Pyelonephritis.
H. Anemia.

CONSULTATIONS

A. Women, Infants, and Children (WIC).
B. History of asthma: needs high-risk consultation.
C. History of cardiac abnormalities: needs high-risk consultation.

EMERGENT ISSUES/INSTRUCTIONS

A. Nausea and vomiting are normal; however, if client is unable to eat or drink without vomiting they need to be sent to the ED due to dehydration and abnormal electrolyte levels. Vitamin B_6 and Unisom are used as the drugs of choice to prevent nausea and ondansetron is used in more severe cases.
B. Abdominal pain and/or cramping, backache, pelvic pressure, or other pain need to be evaluated and sent to the ED.
C. Vaginal bleeding with or without pain needs to be evaluated and sent to the ED.

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PRENATAL CARE: SECOND TRIMESTER OVERVIEW

A. Include components of the first trimester evaluation (see "Prenatal Care: First Trimester Overview") at the client's initial visit.

PHYSICAL EXAMINATION

A. General assessment:
1. Vital signs.
2. Weight.
3. Blood pressure (BP).
4. Fetal heart tones (120–160 beats per minute [bpm]).
5. Fetal movement, usually first felt between 18 and 22 weeks.
6. Fundal height.

B. Abdomen:
  1. Measure the fundal height.
C. Skin:
  1. Striae distensae.
  2. Varicose veins.
D. Extremities:
  1. Edema.

COMMON DIAGNOSTIC TESTS

A. Test of cure for urinary tract infections (UTIs) and sexually transmitted infections (STIs) previously treated.

B. Genetic testing offered to all clients: maternal serum multiple marker screening (quad screen) between 15 weeks, 6 days and 21 weeks, 6 days. Genetic testing offered to those who are advanced maternal age (AMA), history of genetic abnormalities, history of prior fetal abnormalities, and positive quad screen: noninvasive prenatal testing (NIPT). Client consent is needed:
  1. Amniocentesis/chorionic villus sampling (CVS), if applicable.

C. Fetal anatomy ultrasound (US) at 20 weeks' gestation.

D. Gestational diabetes screening with 1-hour glucose challenge test (GCT) between 24 and 28 weeks. If the 1-hour 50-g GCT is above or equal to 135, the client needs to complete the 3-hour 100-g glucose tolerance test (GTT). Gestational diabetes is diagnosed when two or more out of four values from the 3-hour 100-g GTT are equal or higher than the cutoffs.

PLAN

A. Pharmaceutical therapy:
  1. Influenza vaccine (seasonal).
  2. Anemia: Increase elemental iron by 30 to 120 mg/d and instruct the client to take the iron with orange juice to increase absorption in the stomach.
B. Client teaching:
  1. Physiologic changes during pregnancy.
  2. Fetal development.
  3. Father's role during pregnancy.
  4. Genetic testing in pregnancy.
  5. Review symptoms to report immediately.

COMPLICATIONS

A. UTIs.
B. Pyelonephritis.
C. Preeclampsia and eclampsia.
D. Gestational diabetes.
E. Anemia.
F. Fetal abnormalities.
G. Abnormal genetic screening results.
H. STIs.

CONSULTATION

A. Genetic consultation is needed with abnormal genetic testing, AMA, and abnormal US findings.

B. High-risk consultation is needed with gestational diabetes, thyroid disorder, asthma exacerbation in pregnancy, severe anemia unresponsive to treatment, HIV-positive, acute tuberculosis infection, congenital syphilis, positive antibody, fetal abnormalities, and maternal cardiac complications.

EMERGENT ISSUES/INSTRUCTIONS

A. Abdominal pain, contractions, backache, pelvic pressure, or other pain needs to be evaluated and sent to the ED.

B. Vaginal bleeding with or without pain needs to be evaluated and sent to the ED.

C. Fever greater than 100.4°F.

D. Evaluate fetal movement, noting when movement was first felt (quickening). If client reports decreased fetal movement, send to the ED to have biophysical profile (BPP) and nonstress test (NST) to evaluate fetal well-being.

E. Headaches with or without blurry vision need to be addressed. BP should be monitored, and if the client has elevated BP with or without headaches and blurry vision they need to be sent to the ED.

F. Leaking of clear fluid from the vagina needs to be assessed and if client has unexplained fluid from the vagina, they need to go to the ED.

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PRENATAL CARE: THIRD TRIMESTER OVERVIEW

A. If the client presents for entry for prenatal care in the third trimester, complete all of the first and second trimester evaluations at the initial visit.

PHYSICAL EXAMINATION

A. General assessment:
  1. Vital signs.
  2. Weight and height.
  3. Blood pressure (BP).
  4. Fetal heart tones (120–160 beats per minute [bpm]).
  5. Fetal movement (fetal kick counts).
  6. Fundal height.
  7. Maternal heart rate.
B. Abdomen:
  1. Fetal presentation.
C. Skin:
  1. Striae distensae.
  2. Varicose veins.
D. Extremities:
  1. Edema.
E. Cervix: Bishop score:
  1. Dilation.
  2. Effacement.
  3. Station.
  4. Position.
  5. Consistency.

COMMON DIAGNOSTIC TESTS

A. Vaginal and rectal culture to identify the presence of group B *Streptococcus* (GBS) infection at 35 to 36 weeks; repeat the culture every 5 weeks if indicated. ▶

B. Fetal growth ultrasound (US); if indicated.
C. Antenatal testing consisting of biophysical profile (BPP) and nonstress test (NST), if indicated.
D. Complete blood count (CBC) with platelets.
E. HIV.
F. Rapid plasma reagin (RPR) with reflex to *Treponema pallidium* particle agglutination (TP-PA) or fluorescent treponemal antibody absorption (FTA-ABS) or *T. pallidum* enzyme immunoassay (TP-EIA) (treponemal test).

PLAN

A. Pharmaceutical therapy:
  1. Tdap vaccine given to all pregnant individuals during each pregnancy. Advise all family members who will be in contact with newborn to also have the Tdap vaccine.
  2. Anemia: Increase elemental iron by 30 to 120 mg/d and instruct the client to take the iron with orange juice to increase absorption in the stomach.
  3. Rho(D) immune globulin (RhoGAM) is given to pregnant clients who are Rh negative at 28 weeks' gestational age.
B. Client teaching:
  1. Fetal kick counts.
  2. Breastfeeding/bottle feeding.
  3. Circumcision.
  4. Infant sibling preparation.
  5. Safety/car seat.
  6. Birth plan.
  7. Postpartum birth control options.
  8. Labor and delivery expectations.
  9. Choosing a pediatrician.
  10. Review symptoms to report immediately and/or to go to the hospital to labor and delivery.

COMPLICATIONS

A. Intrauterine growth restriction (IUGR).
B. Preeclampsia and eclampsia.
C. Gestational diabetes.
D. Fetal demise.
E. Oligohydramnios.
F. Polyhydramnios.
G. Fetal abnormality.
H. Pruritic urticarial papules and plaques of pregnancy (PUPPP).
I. Cholestasis.
J. Preterm labor (PTL).
K. Placenta previa.
L. Placenta accreta.
M. Placenta abruption.
N. Anemia.
O. GBS.
P. Genital herpes flare due to stress.
Q. Chicken pox case in unvaccinated.

CONSULTATIONS

A. Genetic consultation is needed with abnormal genetic testing, advanced maternal age (AMA), and abnormal US findings.
B. High-risk consultation is needed with gestational diabetes, thyroid disorder, asthma exacerbation in pregnancy, severe anemia, HIV positive, acute tuberculosis infection, congenital syphilis, positive antibody, fetal abnormalities, and maternal cardiac complications.
C. Vaginal birth after Cesarean section counseling if the client wants to attempt a vaginal delivery after having one to two prior Cesarean sections.

EMERGENT ISSUES/INSTRUCTIONS

A. Abdominal pain, contractions, backache, pelvic pressure, or other pain needs to be evaluated and sent to the ED if PTL needs to be ruled out.
B. Vaginal bleeding with or without pain needs to be evaluated and sent to the ED.
C. Fever greater than 100.4°F.
D. Evaluate fetal movement and daily fetal kick counts. If client reports decreased fetal movement, send to the ED to have BPP and NST to evaluate fetal well-being.
E. Headaches with or without blurry vision need to be addressed. BP should be monitored; if client has elevated BP with or without headaches and blurry vision, they need to be sent to the ED.
F. Leaking of clear fluid from the vagina needs to be assessed; if client has unexplained fluid from the vagina, they needs to go to ED.
G. Pruritus on hands and feet causing the client to wake up at night; client needs to be evaluated and sent to the ED.

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POSTPARTUM OVERVIEW

A. The American College of Obstetricians and Gynecologists (ACOG) has released new recommendations for postpartum care, including developing a postpartum care plan prenatally; scheduling an initial postpartum contact in the first 3 weeks to address acute issues followed by a comprehensive visit no later than 12 weeks after delivery; and counseling those who had gestational diabetes, pregnancy-related hypertension, or a preterm birth (PTB) about their increased risk for cardiovascular disease. They also advise documenting pregnancy complications in the electronic medical record to inform future providers regarding screening for diabetes and risk assessment for cardiovascular disease.

PHYSICAL EXAMINATION

A. General assessment:
  1. Vital signs.
  2. Weight.
B. Breasts:
  1. Issues with breastfeeding: discomfort or pain associated with breastfeeding; adequate breast milk production; assess comfort and confidence with breastfeeding.
  2. Engorgement: breast fullness and firmness, which is accompanied by pain and tenderness. Primary engorgement is caused by interstitial edema and onset of copious milk production. It typically occurs between 24 and 72 hours postpartum, with a normal range of 1 to 7 days; ▶

peak symptomatology averages 3 to 5 days postpartum. Secondary engorgement typically occurs later if the mother's milk supply exceeds the amount of milk removed by their infant. Breast engorgement usually resolves spontaneously over a few days, but supportive care (warm compress or a warm shower before feeding to enhance let-down and facilitate milk removal, cool compress after or between feedings, mild analgesics such as acetaminophen or ibuprofen) is appropriate. In those who are not breastfeeding, the use of a tight brassiere and avoidance of breast stimulation suppress lactation in 60% to 70% of clients and are the recommended treatment.

3. Mastitis: Lactation mastitis typically presents as a firm, red, and tender area of one breast with a maternal temperature that exceeds 38.5°C. A tender, fluctuant area is more indicative of an abscess. Systemic complaints may be present and include myalgia, chills, malaise, and flulike symptoms. Differential diagnoses include plugged ducts, galactocele, and in rare cases inflammatory breast cancer.

C. Heart:

1. Postpartum physiologic changes in the cardiovascular system are particularly important in clients with underlying cardiac disease. The cardiac output and stroke volume increase by approximately 50% and 70%, while the heart rate decreases by 15% and blood pressure (BP) remains unchanged. The increases in stroke volume and cardiac output most likely result from improved cardiac preload from autotransfusion of utero placental blood to the intravascular space. As the uterus decompresses following delivery, a reduction in the mechanical compression of the vena cava allows for further increases in cardiac preload.

D. Abdomen:

1. Uterine involution: The uterus recedes by approximately 1 cm/d to lie midway between the symphysis pubis and umbilicus by the end of the first postpartum week. It is not palpable abdominally by 2 weeks postpartum and attains its normal nonpregnant size by 6 to 8 weeks postpartum.

2. Abdominal wall: The abdominal wall is lax postpartum and will regain its normal muscular tone over several weeks; however, separation (diastasis) of the rectus abdominis muscles may persist.

E. Skin:

1. Linea nigra will fade gradually, but may never completely disappear.

2. Striae fade from red to silvery by 6 to 12 months postpartum, but are permanent.

3. Abdominal skin may remain lax if extensive rupture of elastic fibers occurred during pregnancy.

4. Melasma gravidarum resolves spontaneously over a period of months.

F. Extremities:

1. Edema and varicose veins: Varicose veins may appear during the antepartum or postpartum period. Leg elevation, exercise, and compression therapy improve oxygen transport to the skin and subcutaneous tissues, decrease edema, reduce inflammation, and compress dilated veins.

2. Deep vein thrombosis (DVT) is more common in postpartum females than in antepartum and nonpregnant females. The risk is highest in the first few weeks postpartum and then gradually declines to baseline by 12 weeks postpartum. Risk factors include a previous DVT, thrombophilia, sickle cell disease, obesity, smoking, Cesarean delivery, and postpartum hemorrhage.

G. Vagina:

1. The vagina slowly contracts and decreases in size postpartum, but never completely returns to its nulligravid size.

2. The vaginal walls appear smooth immediately postdelivery; rugae are restored in the third week as edema and vascularity subside.

3. Pelvic floor stretching and trauma during childbirth result in pelvic muscle relaxation, which may not return to the pregravid state.

4. Persistent vaginal bleeding: Vaginal bleeding that persists for more than approximately 8 weeks after delivery is unusual and may be caused by infection, retained products of conception, a bleeding diathesis, or rarely choriocarcinoma or a uterine vascular anomaly. A temporary increase in bleeding at this time may represent menses; in such cases, bleeding should stop within a few days.

5. Vaginal dryness: Symptoms of vaginal dryness can be managed by regular use of vaginal moisturizing agents with supplemental use of vaginal lubricants for sexual intercourse.

6. Surgical site infection (episiotomy, laceration, abdominal incision): Perineal infections and subsequent breakdown of previously repaired lacerations or episiotomies are usually localized to the skin and subcutaneous tissue. On examination, the area appears swollen and erythematous with a purulent exudate. Treatment consists of opening the wound, drainage, irrigation, and debridement of foreign material and necrotic tissue. Antibiotics are not necessary unless there is accompanying cellulitis. The area will heal by granulation, but large defects may be resutured when the wound surface is free from exudate and covered by pink granulation tissue.

H. Cervix:

1. The cervix remains 2 to 3 cm dilated for the first few postpartum days and is less than 1 cm dilated at 1 week.

2. The external os never resumes its pregravid shape; the small, smooth, regular circular opening of the nulligravida becomes a transverse, stellate slit after childbirth.

3. Histologically, the cervix does not return to baseline for up to 3 to 4 months postpartum.

I. Lochia:

1. Lochia rubra: normal shedding of blood and decidua is a red/red-brown vaginal discharge that lasts 3 to 5 days following delivery.

2. Lochia serosa: a watery, pinkish brown discharge that lasts for 2 to 3 weeks.

3. Lochia alba: discharge turns yellowish white in the final stage and may last 4 to 6 weeks.

J. Rectal:

1. Symptomatic hemorrhoids are common postpartum, occurring in approximately one-third of females. The treatment approach depends on the specific symptoms: pruritus, bleeding, pain, or prolapse.

2. Postpartum incontinence of urine, flatus, or feces is common in the immediate postpartum period and generally improves over the subsequent weeks, but may persist long term. Females should be asked about incontinence as they may not bring the issue up themselves.

COMMON DIAGNOSTIC TESTS

A. Complete blood count (CBC) with platelets.

B. Two-hour postload glucose (clients with gestational diabetes mellitus [GDM]).

PLAN

A. Pharmaceutical therapy:

1. All household members in the newborn's home should also have up-to-date immunizations to create a protective "cocoon" around the infant and thus minimize newborn exposure to infection; Tdap and influenza vaccines are particularly important in this respect.

B. Client teaching:

1. Weight loss:

a. The mean weight loss from delivery of the fetus, placenta, and amniotic fluid is 13 lbs.

b. Contraction of the uterus and loss of fluid (lochia) and excess intra- and extracellular fluid lead to an additional loss of 5 to 15 lbs in the first 6 weeks postdelivery.

c. Approximately one-half of gestational weight gain is lost in the first 6 weeks after delivery, with a slower rate of loss through the first 6 months postpartum.

d. Breastfeeding mothers may have differing weight loss as they require 500 calories more a day over pregnancy daily caloric requirements.

2. Activity:

a. There are no evidence-based data on which to base recommendations regarding postpartum physical activity. In particular, there are no high-quality data on which to base restrictions on lifting, climbing stairs, bathing, swimming, driving, or resuming vaginal intercourse, exercise, or work after delivery.

3. Depression:

a. Screen all clients for postpartum depression, as recommended by ACOG, the American Academy of Pediatrics, and the U.S. Preventive Service Task Force Recommendation Statement. The validated questionnaire most commonly used for screening pregnant and postpartum females is the Edinburgh Postnatal Depression Scale (EPDS), but other validated tools can be used. Screening is important because postpartum depression is common, the combination of screening and adequate support improves clinical outcomes, and treatment (especially with cognitive behavioral therapy) is associated with symptom remission. Depression screening is performed at the routine postpartum visit, but consideration should be given to additional earlier screening and follow-up in clients at high risk for depression.

4. Intimate partner violence:

a. Screen all postpartum individuals for intimate partner violence. Ideally, screening should be performed at the initial prenatal visit, at least once per trimester, and again during postpartum care. Screening should be routinely performed for those who have not accessed prenatal care or who have concerning signs or symptoms.

5. Substance use:

a. Screen for tobacco use; counsel regarding relapse risk in the postpartum period.

b. Screen for substance use disorder and refer as indicated.

COMPLICATIONS

A. Postpartum depression.

B. Diabetes: Approximately 6 to 8 weeks after delivery, clients with GDM should undergo an oral glucose tolerance test (OGTT). They should be counseled about the increased risk of future diabetes, even when the postpartum glucose tolerance test is normal. These individuals may benefit from lifestyle changes, such as weight loss if they are obese.

C. Complications that occurred during the pregnancy or postpartum should be reviewed in terms of the cause, risk of recurrence, and prevention.

CONSULTATION

A. Behavioral health counseling for postpartum depression.

B. Any underlying medical conditions that were present prior to or during the pregnancy should be addressed at the postpartum visit.

C. Cardiovascular risk: Clients with gestational diabetes, pregnancy-related hypertension, or PTB are at increased risk of future cardiovascular disease, even if postpartum BP and glucose tolerance testing are normal.

D. ACOG recommends that clinicians make individuals with these complications of pregnancy aware of their increased risk for developing cardiovascular disease. Including this information in their health record can facilitate ongoing monitoring and risk assessment. For those with preeclampsia, this may include yearly assessment of BP, lipids, fasting blood glucose, and body mass index (BMI). Increased awareness about their cardiovascular risk may increase the client's motivation to reduce modifiable risk factors, if present, by adopting a healthy lifestyle.

EMERGENT ISSUES/INSTRUCTIONS

A. Severe abdominal pain and/or afebrile with temperature above 100.4°F need to be evaluated in the ED.

B. Excessive vaginal bleeding soaking one pad per hour after the first 3 to 5 days after delivery needs to be evaluated in the clinic. If the client complains of weakness, cold intolerance, dizziness, nausea, and rapid heart rate, they need to go to the ED.

C. Foul-smelling drainage from the vagina and/or incision needs to be evaluated in the clinic.

D. Separation of wound or incision needs to be evaluated in the ED.

E. Elevated BP with complaints of headaches and/or blurry vision needs to be evaluated for preeclampsia in the ED.

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ANEMIA, IRON-DEFICIENCY

DEFINITION

A. The Centers for Disease Control and Prevention (CDC) and American College of Obstetricians and Gynecologists (ACOG) define first- and third-trimester anemia as a hemoglobin (Hgb) of less than 11.0 g/dL and hematocrit (Hct) of less than 33%, and second-trimester anemia as an Hgb of less than 10.5 g/dL and Hct of less than 32%.

INCIDENCE

A. Anemia is a common medical complication of pregnancy. Iron-deficiency anemia constitutes 75% to 95% of pregnancy-related anemias. Although the exact percentage of iron-deficiency anemia in pregnancy is difficult to distinguish, there is an estimated prevalence of 21.55 per 1,000 individuals with Hgb of less than 10g/dL. Prevalence of anemia is twice as high in non-Hispanic Black pregnancies and even higher in teens of all races. The prevalence of anemia during pregnancy increases with each trimester.

PATHOGENESIS

A. Physiologic changes of pregnancy result in an increased maternal blood volume. This increased demand for iron results in Hgb and Hct decrease during the first and second trimesters due to a greater expansion of plasma volume relative to the increase in red blood cell (RBC) mass, and usually increase during the third trimester when plasma expansion has ceased

B. During most pregnancies, diet alone does not provide the necessary iron; however, in lactation, less elemental iron is needed. In clients 19 years and older, 9 mg/d is needed and in adolescents 10 mg/d is recommended.

PREDISPOSING FACTORS

A. Failure to take oral iron; often due to inability to tolerate oral iron supplements.

B. Multiple gestation; increases iron requirement and may contribute to increased blood loss at delivery.

C. Diet high in phosphorus or foods such as tea, coffee, milk, or soy.

D. Low iron and protein diet, eating nonfood items (pica).

E. Not eating foods that help with absorption of iron (orange juice, broccoli, strawberries).

F. History of gastrointestinal surgery may cause iron malabsorption (e.g., gastrectomy).

G. Chronic bleeding during pregnancy (e.g., placenta previa, marginal sinus separation of placenta, hemorrhoidal bleeding).

H. Short intervals between pregnancies.

I. Race: non-Hispanic Black females.

J. Age: teenage females.

K. High altitude and smoking can cause an increase in Hgb and Hct; consider this when interpreting results.

L. Heavy menses.

COMMON COMPLAINTS

A. Fatigue, irritability, dizziness.

B. Inability to take prenatal vitamins due to nausea.

C. Pica.

OTHER SIGNS AND SYMPTOMS

A. Pale mucous membranes and skin.

B. Tachycardia/palpitations.

SUBJECTIVE DATA

A. Elicit the onset, duration, and course of presenting symptoms.

B. Elicit information about the client's "typical" dietary intake for meals and snacks, and review pica (eating clay, starch, ice, and other nonnutritive substances).

C. Review the client's intake of prenatal vitamins and supplemental iron. How often do they take iron? Elicit the reason for skipping the supplemental iron (nausea, constipation), if applicable.

D. Review the client's history of gastrointestinal surgeries, irritable bowel syndrome (IBS), celiac disease, and crohn's disease.

E. Review the client's history for any type of anemia and previous treatment, including blood transfusions.

F. Review pregnancy history for closely spaced pregnancies (two in a calendar year) and multiple gestation.

G. Review the client's intake of medications for use of aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs).

PHYSICAL EXAMINATION

A. Check pulse and blood pressure (BP): Note postural hypotension and tachycardia.

B. Inspect: general appearance:

1. Inspect the skin, mucous membranes, and conjunctivae for pallor.

2. Observe the mouth and tongue: Note atrophy of papillae and smooth, beefy red appearance of tongue with anemia.

3. Note dryness of skin. Inspect texture of nails (brittle, spoon-shaped, concave); inspect the hair for brittleness.

C. Palpate:

1. Palpate the abdomen for masses; assess fundal height if pregnant.

D. Auscultate:

1. Auscultate the heart for systolic flow murmurs; auscultate lungs.

DIAGNOSTIC TESTS

A. Complete blood count (CBC): Hgb and Hct:

1. First trimester: less than 11 g/dL Hgb or less than 33% Hct.

2. Second trimester: less than 10.5 g/dL Hgb or less than 32% Hct.

3. Third trimester: less than 11 g/dL Hgb or less than 33% Hct.

B. Peripheral blood smear: Note microcytic and hypochromic RBCs on peripheral smear.

C. Sickle cell screen, if applicable.

D. Serum iron: low with anemia.

E. Iron-binding capacity: high iron-binding capacity with anemia.

F. Transferrin: saturation less than 15%.

G. Stool for occult blood, if applicable.

H. Emesis for presence of blood, if applicable.

DIFFERENTIAL DIAGNOSES

A. Iron-deficiency anemia.

B. Normal physiologic anemia of pregnancy: During normal pregnancy, concentrations of erythrocytes and Hgb usually fall due to the greater increase in plasma volume (increased by 40%–50%) relative to the increase in erythrocyte volume (increased by 15%–25%).

C. Megaloblastic anemia: This condition is commonly associated with iron-deficiency anemia and is rarely seen alone.

D. Hemolytic anemia: sickle cell anemia, thalassemia, hereditary spherocytosis, and erythrocyte enzyme deficiency.

E. Aplastic anemia: bone marrow failure.

F. Hematologic malignancies: leukemia and lymphoma.

G. Clotting factor or other hemostatic deficiencies: von Willebrand disease, idiopathic thrombocytopenia (ITP), and disseminated intravascular coagulation (DIC).

H. Pica.

PLAN

A. General interventions:

1. Perform initial evaluation of Hgb and Hct at first prenatal visit; repeat at 24- to 28-week blood draw with diabetes testing.

2. Diet counseling and nutrition consultation. Provide a list of iron-rich foods.

3. The client may be eligible for the Women, Infants, and Children (WIC) program, which provides supplemental foods for pregnant persons and young children. Ask your local health department for information available in your community:

a. Advise the client to take supplemental iron in addition to prenatal vitamins. If they are unable to tolerate prenatal vitamins, suggest a children's chewable vitamin, two tablets daily.

b. Encourage the client to continue iron supplementation through the first month postpartum and throughout breastfeeding. Add a stool softener to the daily regimen if constipation develops.

4. Smoking cessation.



B. Client teaching: *See Client Teaching Guide for this chapter, "Iron-Deficiency Anemia (Pregnancy)."*

C. Pharmaceutical therapy:

1. The CDC recommends that all pregnant clients start iron supplementation at the first prenatal visit. Prophylaxis: Increase daily elemental oral iron supplements from 15 to 30 mg/d in pregnancy to prevent iron-deficiency anemia.

2. Most prenatal vitamin formulations contain 30 mg of elemental iron per day; however, if the client has iron-deficiency anemia, they need to increase their elemental iron by 30 to 120 mg/d. Nausea and vomiting occur in 20% to 25% of clients. These side effects are dose-related. Have the client alter times of administration of the iron supplement to determine when the iron is best tolerated.

3. Treatment: With iron-deficiency anemia, three times the prophylactic dose of iron should be given. Instruct the client to take the iron concurrently with orange juice. Vitamin C increases absorption of iron.

4. Intramuscular (IM) or intravenous (IV) iron may be ordered for the small proportion of clients who do not tolerate oral iron due to gastrointestinal complaints, malabsorption syndrome, or noncompliance with the oral iron regimen.

5. Blood transfusion may be needed per institute guidelines.

FOLLOW-UP

A. Carry out routine prenatal and postpartum follow-up care. When the client begins taking the recommended dose of supplemental iron, the RBC response can be measured in 2 weeks by an elevation in their reticulocyte count.

B. Repeat Hct after 4 to 6 weeks of therapy.

C. If no improvement is seen in reticulocyte count or Hct after 4 weeks of therapy and the client has been compliant, another cause of anemia should be investigated.

CONSULTATION/REFERRAL

A. Consider consult with a physician if Hgb is less than 9 g/dL or Hct is less than or equal to 27% and does not improve with the previously noted treatments.

B. Dietitian.

C. WIC.

D. Social services.

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GESTATIONAL DIABETES MELLITUS

DEFINITION

A. Gestational diabetes mellitus (GDM) is a carbohydrate intolerance with onset or recognition during pregnancy.

INCIDENCE

A. It is estimated that up to 6% to 7% of pregnancies are complicated by diabetes mellitus (DM) and approximately 86% of these cases represent individuals with GDM. Additionally, GDM prevalence is related to the prevalence of type 2 diabetes in any ethnic population.

PATHOGENESIS

A. Insulin antagonism caused by the placental hormones leads to gestational diabetes. As greater amounts of these hormones are produced with advancing gestation, the diabetogenic effect of pregnancy becomes more pronounced, reaching significant levels in the second trimester. Those with GDM are at risk for later development of type 1 and type 2 diabetes, metabolic syndrome, and cardiovascular disease. GDM may actually be the expression of pregnancy-induced stresses on carbohydrate metabolism in the genetically predisposed client. It is estimated that 50% to 70% of females with GDM will develop DM within approximately 15 to 25 years of the pregnancy. Some individuals have undiagnosed type 1 or 2 diabetes prior to pregnancy.

PREDISPOSING FACTORS

A. Members of any of the following ethnic groups:

1. Hispanic American.

2. African American.

3. Native American.

4. South or East Asian.

5. Pacific Islander.

B. Maternal age older than 25 years.

C. Obesity (body mass index [BMI] >30).

D. Family history of diabetes, first-degree relatives.

E. Previous birth of a macrosomic, malformed, or stillborn baby.

F. Comorbidity: metabolic syndrome, hypertension, polycystic ovarian syndrome, use of glucocorticoids.

G. Glycosuria at prenatal visit.

H. Gestational diabetes in a previous pregnancy.

I. Multiple gestation.

J. Sedentary lifestyle.

COMMON COMPLAINTS

A. Common complaints of hyperglycemia include polydipsia, polyuria, nocturia, fatigue, and blurred vision. However, gestational diabetes is often asymptomatic.

OTHER SIGNS AND SYMPTOMS

A. Glycosuria.
B. Fundal height measurement greater than gestational age in weeks.
C. Frequent candidal infections.
D. Rapid weight gain.

POTENTIAL COMPLICATIONS

A. Ketoacidosis:
  1. May develop in GDM.
  2. More common in insulin-dependent diabetes.
  3. May develop with glucose levels as low as 200 mg/dL.
  4. May be present in an undiagnosed diabetic receiving beta-mimetic agents (such as terbutaline) for tocolysis or steroids to enhance fetal lung maturity. **Fetal mortality rate is 10% in individuals who come to the hospital in diabetic ketoacidosis (DKA). Glucose and ketones cross the placenta.**
  5. Therapy hinges on timely, aggressive volume resuscitation, insulin administration, and correction of maternal electrolyte imbalance.
B. Polyhydramnios.
C. Increased risk for neonatal morbidity such as hypoglycemia, hyperbilirubinemia, polycythemia, respiratory distress caused by delayed lung maturity, and/or traumatic birth injury related to shoulder dystocia, which is associated with gestational diabetes.
D. Increased risk for stillbirth: Risk is related primarily to poor glycemic control.
E. Preeclampsia.
F. Operative delivery.
G. Fetal organomegaly.
H. Long-term sequelae for the offspring: abnormal glucose tolerance, metabolic syndrome, and obesity.

SUBJECTIVE DATA

A. Review pregnancy history for two or more spontaneous abortions, previous stillbirths, or unexplained neonatal deaths.
B. Review birth weight (macrosomia) and gestational age of previous children.
C. Review pregnancy history for polyhydramnios and/or congenital anomalies.
D. Review the client's history for a predisposition to infections, especially urinary tract infections (UTIs) and candidal vaginitis, and for family history of diabetes.
E. Review pregnancy history for gestational diabetes, diet restrictions, and need for insulin therapy.
F. Review for history of polycystic ovary syndrome.
G. Review physical activity level.

PHYSICAL EXAMINATION

A. Check blood pressure (BP), pulse, and weight.
B. Inspect:
  1. Perform speculum exam to rule out sexually transmitted infections (STIs), bacterial vaginosis, and candidal vaginitis, only if indicated.
C. Palpate:
  1. Check the client's fundal height each visit after 20 weeks.
D. Auscultate:
  1. Fetal heart tones after 10 to 12 weeks' gestation.

SCREENING/DIAGNOSTIC TESTS

A. Clients who have risk factors for type 2 diabetes should be screened at the initial prenatal visit.
B. Perform "one-step" 2-hour 75-g (fasting) oral glucose tolerance test (OGTT) or "two-step" 1-hour 50-g (nonfasting) followed by a 3-hour 100-g (fasting) OGTT for positive results.
C. Blood glucose measurements (fasting blood sugar [FBS] and 2-hour postprandial blood glucose values).
D. Hgb A1C.
E. Urine dipstick for glucose: Early glycosuria needs further evaluation (e.g., Hgb A1C, urine culture, random glucose "finger stick").
F. Ultrasonography: For those with gestational diabetes controlled by diabetes alone, with no other comorbidities, there are no current recommendations for ultrasound (US) prior to 40 weeks' gestation. For those with gestational diabetes controlled by medication, with no other comorbidities, US surveillance once or twice weekly may be considered at 32 weeks' gestation. Twice weekly US surveillance may be considered at 32 weeks' gestation for those with poorly controlled gestational diabetes.

DIFFERENTIAL DIAGNOSES

A. Gestational diabetes:
  1. A1: diet-controlled gestational diabetes (A1 GDM).
  2. A2: insulin-treated gestational diabetes (A2 GDM).
  3. Polyhydramnios.
  4. Fetal macrosomia.

PLAN

A. General interventions:
  1. The American Diabetes Association (ADA) recommends that all pregnant females be screened:
    a. Diabetes mellitus screen (DMS): "Two-step" 1-hour 50-g (nonfasting) oral glucose challenge test (OGCT) followed by a 3-hour 100-g OGTT for positive results:
      i. Administer 50-g oral glucose load (fasting not required; however, no food/nonwater beverages for 2 hours prior to the test).
      ii. Draw blood for glucose assessment 1 hour after glucose load is given.
      iii. Typically performed between 24 and 28 weeks' gestation; performed earlier if the client has glycosuria, predisposing risk factors, or advanced maternal age (AMA), or if fetal size is greater than average for gestational date by fundal height measurement.
      iv. Abnormal result of the 1-hour 50-g OGCT is a glucose level of 130 to 140 mg/dL (use your institutional limits).
      v. If the results of the 1-hour 50-g OGCT is ≥200 mg/dL, the client skips the 3-hour 100-g OGTT and begins dietary modifications and glucose evaluation for insulin needs.
      vi. Follow up all abnormal 1-hour OGCT with a 3-hour OGTT.
    b. 3-hour OGTT:
      i. Draw fasting blood glucose first.
      ii. Administer 100-g glucose load. ▶

iii. Draw blood for glucose assessment after 1 hour, 2 hours, and 3 hours from the time the glucose load is given.

iv. Plasma or serum glucose results (Carpenter–Coustan criteria or use institutional limits):

1) Fasting = 95 mg/dL.
2) 1 hour = 180 mg/dL.
3) 2 hours = 155 mg/dL.
4) 3 hours = 140 mg/dL.

v. If two or more values of 3-hour glucose tolerance test (GTT) are elevated:

1) Refer the client for nutritional counseling for dietary modifications.
2) Glucose evaluation for insulin needs. Daily blood sugar log to record fasting and 2-hour postprandial blood glucose measurements.

c. "One-step" 2-hour 75-mg (fasting) OGTT may be performed instead of the "two-step" approach.

2. Antepartum testing:

a. For clients with well-controlled A1 GDM on nutritional therapy and no other pregnancy complication, there is no national consensus with respect to criteria for initiation and timing of testing.

b. Clients with insulin-dependent diabetes or using oral hyperglycemic agents, or with poorly controlled A2 GDM, need either weekly or twice weekly nonstress test (NST) and biophysical profile (BPP) beginning at 32 weeks.

3. Serial ultrasonography:

a. Evaluate fetal growth, estimate fetal weight, and detect polyhydramnios and malformations.

b. There is no national consensus with respect to criteria for initiation and timing to assess growth; however, practice agrees on one single US at 36 weeks or starting US at 28 weeks and repeating them thereafter, every 4 weeks (28, 32, and 36 weeks).

c. Macrosomia is a leading risk factor for birth injuries due to shoulder dystocia at vaginal delivery for those with GDM. Clients with GDM should be counseled regarding the option of a scheduled Cesarean delivery when the fetus is more than or equal to 4,500 g.

4. Neonate evaluation:

a. Monitor neonate carefully for hypoglycemia, especially if the client has a history of insulin-dependent diabetes.

b. Assess for birth injury.

5. Postpartum:

a. Testing:

i. Two-hour GTT between 6 and 12 weeks postpartum (fasting):

1) Administer 75-g glucose load.
2) Draw blood for glucose assessment at 2 hours after the glucose load is given.
3) Results ≥200: Diabetes management is needed.

ii. Consider serial measurement of total cholesterol, low-density lipoprotein, high-density lipoprotein, and triglycerides.

b. Contraception:

i. Low-dose oral contraceptives (OCs) may be used in clients with GDM who do not have other risk factors.

ii. Rate of subsequent diabetes in OC users is not significantly different from those who do not use OCs.

B. Client teaching: *See Client Teaching Guide for this chapter, "Gestational Diabetes."*

1. GDM requires intensive client and family education to maintain glucose targets to help reduce perinatal complications:

a. Exercise:

i. If the client had an active lifestyle prior to pregnancy, encourage them to continue a program of exercise approved for pregnancy such as walking or swimming for 20 minutes per day.

ii. Upper extremity exercise and walking in previously sedentary individuals with GDM may improve glycemic control.

b. Instruct the client on self-monitoring blood glucose and encourage them to record each blood glucose measurement:

i. Have client take measurements fasting and 2 hours postprandially every day and record the measurements. Postprandial blood sugar values are recommended over preprandial values due to better glycemic control and better maternal/fetal outcomes. FBS should be less than 95 mg/dL. The 1-hour postprandial values should be less than 140 mg/L and the 2-hour postprandial values should be less than 120 mg/L. The Hgb A1C goal is less than 6%. Nighttime levels should not decrease lower than 60 mg/dL.

ii. If the client is taking multiple doses of insulin, they may need to take measurements more frequently.

C. Dietary management: Place the client on a diet that is prescribed for preexisting diabetes in pregnancy, such as the ADA diet, in the following amounts:

1. Current weight less than 80% ideal body weight (IBW): 35 to 40 kcal/kg/d.

2. Current weight 80% to 120% IBW: 30 kcal/kg/d.

3. Current weight 120% to 150% IBW: 22 to 25 kcal/kg/d.

4. Current weight more than 150% IBW: 12 to 14 kcal/kg/d (minimum 1,800 cal/d to prevent ketosis).

5. Dietary consumption:

a. Total calories: 1,800 to 2,500 kcal/d based on IBW.

b. Trimester specific recommendations:

i. First trimester no increase in calories.

ii. Second trimester 340 kcal/d increase above prepregnancy levels.

iii. Third trimester 452 kcal/d increase above prepregnancy levels.

c. Nutrition distribution:

i. Carbohydrate 40% to 50% (minimum 175 g/d).

ii. Protein 20% (71 g/d).

iii. Fat 30% to 40%.

iv. Fiber 28 g/d.

D. Pharmaceutical therapy:

1. Insulin therapy is the drug of choice if medical nutritional therapy does not consistently maintain fasting glucose levels of less than 95 mg/dL. One-hour postprandial values should be less than 140 mg/dL. Two-hour postprandial values should be less than 120 mg/dL (Figure 16.1).

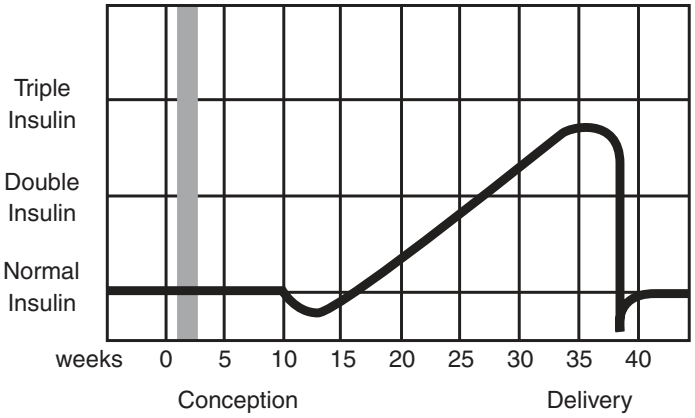


FIGURE 16.1 Insulin requirements during pregnancy.

2. Oral antihyperglycemic medications (glyburide and metformin) are being increasingly used in females with GDM, although they are not yet approved by the U.S. Food and Drug Administration (FDA) for this indication. Glyburide results in increased insulin secretion and insulin sensitivity at the tissue level. Metformin inhibits hepatic gluconeogenesis and glucose absorption and stimulates glucose uptake in peripheral tissues.

3. In general, insulin therapy in pregnancy is no different from vigorous management of diabetes with insulin in nonpregnant females. Insulin does not cross the placenta.

4. Therapy must respond to the changing insulin requirements during pregnancy; typical starting dosage is 0.7 to 2.0 units/kg/d in divided doses based on current pregnancy weight:

a. Individuals with relatively simple insulin programs may require more complex regimens as the pregnancy progresses.

5. Intensive insulin therapy (as opposed to conventional therapy) is often required in pregnancy.

6. Human insulin (Humulin) is preferred to animal or synthetic insulin.

7. Instruct clients to report glucose values on at least a weekly basis. Secure client portals can be used to upload values, which will be sent to their provider. Alternatively, clients may opt to fax or telephone in their values.

8. The blood glucose values should be evaluated at least weekly to adjust to the client's changing needs.

FOLLOW-UP

A. Weekly evaluation of blood glucose values.

B. Antepartum testing consisting of NSTs and BPPs.

C. Fetal growth US.

D. For females who develop diabetes during pregnancy, give the client 75-g glucose load to evaluate for the development of type 2 diabetes at the return postpartum visit at 6 weeks after delivery. These clients should be screened at least every 3 years from then on. Encourage lifestyle changes to prevent the development of diabetes.

E. Contraception.

CONSULTATION/REFERRAL

A. Consult or comanage the client with an obstetrician as indicated and if GDM is not controlled by diet and exercise.

B. Refer to a dietitian for nutritional consult for carbohydrate counting and teaching the client dietary and lifestyle changes needed for tight glucose control.

C. Social services if indicated.

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HYPERTENSIVE DISORDERS

DEFINITION

A. Preeclampsia, also called pregnancy-induced hypertension (PIH; antiquated term *toxemia*), is the new onset of hypertension with proteinuria (300 mg or more of protein in a 24-hour specimen or a protein/creatinine ratio of 0.3 or higher) that develops after 20 weeks' gestation in pregnancy or develops up to 6 weeks postpartum in a previously normotensive female. In the absence of proteinuria, any of the following can establish the diagnosis: new-onset thrombocytopenia, impaired liver function, renal insufficiency, pulmonary edema, or visual or cerebral disturbances.

B. Preeclampsia with severe features: In 2013, the American College of Obstetricians and Gynecologists (ACOG) replaced the term *severe preeclampsia* with the term *preeclampsia with severe features.* In a client with preeclampsia, the presence of one or more of the following indicates a diagnosis of "preeclampsia with severe features":

1. Symptoms of central nervous system (CNS) dysfunction:

a. New-onset cerebral or visual disturbances, such as:

i. Photopsia, scotomata, cortical blindness, retinal vasospasm.

ii. Severe incapacitating headache ("the worst headache in my life") or a headache that persists and progresses despite analgesic therapy.

iii. Altered mental status.

2. Hepatic abnormality:

a. Severe persistent right upper quadrant (RUQ) or epigastric pain unresponsive to medication and not accounted for by an alternative diagnosis or a serum transaminase concentration greater than 2 times the upper limit of the upper range, or both.

3. Severe blood pressure (BP) elevation:

a. Systolic BP greater than 160 mmHg or diastolic BP greater than 110 mmHg on two or more occasions at least 4 hours apart. Antihypertensive therapy may be initiated upon confirmation of severe hypertension in which case criteria for severe BP elevation can be satisfied without waiting until 4 hours have lapsed.

4. Thrombocytopenia:

a. Platelets less than 100,000/μL. ▶

5. Renal abnormality:
   a. Progressive renal insufficiency (serum creatinine >1.1 mg/dL [97.2 µmol/L] or a doubling of the serum creatinine concentration in the absence of other renal disease).
6. Pulmonary edema.

C. Hypertension: The 2017 changes in the definition for elevated BP and hypertension in nonpregnant adults do not affect the diagnostic criteria for pregnancy-related hypertensive disorders:

1. Either a systolic BP more than 140 mmHg or a diastolic BP more than 90 mmHg, or both. The values must be elevated on at least two separate occasions at least 4 hours apart. **Severity of hypertension is not necessarily associated with the severity of preeclampsia.**
2. Eclampsia is the development of grand mal seizures in a client with preeclampsia in the absence of other neurologic conditions that could account for the seizures.
3. HELLP syndrome: HELLP = hemolysis, elevated liver enzymes, and low platelets. It probably represents a severe form of preeclampsia, but this relationship is controversial; HELLP may be an independent disorder.
4. Chronic hypertension in pregnancy is often complicated by superimposed preeclampsia.
5. Gestational hypertension (GHTN) is hypertension without proteinuria or other signs and symptoms of preeclampsia or related end-organ dysfunction that develops after 20 weeks' gestation and resolves by 12 weeks postpartum. Up to 50% of females with GHTN may develop proteinuria.

INCIDENCE

A. Hypertensive disorders are the most common medical complication of pregnancy, with a reported incidence of up to 10% worldwide. Incidence varies among different regions and countries. The incidence of preeclampsia has increased by 25% in the United States between 1987 and 2004, and 2% to 8% of all pregnancies worldwide are complicated by preeclampsia. The risk of recurrent preeclampsia is between 5% and 70%. Individuals who develop severe features of preeclampsia prior to 30 weeks' gestation have the highest risk of preeclampsia in future pregnancies.

B. Prognosis:

1. Those with preeclampsia are at an increased risk for life-threatening obstetric or medical complications. Worldwide, 10% to 15% of direct maternal deaths (resulting from obstetric complications of pregnancy) are associated with preeclampsia/eclampsia. In the United States, preeclampsia/eclampsia is one of the four leading causes of maternal death, along with hemorrhage, cardiovascular conditions, and thromboembolism. There is approximately one maternal death caused by preeclampsia/eclampsia per 100,000 live births, with a case-fatality rate of 6.4 deaths per 10,000 cases.
2. Fetal implications: Preeclampsia can lead to intrauterine growth retardation (IUGR) and oligohydramnios, as well as medically or obstetrically indicated preterm birth (PTB). As a result, perinatal morbidity and mortality are increased.

C. Prevention:

1. Low-dose aspirin: This reduces the frequency of preeclampsia, as well as related adverse pregnancy outcomes (PTB, growth restriction), by about 10% to 20% when given to females at moderate to high risk of the disease; it has an excellent maternal/fetal safety profile in pregnancy.
2. Candidates: There is no consensus on the criteria that confer high risk for developing preeclampsia.
3. U.S. Preventive Services Task Force (USPSTF) criteria for high risk include one or more of the following (the incidence of preeclampsia is estimated to be at least 8% in a pregnant individual with one of these risk factors):
   a. Previous pregnancy with preeclampsia, especially early onset and with an adverse outcome.
   b. Multifetal gestation.
   c. Chronic hypertension.
   d. Type 1 or 2 diabetes mellitus (DM).
   e. Chronic kidney disease.
   f. Autoimmune disease (antiphospholipid syndrome, systemic lupus erythematosus).
4. The USPSTF criteria for moderate risk include the following (the incidence of preeclampsia is estimated to be less than 8% in a pregnant individual with only one of these risk factors):
   a. Nulliparity.
   b. Obesity (body mass index [BMI] >30 kg/m).
   c. Family history of preeclampsia in mother or sister.
   d. Age ≥35 years.
   e. Sociodemographic characteristics (African American race, low socioeconomic level).
   f. Personal risk factors (previous pregnancy with low birth weight or small for gestational age infant, previous adverse pregnancy outcome [fetal demise], interval >10 years between pregnancies).

D. The following summaries represent the recommendations of some national organizations:

1. The American Heart Association and the American Stroke Association recommend low-dose aspirin for females with chronic primary or secondary hypertension or previous pregnancy-related hypertension for prevention of preeclampsia-related stroke.
2. The USPSTF recommends the use of low-dose aspirin 81 mg/d in individuals at high risk of developing preeclampsia to reduce the risk for preeclampsia, PTB, and fetal growth restriction. Those with ≥1 high-risk factors should receive low-dose aspirin. For individuals with multiple moderate risk factors, the benefit of aspirin therapy is less clear, so clinicians should use clinical judgment and talk with their clients about the benefits and harms of low-dose aspirin use. Aspirin should be initiated between 12 and 28 weeks of gestation.
3. In July 2016, ACOG endorsed the USPSTF recommendation for use of low-dose aspirin 81 mg/d in individuals at high risk of developing preeclampsia and based high-risk status on the same high-risk factors designated by the USPSTF.
4. The World Health Organization recommends the use of low-dose aspirin 75 mg/d for high-risk females (history of preeclampsia, diabetes, chronic hypertension, renal or autoimmune disease, or multifetal pregnancies).

PATHOGENESIS

A. The etiology of preeclampsia is unknown, although several theories exist. Generalized vascular endothelial damage is a hallmark of the pathophysiologic responses.

PREDISPOSING FACTORS

A. Nulliparity.
B. Chronic hypertension.
C. Age extreme (<18 years and >35 years old).
D. Race (African American females are at higher risk).
E. Pregestational and gestational diabetes.
F. Renal disease.
G. Family history of preeclampsia in a sister or mother.
H. Previous pregnancy with PIH.
I. Multiple gestation.
J. Hydatidiform mole.
K. Obesity (BMI >30).
L. Autoimmune disease (systemic lupus erythematosus or antiphospholipid syndrome).
M. Hydrops fetalis.
N. Vascular disease.
O. IUGR, abruptio placenta, or fetal demise in a previous pregnancy.
P. Prolonged interpregnancy interval if previous pregnancy was normotensive.
Q. Short interpregnancy interval if previous pregnancy was preeclamptic.
R. New partner.

COMMON COMPLAINTS

A. Persistent and/or severe headache unrelieved by analgesics.
B. RUQ epigastric pain.
C. Severe heartburn unrelieved by antacids.
D. Nausea and vomiting.
E. Edema: peripheral and/or facial.
F. Visual disturbances (photophobia, scotomata, ametropia).

OTHER SIGNS AND SYMPTOMS

A. Hypertension (BP of 140 mmHg systolic or greater or 90 mmHg diastolic or greater that occurs after 20 weeks' gestation in a client without a history of hypertension).

First-trimester signs of PIH require ultrasonographic evaluation for the presence of a gestational trophoblastic disease (molar pregnancy) as well as the other differential diagnoses.

B. Proteinuria:
1. Random urine protein: creatinine ratio of greater than 0.3 mg protein/mg creatinine.
2. Timed excretion of greater than 0.3 g protein in a 24-hour urine collection specimen. The completeness of the 24-hour collection can be estimated from the creatinine excretion, which should be 15 to 20 mg/kg of lean body weight in women.
C. Brisk deep tendon reflexes (DTRs; hyperreflexia) or ankle clonus.

POTENTIAL COMPLICATIONS

A. Multiple organ involvement.
B. HELLP syndrome.
C. Eclampsia, which may lead to maternal and fetal demise.
D. Fetal complications: IUGR, oligohydramnios, abruptio placenta.

SUBJECTIVE DATA

A. Elicit information about headaches, their onset and duration, the progression of headache, and/or other symptoms.
B. What part of the head hurts? Differentiate headache from sinus headache. Note severity and any relief measure tried (acetaminophen, massage, sleep).
C. Is the headache "new"? Does the client have a history of migraines? Is this like a previous migraine?
D. What are other concurrent symptoms? Nausea, vomiting, RUQ pain, and visual changes?
E. Question the client about edema. If edema is present, has it significantly worsened over the past few days? Have they been able to wear rings up to this point? Have they had to wear different shoes due to pedal edema?
F. What are their usual weight and today's weight (on the same scales)? Have they gained more than 2 lbs in 1 week?
G. Ask specifics about RUQ pain, sometimes identified as "severe heartburn." Note the duration, severity, and relief measures tried. Have the client point to the area of discomfort (midsternum or under right breast).
H. Are there any visual disturbances, such as black dots they cannot see through?
I. Review other gastrointestinal symptoms such as diarrhea, abdominal pain, and gallbladder attack.
J. Review for signs of fever and thyroid storm.
K. Review the client's history for seizures.
L. Review for signs of pulmonary edema: dyspnea, chest pain, and/or decreased (≤93%) oxygen saturation by pulse oximetry.

PHYSICAL EXAMINATION

A. Check BP, pulse, respirations, oxygen saturation via pulse oximetry, weight, and fetal heart tones.
B. Inspect:
1. Check pedal, hand, and facial edema.
2. Check fundal height.
C. Palpate:
1. Palpate the abdomen, noting any hepatosplenomegaly and RUQ tenderness to the palpation. Assess fundal height. Note any fetal movement.
2. Palpate the lower extremities for pitting edema. Peripheral edema may be related to capillary leaking or "overfill" edema.
D. Percuss:
1. Gently check for liver enlargement or pain. Hepatic tenderness is caused by stretching of Glisson capsule from hepatic swelling or bleeding.
2. Perform neurologic exam for hyperreflexia: Check lower extremities for DTR and ankle clonus.
E. Auscultate:
1. Auscultate the heart and lungs.
2. Auscultate the fetal heart tone.

DIAGNOSTIC TESTS

A. Complete blood count (CBC) and platelets.
B. Liver profile (alanine aminotransferase [ALT] and aspartate aminotransferase [AST]).
C. Renal workup:
1. Uric acid and serum creatinine.
2. Urine protein:
a. ≥0.3 g protein in a 24-hour urine specimen.
b. Random urine protein: creatinine ratio ≥0.3 mg protein/mg creatinine.
c. Protein ≥1 + (30 mg/dL) on a paper test strip dipped into a fresh, clean voided midstream urine specimen (only if one of the previous quantitative methods is not available).

▶

3. Urine culture if proteinuria is present to rule out urinary tract infection (UTI).

D. Coagulation studies: prothrombin time (PT), partial thromboplastin time (PTT), and fibrinogen are indicated in clients with additional complications such as abruptio placenta, severe bleeding, severe liver dysfunction, or severe thrombocytopenia.

E. Ultrasonography to evaluate the amniotic fluid index and the estimated fetal weight, due to the increased risk for oligohydramnios and IUGR with preeclampsia.

DIFFERENTIAL DIAGNOSES

A. Preeclampsia.

B. Chronic hypertension.

PLAN

A. General interventions:

1. Any client with an elevated BP should be reassessed, using proper cuff size, after the client is allowed to relax for several minutes prior to repeat BP measurement.

2. BP is repeated after 5 minutes of rest, with the client sitting with feet on the ground and legs uncrossed or in a semireclining position with their back supported. Whether sitting or in semi-Fowler, the arm should be supported and at heart level. Measurement of BP in left lateral recumbency, on the left arm, does not differ substantially from BP that is recorded in the sitting position and may be used if a seated BP is not feasible.

3. If client's BP begins to rise above baseline values:

a. Advise the client to maintain a modified bedrest schedule (stop working).

b. Recommend frequent BP evaluation at home with documentation in a BP log.

c. See the client weekly to review their home BP log and further maternal and neonatal assessment; administer nonstress test (NST) and take biophysical profile (BPP) if appropriate.

4. If hypertension and proteinuria are present, refer the client to an obstetrician, the hospital's obstetrics triage unit, or a perinatologist for further assessment and management. They may need immediate admission to the hospital for inpatient management or delivery.

5. Clients with preeclampsia with severe features are usually delivered promptly to prevent maternal and fetal complications. Since the disease is progressive and there is no medical treatment, delivery is always in the best interest of the mother. However, preterm delivery is not always in the best interest of the fetus; therefore, a decision to delay delivery can be considered under certain circumstances. The rationale for delaying delivery in these pregnancies is to reduce perinatal morbidity and mortality by delivery of a more mature fetus and, to a lesser degree, to achieve a more favorable cervix for vaginal birth. The risk of prolonging pregnancy is worsening maternal endothelial dysfunction and continued poor perfusion of major maternal organs with the potential for severe end-organ damage to the brain, liver, kidneys, placenta/fetus, and hematologic and vascular systems.

6. If the client has a grand mal seizure, the primary consideration is to protect the client. Monitor seizure type and duration. Call for immediate transport to the hospital labor and delivery unit. Place the client in the lateral recumbent position following the seizure.

7. Immediately transfer the client to a hospital for eclampsia after stabilization.

B. Client teaching:

1. Educate the client and family about the concerns of high BP during pregnancy.

2. Discuss risks of having high BP during pregnancy for the mother and fetus.

C. Pharmaceutical therapy:

1. Diuretics are *not* prescribed during pregnancy for edema.

2. Angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), and direct renin inhibitors are *not* recommended during pregnancy due to their teratogenic effects on the fetus that may occur, such as renal dysgenesis and/or fetal death.

3. Aldomet (methyldopa), labetalol, and nifedipine are commonly used during pregnancy to control severe acute-onset hypertension, severe chronic hypertension, or superimposed hypertension during pregnancy. These antihypertensives are recommended as first-line therapy by the ACOG Task Force on Hypertension in Pregnancy when the systolic BP is greater than 160 mmHg or diastolic BP is greater than 105 mmHg. The ACOG Task Force also suggests avoiding antihypertensive therapy in pregnant females below this level if no evidence of end-organ damage. The goal of management is the maintenance of BP between 120/80 and 160/105 mmHg.

4. Daily low-dose aspirin is recommended as a preventive measure for females with known risk factors associated with preeclampsia.

5. Inpatient therapy is determined by a physician and may include the following:

a. Magnesium sulfate for seizure prophylaxis.

b. Hydralazine or labetalol, which are first-line antihypertensive agents if a client's systolic BP is greater than 160 mmHg and/or diastolic BP is greater than 105 mmHg.

c. Corticosteroids, which may be given to enhance fetal lung maturity prior to delivery in clients between 24 and 34 weeks' gestation.

d. In severe preeclampsia, therapy may include cervical ripening agents such as prostaglandins or misoprostol and/or oxytocin induction of labor.

e. Narcotics may be used for severe headaches.

f. Diazepam (Valium) and phenytoin (Dilantin) are not recommended for seizures in pregnancy. There is increased risk of recurrent seizures.

D. Dietary management:

1. Salt restriction does not stop swelling and high BP problems in pregnancy.

FOLLOW-UP

A. The only "cure" for preeclampsia is delivery. See the client in 1 week after delivery for BP assessment, or sooner if symptoms persist.

CONSULTATION/REFERRAL

A. If the client is diagnosed with preeclampsia, refer them to a physician for continued care and delivery.

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PRETERM LABOR

DEFINITION

A. Preterm labor (PTL) is defined as regular uterine contractions that produce documented cervical changes or spontaneous rupture of membranes after 20 weeks and prior to 36 6/7 weeks of gestation. Cervical changes may include dilation, effacement, or both.

INCIDENCE

A. PTL occurs in approximately 10% of the live births in the United States and precedes 50% of the preterm births (PTBs). It also accounts for more than 70% of neonatal mortality.

PATHOGENESIS

A. Infection, inflammation, decidual hemorrhage, and ischemia are common causes of PTL. Infection may originate from several sites, including the bladder, kidney(s), cervix, uterus, gastrointestinal tract, and upper respiratory tract. Ischemia may be caused by decreased oxygen delivery to the uterus due to maternal hypoxia, hypovolemia, or vena caval compression. Pathologic overdistension of the uterus in the presence of polyhydramnios or multiple gestation may cause PTL symptoms. Vaginal bleeding from a decidual hemorrhage (placental abruption) is associated with a high risk of PTL. The bleeding originates in damaged decidual blood vessels and presents clinically as vaginal bleeding or a retroplacental hematoma formation. However, in most cases, the cause is unknown.

B. Paternal risk factors: No paternal risk factors for the development of PTB in their partners have been identified. PTB risk is not increased by the father's history of preterm children with other partners or PTBs to members of the father's family.

PREVENTION

A. Progesterone supplementation decreases the risk of PTB by approximately 30% in females with a singleton pregnancy and a history of a spontaneous single preterm delivery.

B. Those with a singleton pregnancy and a history of spontaneous PTB should be offered referral to high risk obstetrics (OB) specialist. Treatment by the specialist may include 17-hydroxyprogesterone caproate to reduce the risk of recurrent spontaneous PTB.

C. In asymptomatic clients with a singleton gestation without a prior PTB with an incidentally identified very short cervix (<20 mm) before or at 24 weeks' gestation, refer to a high-risk OB specialist. Treatment by the specialist may include vaginal progesterone to reduce the risk of PTB.

PREDISPOSING FACTORS

A. Previous PTL or preterm delivery is the major risk factor for recurrence.

B. Preterm premature rupture of membranes (PPROM).

C. Uterine anomalies, surgery, and fibroids.

D. Multiple gestations.

E. History of second-trimester abortion(s).

F. Cervical insufficiency.

G. History of cervical surgery (cone biopsy or loop electrosurgical excision procedure [LEEP] for the treatment of cervical dysplasia).

H. Recurrent urinary tract and kidney infections, asymptomatic bacteriuria.

I. Polyhydramnios.

J. Fetal macrosomia.

K. Maternal age extremes (<18 or >40 years old).

L. Placenta previa.

M. Abruptio placentae.

N. Poor nutritional status and low prepregnancy weight.

O. Maternal dehydration.

P. Maternal race (occurs more frequently in African American population).

Q. Low socioeconomic status.

R. Inadequate prenatal care.

S. Anemia (hemoglobin <10 g/dL).

T. Substance use/abuse (smoking, drug, alcohol).

U. Vaginal infection.

V. Presence of fetal fibronectin, a protein produced by the trophoblast and other fetal tissues, has been noted in cervical-vaginal secretions between 24 and 34 weeks' gestation in a subgroup of individuals who are at increased risk for PTB.

W. Short cervical length.

X. Short interpregnancy interval.

Y. Abdominal surgery during pregnancy.

Z. Episodes of vaginal bleeding in more than one trimester.

AA. Maternal first-degree family history of PTB, especially if the client themselves were born preterm.

AB. Fetal anomaly or fetal growth restriction.

AC. Intrauterine fetal demise.

COMMON COMPLAINTS

A. Abdominal pain or menstrual-like cramping.

B. Low backache.

C. Increase or change in vaginal discharge, "gush" of fluid, loss of mucus plug, and bloody show or vaginal spotting.

D. Diarrhea.

E. "Something's not right."

OTHER SIGNS AND SYMPTOMS

A. Pelvic pressure (vaginal and/or rectal).

B. Contractions.

SUBJECTIVE DATA

A. Review the client's medical history and obstetric history, including risk factors for PTL.

B. Elicit information about the onset, frequency, duration, and course of abdominal pain or cramping; presence or absence of backache; the duration and frequency of these symptoms; and whether symptoms began subsequent to a certain event or activity. What, if anything, makes these symptoms better or worse?

C. Question the client about color, odor, consistency, and amount of vaginal discharge or bleeding. Is the discharge clear, pink, blood-tinged, or bright red vaginal bleeding? Was there a spot the size of a quarter or a half-dollar? Have ▶

they been wearing a perineal pad? How often do they have to change the pad? Is the pad soaked with blood when they change it?

D. For fetuses older than 18 weeks' gestation, question the client about frequency of fetal movements.

E. Question the client about urinary frequency, presence of urgency, or dysuria. Is the urine discolored, blood-tinged, or malodorous?

F. Question the client about recent sexual activity (e.g., has there been recent intercourse?).

G. If the client complains of diarrhea, ask if they have a fever and if anyone else in the family is ill.

PHYSICAL EXAMINATION

A. Check temperature, blood pressure (BP), pulse, respiratory rate, and fetal heart tones.

B. Inspect:

1. Note general appearance of discomfort.

C. Palpate:

1. Abdomen: Note presence, frequency, and intensity of uterine contractions; resting tone; and the presence of uterine tenderness or suprapubic pain.

2. Measure fundal height and gently assess fetal lie, position, and estimated fetal weight via Leopold maneuvers (typically reliable after 24 weeks' gestation). It may be difficult to palpate fetal parts in the presence of maternal obesity or polyhydramnios.

3. Back: Check for costovertebral angle (CVA) tenderness.

D. Auscultate:

1. Auscultate the heart and lungs (especially if the client is on tocolytics).

E. Pelvic exam:

1. Sterile speculum exam: Evaluate rupture of membranes and vaginal discharge or bleeding.

2. Bimanual exam: After rupture of membranes and placenta previa have been excluded by the client history, sterile speculum exam, physical exam, laboratory testing, and ultrasound (US) evaluation, as appropriate, perform a gentle bimanual examination. Note cervical dilation, effacement, station, cervical position, and softness of cervix.

3. Cervical exam during pregnancy: See Section II: Procedures, "Bimanual Examination: Cervical Evaluation During Pregnancy." **Do not perform a digital examination of the cervix if PROM is present without active labor or before a US has been performed to confirm placental location.**

DIAGNOSTIC TESTS

A. White blood cell (WBC), if indicated.

B. Urine dipstick for ketones, leukocyte, esterase, protein, and nitrite.

C. Evaluate vaginal discharge for pH with phenaphthazine (nitrazine) tape.

1. *Note*: False-positive and false-negative nitrazine test results occur in approximately 5% of cases.

2. False-negative test results can occur when leaking of amniotic fluid is intermittent or is diluted by other vaginal fluids. False-positive results can be caused by alkaline fluids in the vagina such as blood, seminal fluid, or soap.

D. Check ferning, if discharge is nitrazine-positive or PPROM is suspected. When viewed under a microscope, amniotic fluid produces a delicate ferning pattern in contrast to the thick, wide-branching pattern seen with cervical mucus.

1. *Note*: A false-positive fern test can be caused by well-estrogenized cervical mucus or a fingerprint on the microscope slide. A false-negative result can be caused by inadequate amniotic fluid collection on the vaginal swab or if the sample collected is heavily contaminated by other vaginal fluids or blood.

E. Wet prep, if indicated.

F. Cervical cultures for sexually transmitted infections (STIs; gonorrhea and chlamydia).

G. Culture for group B strep (if not performed in the previous 5 weeks).

H. Fetal fibronectin (fFN) prior to digital exam. Candidates for fFN testing must meet the following criteria:

1. Intact fetal membranes.

2. Cervical dilatation less than 3 cm.

3. Gestational age 24 0/7 weeks to 34 6/7 weeks.

4. No sexual intercourse in preceding 24 hours.

5. No gross vaginal bleeding.

a. A negative fFN indicates very low risk of preterm birth within the next 2 week. A positive fFN is less predictive and alerts the provider of an increased risk of preterm birth.

b. *Note*: False-positive results can occur due to ejaculate from coitus within the previous 24 hours, a grossly bloody specimen, or a digital cervical exam. A digital cervical exam can also produce a false-negative result. Administration of intravaginal substances, such as lubricants, medications, or douching, may also interfere with the assay.

I. Urine culture: Asymptomatic bacteriuria is associated with an increased risk of PTL.

J. Urine drug screen in clients with risk factors for substance abuse due to the link between cocaine use and placental abruption.

K. Ultrasonography for fetal biometry and dating, cervical length, amniotic fluid volume, biophysical profile (BPP), placental location, fetal presentation, ruling out fetal, placental, and maternal anatomic anomalies.

L. Electronic fetal monitoring (EFM) for contractions and fetal heart rate (FHR).

DIFFERENTIAL DIAGNOSES

A. PTL.

B. Preterm (or Braxton Hicks) contractions, with no cervical change.

C. Insufficient cervix.

D. PPROM.

E. Low back muscle strain.

F. Pyelonephritis or urinary tract infection (UTI).

G. Placenta previa.

H. Abruptio placentae.

I. Gastroenteritis.

J. Vaginal infection.

K. Maternal dehydration.

L. Ketoacidosis.

M. Round ligament pain.

PLAN

A. General interventions:

1. Regular uterine contractions with cervical dilation or effacement, with pressure on the lower uterine segment, strongly indicate PTL.

2. If the cervix is dilated more than 3 cm with contractions upon presentation, the client is probably having PTL. Consult with a physician for hospital admission and tocolytic candidacy. ▶

3. If the client is symptomatic with a positive fetal fibronectin test, consult with a physician for appropriate follow up.

4. Second trimester: If the client shows signs and symptoms of PTL, consider diagnosis of insufficient cervix. Refer the client to a physician for ultrasonography for cervical length and possible cerclage placement.

B. Outpatient management:

1. Outpatient bedrest is not beneficial. While bedrest improves uteroplacental blood flow and can lead to a slight increase in fetal birth weight, there is no evidence that it decreases the incidence of PTB. Additionally, prolonged immobility increases the risk of thrombolytic events, has clear negative psychosocial effects on clients and families, and leads to musculoskeletal deconditioning.

2. Limit physical activity: Clients at high risk for PTL should reduce their level of physical activity/exercise, particularly strength training, and occupational activities that require heavy lifting.

3. Prophylactic treatment against infection is not recommended. There is no clear evidence of benefit from prophylactic antibiotic therapy in those with PTL with intact membranes and no evidence of infection.

4. Sexual activity: Clients should consider avoiding sexual activity if they experience an increased frequency or intensity of contractions after sexual intercourse.

5. Administer corticosteroids to enhance fetal lung maturity if less than 34 weeks' gestation. Observe the client for contractions in the office.

6. Fetal fibronectin testing, where available, using American College of Obstetricians and Gynecologists (ACOG) guidelines.

C. Inpatient management:

1. Collaborate with physician.

2. Observation, possibly with intravenous (IV) hydration. Continuous fetal monitoring and uterine activity should be obtained with the external fetal monitor.

3. Cervical cerclage.

4. Antibiotic treatment against infection (group B *Streptococcus*) until urine and cervical culture results are available.

5. Parenteral tocolysis either to stop PTL or delay delivery long enough to allow transfer to a facility with the ability to care for preterm infants. According to numerous clinical studies, predelivery administration of magnesium sulfate that is given prior to 32 weeks' gestation offers fetal neuroprotection, which reduces the occurrence and severity of cerebral palsy and therefore may be the drug of choice for parenteral tocolysis in the hospital setting.

6. Administer corticosteroids to enhance fetal lung maturity if less than 34 weeks. Predelivery of betamethasone reduces the risk of neonatal death, respiratory distress syndrome, intraventricular hemorrhage, and necrotizing enterocolitis in premature neonates.

7. Transport the client to a perinatal center for neonatal care.

D. Client teaching: *See Client Teaching Guide for this chapter, "Preterm Labor."*

E. Pharmaceutical therapy:

1. Tocolytics short term may be utilized between 24 and 34 weeks' gestation. Maintenance therapy with tocolytics is generally ineffective, while short-term use is recommended mainly to allow for administration of antenatal steroids. Recently, the Food and Drug Administration (FDA) posted warnings cautioning against the use of maintenance oral terbutaline during pregnancy due to lack of efficacy and potential maternal cardiac risks and death; it should not be prescribed on an outpatient basis. Injectable terbutaline may be used on a short-term basis (48–72 hours) in a hospital setting. Calcium channel blockers and nonsteroidal anti-inflammatory drugs (NSAIDs) are also used in first-line treatment for up to 48 hours, allowing for antenatal steroids to be given and transfer to a tertiary care center.

2. Corticosteroids:

a. Steroids are given to enhance lung maturity in the fetus.

b. A single rescue course may be considered if the antecedent treatment was given more than 2 weeks prior and the client is judged to be likely to deliver within the next week.

FOLLOW-UP

A. Depending on the clinical scenario, the client may need to be seen weekly or biweekly. Fetal fibronectin test may be repeated every 2 weeks, but positive results alone should not be used to exclusively direct management. Repeat cultures as indicated, discontinuing antibiotics if cultures are negative. Encourage close phone contact with the client regarding questions or concerns.

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PYELONEPHRITIS IN PREGNANCY

DEFINITION

A. Pyelonephritis is an upper urinary tract infection (UTI) that occurs in one or both kidneys.

Pyelonephritis may evolve into acute respiratory distress syndrome (ARDS) in pregnancy.

INCIDENCE

A. The incidence of pyelonephritis in pregnancy is 0.5% to 2%. Due to the physiologic changes in pregnancy, UTIs are a common concern. Most cases develop as a consequence of undiagnosed or inadequately treated lower UTI. Approximately 75% to 80% of pyelonephritis cases occur on the right side, with a 10% to 15% incidence on the left side. A small percentage of cases are bilateral.

PATHOGENESIS

A. *Escherichia coli* is the main pathogen in pyelonephritis, although *Klebsiella pneumoniae* and *Proteus* species are also important causes of infection. Occasionally highly virulent gram-negative bacilli, such as *Pseudomonas, Enterobacter*, and *Serratia*, are responsible (more commonly noted in immunocompromised clients). Gram-positive group B strep (GBS) may also be responsible. Anaerobes also are unlikely pathogens in pyelonephritis except in cases of chronic obstruction or instrumentation.

PREDISPOSING FACTORS

A. Pregnancy: due to pregnancy-related anatomic changes in the urinary tract, such as dilated ureters caused by smooth muscle relaxation and pressure on the bladder from the enlarging uterus; immunosuppression of pregnancy may also contribute.
B. History of UTI, cystitis, and pyelonephritis.
C. Sickle cell disease.

COMMON COMPLAINTS

A. Fever.
B. Chills.
C. Flank pain or tenderness.
D. Urinary frequency or urgency.
E. Hematuria and dysuria.

OTHER SIGNS AND SYMPTOMS

A. Asymptomatic bacteriuria occurs in some pregnancies.
B. Abdominal pain and uterine contractions, risk of preterm labor (PTL) and birth.
C. Shortness of breath.

POTENTIAL COMPLICATIONS

A. Sepsis and septic shock.
B. ARDS: mortality rate 50% to 70%.
C. Pulmonary embolus, usually presents as sudden-onset costovertebral angle (CVA) tenderness.
D. Preterm birth.

SUBJECTIVE DATA

A. Elicit information on the onset, duration, and progression of symptoms.
B. Elicit problems with voiding. Ask the client about urinary frequency, urgency, and dysuria.
C. Ask the client if they have experienced preterm contractions.
D. Ask if the client is complaining of fever or chills.
E. Ask the client if their urine has a bad odor.
F. Ask the client if they have felt more tired than usual.
G. Ask the client if they have felt more nauseated than usual or if they have been vomiting.
H. Do they have a backache? Note location (unilateral or bilateral) and what, if anything, makes the backache better or worse.
I. Review the client's history for sickle cell disease, if appropriate; have they been tested?
J. Review prenatal history for recurrent UTIs, previous pyelonephritis, and any abnormalities of the genitourinary (GU) tract.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure (BP): fever more than 100.4°F, tachycardia, tachypnea, hypotension associated with sepsis, septic shock, and ARDS.
B. Inspect:
  1. Note general appearance for respiratory distress.
C. Palpate:
  1. Back: Check CVA tenderness (right CVA tenderness is more common in pregnancy).
  2. Abdomen:
    a. Palpate for uterine tenderness and contractions.
    b. Palpate for suprapubic tenderness.
D. Auscultate:
  1. Lungs and heart.
  2. Fetal heart rate (FHR).
E. Pelvic exam:
  1. Check for cervical dilation.

DIAGNOSTIC TESTS

A. Complete blood count (CBC) with differential or white blood cell (WBC): leukocytosis with left shift on differential seen.
B. Blood culture, if indicated.
C. Respiratory function:
  1. Arterial blood gases (ABGs), if indicated.
  2. Pulse oximetry, if indicated.
D. Renal function:
  1. Urinalysis:
    a. Check urinalysis for WBCs, red blood cells (RBCs), leukocyte esterase, and/or nitrites.
    b. Glucosuria may be normal in pregnancy due to decreased tubular capacity to reabsorb glucose. If it is consistently noted, further testing is needed.
    c. Proteinuria is *not* normal during pregnancy. All cases warrant further investigation.
  2. Urine culture and sensitivity: Bacterial count greater than 100,000 colonies/mL indicates a UTI and should be treated. The consensus is unclear whether GBS bacteriuria less than 100,000 colonies/mL should be treated prior to 35 weeks to prevent pyelonephritis, preterm delivery, and postpartum endometritis. Currently the American College of Obstetricians and Gynecologists (ACOG) recommends treating GBS bacteriuria greater than 100,000 colonies/mL even if the client is asymptomatic.
  3. Intravenous pyelogram (IVP), if indicated.
  4. Renal ultrasonography, if indicated.
  5. Blood urea nitrogen, creatinine.
  6. Electrolytes.

▶

DIFFERENTIAL DIAGNOSES

A. Pyelonephritis.
B. Cystitis.
C. Urethritis.
D. Urethral stricture.
E. Urolithiasis.
F. Genital infection.
G. Chorioamnionitis.
H. Septic abortion.
I. Postpartum endometritis.
J. Muscular strain.
K. Pulmonary embolus.
L. Severe upper respiratory tract infection.
M. Postprocedural dysuria or urinary frequency (e.g., following bladder catheterization or cystoscopy).
N. Chemical irritants.
O. Postpartum septic pelvic thrombophlebitis.
P. Renal calculi.

PLAN

A. General interventions:
  1. Rule out other sources of infection.
  2. Assess for PTL.
B. Client teaching:
  1. Educate client regarding signs/ symptoms to monitor for at home (urinary frequency, burning with urination, dysuria, fever, back pain, overall myalgia).
  2. Encourage client to call for appointment and/or seek medical care at the ED if symptoms occur.
  3. Educate client importance of early diagnosis to prevent pregnancy complications.
C. Dietary management:
  1. Advise the client to eat a regular diet as tolerated.
  2. Encourage client to drink 8 to 10 glasses of water a day.
  3. Warn the client to avoid beverages with caffeine. 100% cranberry juice and cranberry and blueberry capsules are good for urinary tract problems.
D. Pharmaceutical therapy: Most pregnant people with pyelonephritis will require inpatient intravenous treatment that can be followed with oral treatment. On occasion, some may be treated on a carefully monitored outpatient basis.
  1. Broad-spectrum antibiotic coverage until cultures and sensitivity results are back.
  2. Drug of choice for outpatient treatment:
    a. Nitrofurantoin (Macrobid) 100 mg orally Q12H for 7 days. Use with caution in those in the first trimester and after 36 weeks' gestation.
    b. Amoxicillin 500 mg orally Q12H for 10 days or 250 mg orally Q8H for 10 days.
    c. Augmentin 500 mg orally Q8H for 7 days or 875 mg orally Q12H for 7 days.
  3. If dysuria is present: phenazopyridine (Pyridium) 200 mg orally TID after meals for 3 days. Warn the client that phenazopyridine (Pyridium) turns urine orange.
  4. Alternative medications:
    a. Cephalexin (Keflex) 500 mg orally Q12H for 7 days.
  5. Inpatient treatment:
    a. Intravenous (IV) antibiotics such as cefazolin.
    b. Once afebrile and stable, client may be placed on oral antibiotics.

FOLLOW-UP

A. Once antibiotic therapy is initiated, most clients have a decrease in symptoms within 48 hours. By the end of 72 hours, almost 95% of clients are afebrile and asymptomatic. Stress to clients the importance of completing the course of antibiotics regardless of the absence of symptoms.
B. The most likely causes of treatment failure are a resistant microorganism or obstruction; common causes of obstruction in pregnancy are urolithiasis or compression of the ureter by the gravid uterus.
C. Repeat a urine culture at a 2-week follow-up visit.
D. Recurrence rates are very high. After the initial antibiotic therapy course is completed, start a prophylactic dose of an antibiotic that is to be taken daily until delivery, such as nitrofurantoin (Macrodantin) 50 to 100 mg PO at bedtime for recurrent infections.
E. Clients receiving prophylactic antibiotics should have their urine screened for bacteria at each subsequent office visit and be questioned about the recurrence of symptoms.
F. If no prophylactic treatment is undertaken, obtain a urine culture if symptoms recur or if urine dipstick is positive for leukocyte esterase or nitrites.

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VAGINAL BLEEDING: FIRST TRIMESTER

DEFINITION

A. Vaginal bleeding is common during the first trimester of pregnancy, occurring in approximately 25% of all pregnancies. It may range from spotting to massive hemorrhage and be intermittent or constant, with or without pain.

INCIDENCE

A. Vaginal bleeding is a common event in pregnancy. Early pregnancy loss, defined as an intrauterine pregnancy loss within the first 12 6/7 weeks of gestation, occurs in about 10% of pregnancies. Ectopic pregnancy is much less common, occurring in 2% of reported pregnancies. Pregnancies occurring after failure of tubal sterilization are at an increased risk of being ectopic. Rupture of an extrauterine pregnancy is life-threatening and must be excluded in every pregnant client with vaginal bleeding.

PATHOGENESIS

A. The pathogenesis varies according to cause. In most cases, it is caused by embryonic death, with resultant decrease in hormone levels and subsequent sloughing of the uterine decidua. Many of the embryonic deaths occur due to chromosomal abnormalities that are incompatible with life.
  1. Threatened abortion: vaginal bleeding with absent or minimal pain *and* a closed, long, thick cervix.
  2. Inevitable abortion: vaginal bleeding with pain and cervical dilation and/or effacement.
  3. Spontaneous abortion: nonviable products of conception expelled from the uterus spontaneously; this process may be complete or incomplete.
  4. Missed abortion: in utero death of the fetus before 20 weeks' gestation, with retention of the pregnancy. Vaginal bleeding may occur but the cervix usually remains closed. Ultrasound (US) reveals an intrauterine gestational sac with or without an embryonic/fetal pole but no cardiac activity.

5. Vanishing twin: a single pregnancy that results from very early loss of one embryo of a multiple gestation; may be associated with vaginal bleeding.

6. Vaginal bleeding may also be related to postcoital bleeding, cervical inflammation/infection or cervical, vaginal, or uterine pathology.

7. Ectopic pregnancy: fertilized ovum implanted outside of the uterus, most commonly in the fallopian tube.

PREDISPOSING FACTORS

A. Spontaneous abortion: In most cases, the cause is unknown:

1. Advanced maternal age (AMA; occurs more often in older females): A chromosomal abnormality in the embryo accounts for approximately 50% of cases in older females.

2. Abnormal uterine environment: uterine septum submucosal leiomyoma, intrauterine adhesions.

3. Systemic disease: diabetes, thyroid disease, thrombophilia.

4. Weight extremes (body mass index [BMI] <18.5 or >30).

5. Immunologic deficiencies.

6. Substance use, including caffeine, alcohol, cigarettes, and cocaine.

7. Exposure to teratogens and infections: food contaminated with *Listeria monocytogenes*, radiation.

8. Trauma.

9. Previous spontaneous abortion (AB): a history of two or more consecutive spontaneous abortions or a condition associated with spontaneous abortions such as maternal antiphospholipid syndrome or a uterine anomaly.

B. Ectopic pregnancy: In about half of the cases of ectopic pregnancy, cause is unknown.

1. Previous damage to the fallopian tube; frequently caused by pelvic inflammatory disease, tubal surgery for infertility, or bilateral tubal ligation.

2. Previous ectopic pregnancy.

3. Assisted reproductive technology pregnancy.

4. Infertility.

5. Smoking.

6. Endometriosis adhesions.

COMMON COMPLAINTS

A. Spontaneous abortion: Vaginal bleeding occurs that may or may not be associated with cramping or uterine contractions. When a pregnancy is more than 8 weeks' gestation, the presence of uterine bleeding, uterine contractions, and/or pain are indications of a threatened abortion.

B. Ectopic pregnancy: Vaginal bleeding and/or pelvic pain may occur soon after the first missed period; the client may be unaware of pregnancy. Sudden, acute, localized abdominal pain is associated with fallopian tube rupture. All vaginal bleeding in early pregnancy must be evaluated for ectopic pregnancy and considered ectopic until proven otherwise.

OTHER SIGNS AND SYMPTOMS

A. Threatened abortion: Slight bleeding may be present over several weeks; cramping; no passage of tissue; and positive pregnancy symptoms are present, including nausea, vomiting, fatigue, breast tenderness, and urinary frequency.

B. Inevitable abortion: Moderate to profuse vaginal bleeding occurs. Tissue may or may not be passed; uterine cramps or abdominal pain are present; and symptoms of pregnancy may be decreased or absent.

C. Incomplete abortion: Moderate to profuse vaginal bleeding, sometimes for several weeks, occurs; reports of passage of tissue; painful uterine cramping or "contractions"; and symptoms of pregnancy are often absent.

D. Complete abortion: Client experiences profuse bleeding, passage of tissue and large clots, abdominal cramping, or uterine contractions.

E. Ectopic pregnancy: Client experiences amenorrhea or irregular vaginal bleeding; abdominal pain is usually present, may be unilateral or generalized and may be associated with vertigo and syncope; shoulder pain, with irritation of phrenic nerve, may be present. Anxiety or palpitations are often noted.

SUBJECTIVE DATA

A. Elicit information about the onset, duration, amount of bleeding, and progression of symptoms.

B. When did it start? Is it continuous bleeding, "like a period," or is it spotting? How much bleeding has occurred? How many pads have been saturated? What is the size of the blood spots? Determine amount of bleeding: How much blood is on Peri-Pad? (a) scant amount: less than 1-in. diameter; (b) light amount: less than 4-in. diameter; (c) moderate amount: less than 6-in. diameter; (d) heavy amount: saturates the Peri-Pad within 1 hour. Have they passed any blood clots or tissue? Do they feel dizzy or light-headed?

C. Has the client had recent intercourse or pelvic exam?

D. What is the client's current method of birth control? Was the birth control method used consistently? Have they had a tubal ligation, or have they recently used an intrauterine contraceptive device (IUD)?

E. Ask the client the first day of their past menstrual period to date the pregnancy. Did they have a positive pregnancy test? If so, when? Have they had US to confirm pregnancy location and dating?

F. Does the client have a history of ectopic pregnancy, adnexal surgery, or pelvic inflammatory disease?

G. Question the client regarding the presence or absence of abdominal and/or back pain. If present, is it a continuous discomfort, or is it intermittent cramping? Was the onset sudden? How severe is the pain?

H. Is the client experiencing shoulder pain? This may be referred pain from phrenic nerve irritation due to intraperitoneal bleeding.

I. Does the client have a history of assisted reproductive techniques for conception with current pregnancy? This increases the risk for a heterotopic pregnancy, a rare complication of pregnancy in which both an intrauterine and an extrauterine pregnancy occur simultaneously. This condition is also known as a combined pregnancy, multiple-sited pregnancy, or coincident pregnancy.

PHYSICAL EXAMINATION

A. Check temperature (if indicated), pulse, respirations, and blood pressure (BP). Note postural hypotension and tachycardia. Hemodynamic instability may be noted in cases of profuse bleeding; assess vital signs and be alert for hypotension, tachycardia, tachypnea, and/or labored breathing.

B. Inspect:

1. Note general overall appearance of discomfort or pain and signs of shock before, during, and after examination.

2. Examine the Peri-Pad to determine amount and color of bleeding, if available.

C. Palpate:

1. Perform abdominal examination for rebound tenderness, masses, softness, tenderness, or abdominal wall distension. Sudden, acute, localized abdominal pain with ▶

signs of internal hemorrhage suggests rupture of the fallopian tube.

2. Palpate uterine size. Measure fundal height for consistency with pregnancy dates. If fundal height suggests pregnancy has advanced beyond first trimester, bleeding may be caused by abruption, placenta previa, or rupture of membranes with heavy bloody show.

3. Check iliopsoas and obturator muscle.

D. Auscultate:

1. Auscultate the heart, lungs, and bowel sounds to rule out other abdominal problems.

2. If the pregnancy is beyond 10 to 12 weeks' gestation, a handheld Doppler can be used to auscultate fetal heart tones.

E. Pelvic examination:

1. Place the client in a lithotomy position to examine external genitalia to access the volume and potential source of bleeding.

2. Perform sterile speculum examination: Assess color and amount of bleeding. Tissue and the products of conception may be noted at the cervical os or in the vaginal vault. The speculum exam may reveal a cause of bleeding unrelated to the pregnancy, such as a vaginal laceration, vaginal neoplasm, condyloma acuminata, vaginitis, cervical lesions (polyps, fibroids, ectropion), acute cervicitis, or a cervical neoplasm. Cervical dilation may be visualized.

3. Bimanual examination: cervical motion tenderness, adnexal, or abdominal tenderness. An adnexal mass and mild uterine enlargement are often present with ectopic pregnancy. A bulging cul-de-sac may represent a hemoperitoneum. Adnexal mass is present in 50% of ectopic pregnancies.

DIAGNOSTIC TESTS

A. Pregnancy test: quantitative serum beta human chorionic gonadotropin (HCG); serial tests at least 48 hours apart, making sure to perform test at same lab for accurate results. Of viable pregnancies, 99.9% display a rise in HCG greater than 35% over 48 hours. HCG levels that plateau or rise slowly (<35% in 48 hours) suggest an ectopic pregnancy or an abnormal intrauterine pregnancy.

B. Complete blood count (CBC) with differential and platelet.

C. Blood type, Rh, antibody screen, and cross-match if indicated.

D. Prothrombin time (PT) and partial thromboplastin time (PTT).

E. Doppler ultrasonography for fetal heart tones, for fetuses older than 11 weeks.

F. Ultrasonography: transvaginal and/or abdominal scan.

DIFFERENTIAL DIAGNOSES

A. First-trimester vaginal bleeding secondary to:

1. Threatened abortion.

2. Inevitable abortion.

3. Incomplete abortion.

4. Complete abortion.

5. Septic abortion.

6. Ectopic pregnancy: There is strong suspicion of ectopic pregnancy or fallopian tube rupture if symptoms present with history of fallopian tube damage (e.g., tubal surgery for infertility, previous ectopic pregnancy), pelvic infection, or IUD use.

7. Hydatidiform mole.

8. Anovulatory bleeding with an antecedent period of amenorrhea.

9. Benign or malignant genital tract lesion.

10. Menstrual bleeding.

11. Genital trauma.

12. Advanced pregnancy with placenta previa or abruptio placentae.

13. Salpingitis.

14. Appendicitis.

15. IUD-related symptoms.

16. Pelvic inflammatory disease.

17. Acute vaginitis or cervicitis.

18. Physiologic/implantation bleeding.

19. Postcoital bleeding.

PLAN

A. General interventions: Stabilize maternal condition and then determine the cause of bleeding.

1. Threatened abortion: expectant management. Bedrest is often prescribed, but multiple randomized trials have not found bedrest at home or in the hospital to be beneficial in preventing fetal loss. Symptoms either subside, leading to normal gestation, or worsen, leading to inevitable abortion. If bleeding persists without leading to spontaneous abortion, the client should be evaluated frequently, usually on a weekly basis, by means of ultrasonography to assess fetal viability. The client should be placed on pelvic rest, avoiding intercourse, and should not use tampons to absorb bleeding. Treatment with progestins for threatened abortion has not been proven to be effective in prevention of abortion.

2. Inevitable abortion: Care may include expectant management or medical management (Misoprostol), or a surgical evacuation can be undertaken.

3. Incomplete abortion: Medical management (Misoprostol) or a surgical evacuation is generally performed. Expectant management may be appropriate and should be done in collaboration with an obstetrical physician.

4. Complete abortion: If abortion is complete and the products of conception are delivered with complete membranes present and cessation of bleeding has occurred, no surgical intervention is indicated. In these cases, the tissue specimens must be carefully examined for completeness. Send all specimens to the laboratory for pathologic examination. If there is any question regarding complete passing of the placenta, do serial quantitative HCGs until back to nonpregnant levels.

5. Ectopic pregnancy: Refer the client to a physician for medical management or surgical intervention. The physician may perform culdocentesis to assess for hemoperitoneum. If the client is in shock, resuscitation with intravenous (IV) fluids should be started immediately by means of two large-bore angiocatheters. IV fluids such as lactated Ringer solution or normal saline should be infused at a rapid rate. The client is taken to the operating room, where the indicated procedure is one that controls hemorrhage in the shortest period of time. Salpingectomy and/or hysterectomy may be included.

B. Client teaching: *See Client Teaching Guide for this chapter, "Antepartum: First-Trimester Vaginal Bleeding."*

C. Pharmaceutical therapy:

1. Rho(D) immune globulin should be administered to any Rh-negative client.

2. Acetaminophen (Tylenol) or ibuprofen as needed for discomfort.

3. Ectopic pregnancy: In current practice, pharmacologic therapy is the preferred treatment for an ectopic pregnancy, and methotrexate (MTX) is the main agent used. MTX is a folic acid antagonist that has been used to inhibit the growth of trophoblastic cells. This chemotherapy is also the treatment of choice for ectopic pregnancy when surgery is contraindicated, or in the management of postoperative persistent trophoblast. Refer the client to a physician to evaluate them for MTX or operative intervention.

FOLLOW-UP

A. Threatened abortion: Follow the client weekly to assess for interval growth and presence of fetal cardiac motion. Instruct the client on Peri-Pad count. Repeat HCG every 2 to 4 days.

B. Spontaneous abortion: Repeat HCG observing for a decrease in the level. Once the uterine contents have been evacuated, follow up within 4 weeks unless the situation warrants an earlier follow-up visit. Contraception needs to be discussed with the client. Females who experience pregnancy loss may attempt conception as soon as they are medically recovered and psychologically ready. Studies do not report benefits for a delayed interval to conception.

C. Ectopic pregnancy: Once the ectopic pregnancy has been removed, the client should be seen in 2 to 6 weeks for a postoperative examination, unless the situation warrants an earlier follow-up visit. If MTX is used, do serial quantitative HCGs until they return to nonpregnant levels.

CONSULTATION/REFERRAL

A. Consult with a physician if the client has any frank bleeding, signs of fetal compromise, or maternal shock, or if the cause of bleeding cannot be determined.

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VAGINAL BLEEDING: SECOND AND THIRD TRIMESTERS

DEFINITION

A. Bright or dark red vaginal bleeding during the second or third trimester (>12 weeks' gestation) is less common. It may be painless, or it may be associated with uterine contractions or severe abdominal pain. Antepartum bleeding (uterine bleeding after 20 weeks' gestation that is unrelated to labor and delivery) occurs in 4% to 5% of pregnancies. Common causes of bleeding include:

1. Bloody show: This is associated with labor (by definition, labor occurs after 20 weeks' gestation) and vaginal discharge of mucus, which may be clear, pink, or slightly bloody.

2. Cervical insufficiency: The American College of Obstetricians and Gynecologists (ACOG) defines cervical insufficiency as "the inability of the uterine cervix to maintain a pregnancy in the second trimester in the absence of clinical contractions, labor or both."

3. Low-lying placenta: The edge of the placenta grows into the area of the lower uterine segment located less than 2 cm from the internal cervical os.

4. Placenta previa: Implantation of the blastocyst occurs in the lower uterine segment, followed by placental growth. Eventually, the placenta may partially or completely cover the cervix.

5. Abruptio placentae: This is partial or premature separation of a normally implanted placenta prior to delivery of the infant.

6. Uterine rupture: Complete uterine rupture extends through the entire uterine wall, and the uterine contents are extruded into the abdominal cavity. Incomplete rupture extends through the endometrium and myometrium, but the peritoneum remains intact. This occurs almost exclusively during labor and/or delivery.

7. Uterine dehiscence: This refers to separation of an old surgical scar.

8. Vasa previa: The fetal blood vessels are present in the membrane covering the internal cervical os. The membranous vessels may be associated with a velamentous umbilical cord or they may connect the lobes of a bilobed placenta or the placenta and a succenturiate lobe. Rupture of the vasa previa is an obstetric emergency and may lead to fetal death.

9. Bloody discharge is *not* normal prior to 37 weeks' gestation unless associated with recent sexual intercourse or pelvic exam. Light spotting or bleeding may be caused by recent sexual intercourse, preterm labor (PTL), rupture of membranes, or cervicitis. *Note:* The evaluation of vaginal bleeding prior to 20 weeks is similar to that in the first trimester.

INCIDENCE

A. Placenta previa: approximately 0.5% of pregnancies, but varies worldwide and is more common in parous females.

B. Abruptio placenta: approximately 0.4% to 1% of pregnancies.

C. Uterine rupture: approximately 0.08% to 0.22% of pregnancies. If uterus is unscarred, incidence is approximately 1:16,000 to 20,000 pregnancies. If uterus has a scar, the incidence varies depending on the type and location of the prior uterine incision.

PATHOGENESIS

A. Placenta previa: The pathogenesis of placenta previa is unknown. One hypothesis is that the presence of areas of suboptimal endometrium in the upper uterine cavity caused by previous surgery or pregnancies promotes implantation of trophoblast in or toward the lower uterine segment. Another hypothesis is that a particularly large placental surface area, as in multiple gestation or in response to reduced uteroplacental perfusion, increases the likelihood that the placenta will cover or encroach upon the cervical os.

B. Abruptio placenta: This is initiated by bleeding into the decidua basalis. The decidua then splits, and the placenta is sheared off, either partially or totally. Blood may move into and through the myometrium, leading to a board-like uterus.

C. Uterine rupture: This may occur from uterine injury, caused by previous surgery or trauma.

PREDISPOSING FACTORS

A. Placenta previa: Factors include late fertilization with delayed implantation, previous uterine scar, advanced maternal age (AMA), multiple gestation, large placenta, previous previa, and smoking.

B. Abruptio placenta: A prior placental abruption is the strongest risk factor for abruption. Hypertension (chronic, gestational, or preeclampsia), cocaine use, trauma, high parity, sudden decompression of overdistended uterus (e.g., when membranes rupture), smoking, chorioamnionitis, abdominal trauma, and external cephalic version are other factors.

C. Uterine rupture: Factors include multiparity, previous uterine incision, tetanic contractions, or prolonged labor, especially with excessive use of oxytocin.

COMMON COMPLAINTS

A. Placenta previa: Painless vaginal bleeding, usually in amounts of spotting to frank hemorrhage, is a common complaint. Bleeding occasionally is accompanied by cramping or uterine contractions. A "gush" of fluid associated with sudden onset of massive vaginal bleeding may be reported. Painless vaginal bleeding should be treated as a placenta previa until proven otherwise.

B. Abruptio placentae: Firm, tender uterus and hypertonic uterine contractions with high frequency and low amplitude are common. Typically presents with vaginal bleeding, uterine tenderness, and uterine contractions with or without nonreassuring fetal heart rate (FHR):

1. Marginal abruption: Vaginal bleeding may be absent or minimal and bright red; there may be some old, dark blood. Abdominal pain is usually mild.

Vaginal bleeding with abdominal pain should be treated as an abruption until proven otherwise.

2. Moderate abruption: Vaginal bleeding may be moderate or absent. Abdominal pain is usually significant and associated with contractions.

3. Severe abruption: Vaginal bleeding may be moderate, severe, or absent. Abdominal pain is severe. **The client may have a concealed placental abruption without vaginal bleeding.**

C. Uterine rupture: Vaginal bleeding is moderate, severe, or absent. The client may experience a sudden onset of extreme abdominal pain (commonly at the previous uterine scar site) or a cessation of uterine contractions. Recession of the fetal presenting part and maternal hypotension and tachycardia may occur.

D. Cervical insufficiency: A feeling of vaginal fullness or pressure; vaginal spotting or bleeding; an increased volume of watery, mucousy or brown vaginal discharge; and vague discomfort in lower abdomen or low back may be reported.

OTHER SIGNS AND SYMPTOMS

A. External fetal monitor (EFM) tracings: EFM may exhibit characteristics that are associated with anemia or hypoxemia, such as decreased or absent variability, bradycardia, tachycardia, recurrent late or prolonged decelerations, or a sinusoidal pattern.

B. Placenta previa: Uterine resting tone is usually relaxed. Fetal status at first exam is usually stable. Recurrence of bleeding is common. First bleeding episode in placenta previa is rarely significant. Second or third bleeding episode is often associated with significant vaginal bleeding.

C. Abruptio placentae: Rupture of membranes has bloody show with copious amounts of clear or greenish-brown fluid; the fluid is probably meconium-stained, which signifies fetal distress:

1. Marginal abruption: Uterine resting tone is usually relaxed. Fetal status on the fetal monitor at first exam is usually stable. Labor progresses rapidly with vaginal bleeding or large amounts of bloody show.

2. Moderate abruption: Uterine resting tone is hypertonic. At first exam, the fetus is usually alive. FHR may exhibit characteristics that are associated with hypoxemia or anemia such as decreased/absent variability, tachycardia, bradycardia, recurrent late or prolonged decelerations, or a sinusoidal pattern. Labor progresses rapidly with vaginal bleeding or large amounts of bloody show.

3. Severe abruption: Uterine resting tone is hypertonic or "board-like." At first exam, fetus may be dead. If fetus is alive, EFM is consistent with hypoxemia or anemia as listed above.

D. Uterine rupture: Uterine resting tone may be normal or hypertonic. At first exam, fetus may be dead or FHR pattern is consistent with hypoxemia or anemia as previously listed.

SUBJECTIVE DATA

A. Elicit information about onset, duration, and progression of vaginal bleeding. When did it start? Is it continuous bleeding, "like a period," or is it spotting?

B. Ask: How much bleeding has occurred? How many pads have been saturated? What is the size of the blood spots—the size of a quarter or a half dollar?

C. Elicit information regarding the presence or absence of abdominal pain. If present, review the onset, duration, and progression of pain. Is it a continuous discomfort or intermittent cramping? How severe is the pain? Did it have a sudden onset?

D. Is the client experiencing shoulder pain? This is likely to be referred pain from phrenic nerve irritation caused by intraperitoneal bleeding.

E. Elicit the first day of the client's past menstrual cycle, to date pregnancy. Have they had an ultrasound (US) to confirm dating and placental location?

F. Ask the client if they feel the baby move and if the movement has been normal this day, if more than 18 weeks' gestation.

G. Recent events such as vaginal exam, intercourse, trauma.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure (BP); peripheral oxygen saturation; urine output; and estimated blood loss. Continuous FHR monitoring is used for patterns suggestive of hypoxemia or anemia:

1. A pregnant individual does not demonstrate signs and symptoms of hypovolemic shock until they have lost 30% of their circulating volume. Tachypnea, tachycardia, hypotension, low oxygen saturation, and air hunger are signs of hypovolemia.

2. Prepare the client for emergency transport to a hospital even if they are hemodynamically stable.

B. Inspect:

▶

1. Inspect the client's general appearance related to discomfort and pain. Observe bleeding characteristics and pooling. Estimate blood loss.

C. Palpate:

1. Check for palpable fetal parts on abdominal wall; note fetal movement.

2. Palpate the uterus for relaxed or hypertonic uterus. Check for contractions. If present, note frequency, duration, and intensity and resting tone to palpation.

D. Auscultate:

1. Auscultate the abdomen; check fetal heart tones, or EFM, for baseline and periodic FHR patterns. Verify FHR versus maternal heart rate.

2. Auscultate the maternal heart and lungs.

E. Once placenta previa is ruled out, perform sterile speculum exam to look for the source of bleeding. **Do not perform vaginal bimanual exam until placenta previa is ruled out.**

DIAGNOSTIC TESTS

A. Complete blood count (CBC) and platelets.

B. Prothrombin time (PT), partial thromboplastin time (PTT), and fibrinogen (fibrinogen levels have the best correlation with severity of bleeding).

C. Blood type, Rh status, and type and cross-match if indicated.

D. Fetal cell stain, Kleihauer–Betke test, or a flow cytometry test on a specimen of vaginal blood. Fetal cell stain can determine the amount of fetal blood in the maternal circulation.

E. Determine if Rho(D) immune globulin (RhoGAM) is indicated.

F. Ultrasonography/biophysical profile (BPP).

G. Nonstress test (NST)/EFM.

DIFFERENTIAL DIAGNOSES

A. Placenta previa.

B. Abruptio placentae.

C. Uterine rupture or dehiscence.

D. Ruptured vasa previa.

E. Rupture of membranes.

F. Normal bloody show associated with labor.

G. Rectal hemorrhoidal bleeding.

H. Cervical insufficiency.

PLAN

A. General interventions:

1. Tocolysis may be considered if the client has no active hemorrhage and reassuring FHR pattern.

2. If significant vaginal bleeding is present, the primary goal is to maintain oxygen delivery to the mother and fetus while preparing them for transport. Interventions include lateral maternal positioning to avoid vena caval compression; administering supplemental oxygen; initiating large-bore intravenous line; delivery of fluid bolus of normal saline or lactated Ringer solution; keeping flow sheet of vital signs, assessments, actions, and responses; maintaining continuous recording of FHR and uterine activity on EFM; and providing emotional support and anticipatory guidance.

3. If vaginal bleeding is minimal and home management is being considered, discuss risks with the client and assess their ability to modify their activity to avoid moderate or strenuous exercise, heavy lifting (greater than 20 lbs), or standing for prolonged periods (more than 4 hours). Also assess client's access to telephone and transportation in case of major bleeding episode. Consider the distance from the client's home to the nearest hospital.

B. Client teaching: *See Client Teaching Guide for this chapter, "Vaginal Bleeding: Second and Third Trimesters."*

C. Pharmaceutical therapy:

1. Tocolytics: In clients with contractions, tocolytics may be used while administering a course of betamethasone if bleeding is diminishing or has ceased and delivery is not otherwise mandated by the maternal or fetal condition. Tocolysis may reduce or eliminate uterine contractions, which promote uterine separation and bleeding. Tocolytics should not be used in clients with active vaginal bleeding.

2. Antenatal corticosteroids may be given to enhance fetal lung maturity if preterm delivery is a possibility within the next week.

3. If the client is Rh-negative, give them Rho(D) immune globulin (RhoGAM) intramuscular by injection after the initial vaginal bleeding episode. Repeat administration is not necessary if bleeding occurs within 3 weeks of the previous dose. If repeated episodes of bleeding occur, the anti-D antibody can be checked: A low titer suggests that the anti-D level is sufficient to provide ongoing protection against alloimmunization.

4. A Cesarean delivery is always indicated with sonographic evidence of a placenta previa and a viable fetus.

FOLLOW-UP

A. Follow-up depends on client diagnosis and on whether client hospitalization is needed.

EMERGENT ISSUES/INSTRUCTIONS

A. Call 911/go to the nearest ED for frank, bright red bleeding.

CONSULTATION/REFERRAL

A. Consult a physician for all clients noted to have second- and third-trimester vaginal bleeding.

B. Refer to a physician if the client has any frank, bright red bleeding, signs of fetal compromise, or maternal shock, or if the cause of bleeding cannot be determined.

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POSTPARTUM BREAST ENGORGEMENT

DEFINITION

A. Breast engorgement is swollen, tender breasts caused by overfilling of milk, increased blood flow, and fluids in the breasts.

INCIDENCE

A. Breast engorgement may affect 40% of postpartum mothers.

PATHOGENESIS

A. *Primary engorgement* is the result of distension and stasis of the vascular and lymphatic circulations occurring 2 to 4 days following delivery. It is prompted by the decrease in progesterone levels after the placenta is delivered.

B. *Secondary engorgement* is caused by distension of the lobules and alveoli with milk as lactation is established. It may occur from excessive stimulation of milk production via pumping, taking medications to increase milk supply, or decreased milk extraction from not feeding the baby as often or baby not nursing well. Without stimulation by suckling and removal of milk, secretion of prolactin decreases and milk production decreases and finally ceases.

PREDISPOSING FACTORS

A. Engorgement often develops if early feedings are not frequent enough, suckling is inadequate, or breastfeeding is not conducted in a relaxed atmosphere.

COMMON COMPLAINTS

A. Swollen, tender breasts.
B. Discomfort when breastfeeding.
C. A low-grade fever lasting between 4 and 16 hours.

OTHER SIGNS AND SYMPTOMS

A. Cracked nipples.
B. Decreased milk production.

SUBJECTIVE DATA

A. Elicit onset, duration, and course of symptoms. Review the frequency of breastfeeding and/or use of breast pump. Is the client still breastfeeding, or has they stopped because of the discomfort?

B. Exclude other causes of fever, such as urinary tract infection (UTI) and wound infection.

C. Quantify pain symptoms and relief measures, including heat packs, ice packs, breast binder, and analgesics such as Tylenol.

PHYSICAL EXAMINATION

A. Check temperature, blood pressure (BP), and pulse.
B. Inspect:
  1. Examine the breasts for erythemic streaks on breasts or cracked nipples, previous surgeries, or bra compression.
  2. Check episiotomy or abdominal incision, if indicated.
  3. Observe infant feeding if possible looking at latch, watching for signs of swallowing.
C. Palpate:
  1. Examine the breasts for tenderness, hardness, warmth, and lumps.
  2. Palpate axilla for lymphadenopathy.
  3. Check the back for costovertebral angle (CVA) tenderness.

DIAGNOSTIC TESTS

A. Tests generally are not indicated for breast engorgement.
B. Urine culture or wound culture, if applicable.

DIFFERENTIAL DIAGNOSES

A. Breast engorgement.
B. Mastitis.

PLAN

A. General interventions:
  1. Encourage the client to take analgesics prior to breastfeeding and continue breastfeeding.
  2. Encourage ice packs for discomfort and frequent breastfeeding. There should be *no stimulation* to the breasts other than that provided by the baby when nursing, and the client should take analgesics for discomfort. Reassure them that engorgement is temporary and usually resolves within 24 to 48 hours.
B. Client teaching: *See Client Teaching Guide for this chapter, "Postpartum: Breast Engorgement and Sore Nipples."*
  1. Educate the client regarding milk production.
  2. Advise the client to breastfeed frequently to reduce chances of engorgement.
  3. Provide reassurance and support for the client to continue breastfeeding through this temporary period of discomfort. Engorgement may last 2 to 3 days before milk supply meets demand; continuation of breastfeeding will resolve discomfort and problem.
C. Pharmaceutical therapy:
  1. Acetaminophen (Tylenol) two tablets 500 to 1,000 mg every 4 to 6 hours, or ibuprofen 400 to 600 mg orally with food 30 to 45 minutes prior to breastfeeding and as needed.

FOLLOW-UP

A. Follow-up may not be required for engorgement.
B. Lactation consultation if indicated. Other treatments for engorgement such as cabbage leaves and herbal treatment are possible with a lactation specialist.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy loss: It is imperative to discuss breast care and engorgement prevention with females who have a second trimester termination of pregnancy, have a stillbirth, or experienced a neonatal loss.

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ENDOMETRITIS

DEFINITION

A. Endometritis is an infection of the endometrium (the interior lining of the uterus) that occurs postpartum. Endometritis is the most common cause of puerperal fever in obstetrics.

INCIDENCE

A. The incidence of endometritis has been noted to be as high as 27% after Cesarean section; the incidence is 1% to 3% after vaginal delivery.

PATHOGENESIS

A. During labor and delivery, indigenous cervicovaginal flora enter the uterine cavity. Onset is usually 3 to 5 days after delivery, unless it is caused by beta-hemolytic streptococcus, in which case the onset is earlier and more precipitous. Infection is usually polymicrobial in nature. Undiagnosed or ▶

unsuccessfully treated infection of the endomyometrium can progress to involve the entire uterus and may spread to accessory pelvic structures. Sources of bacteria may be any one or a combination of the following:

1. Indigenous vaginal bacteria, usually pathogenic only when tissue is damaged:
   a. Beta-hemolytic streptococcus.
   b. *Streptococcus viridans.*
   c. *Neisseria gonorrhoeae.*
   d. *Gardnerella.*
2. Contamination by normal bowel bacteria:
   a. *Clostridium perfringens.*
   b. *Escherichia coli.*
   c. *Proteus mirabilis.*
   d. *Aerobacter aerogenes.*
   e. *Enterococcus.*
   f. *Pseudomonas aeruginosa.*
   g. *Klebsiella pneumoniae.*
3. Contamination from environment; *Staphylococcus* is a common organism.

PREDISPOSING FACTORS

A. Operative delivery: Cesarean section is the major predisposing factor for pelvic infection. The most important determinant of infection for clients undergoing Cesarean delivery is the duration of labor.

B. Intrapartum: Prolonged rupture of membranes, numerous vaginal exams in labor, use of internal monitoring devices during labor, use of instruments in delivery, prolonged labor, and intrauterine manipulation such as internal rotation or manual removal of placenta can all lead to endometritis.

C. Postpartum: Retained placental fragments or membranes, improper perineal care, and host resistance also predispose a client to infection.

D. Anemia: This probably represents a marker of poor nutrition.

E. Obesity.

COMMON COMPLAINTS

A. "Feeling ill" with fever or chills.
B. Muscle aches.
C. Headache.
D. Uterine pain and tenderness.
E. Foul-smelling lochia.

OTHER SIGNS AND SYMPTOMS

A. Fever (100.4°F–104.0°F).
B. Subinvolution.
C. Uterus may be atonic.
D. Abnormalities of lochia:
   1. May be scant and odorless if anaerobic infection.
   2. May be moderately heavy, foul, bloody, or seropurulent if aerobic infection.
E. Tachycardia.

SUBJECTIVE DATA

A. Elicit onset, duration, and course of symptoms.
B. Review the color, odor, and amount of lochia.
C. Review the client's pain or discomfort and the relief measures used.
D. Review other body symptoms to rule out other infections such as urinary tract infection (UTI), breast engorgement, or mastitis.
E. Review labor and delivery events for complications (see section "Predisposing Factors").

PHYSICAL EXAMINATION

A. Check temperature, pulse, and blood pressure (BP): The client may be tachycardic with heart rate 100 to 140 beats per minute.
B. Inspect:
   1. Observe color, amount, and odor of lochia.
   2. Check abdominal incision, if applicable.
   3. Check the perineum for lacerations, breakdown of incision, redness, and drainage.
C. Palpate:
   1. Palpate the abdomen; check uterine tenderness.
   2. Palpate the back; check costovertebral angle (CVA) tenderness.
D. Auscultate:
   1. Auscultate the heart and lungs.
E. Pelvic exam:
   1. Speculum exam: Inspect the cervix for lacerations, drainage, or redness.
   2. Bimanual exam: Check for cervical motion tenderness; palpate adnexa for masses and tenderness; note "heat" of the pelvis.

DIAGNOSTIC TESTS

A. Complete blood count (CBC) with differential.
B. Blood and urine cultures.
C. Cervical cultures, to rule out a sexually transmitted infection (STI), if indicated.
D. Wet mount, to rule out bacterial vaginosis and candidal vaginitis, if indicated.

DIFFERENTIAL DIAGNOSES

A. Endometritis.
B. STIs, such as chlamydia, gonorrhea, or trichomoniasis.
C. Septic pelvic thrombophlebitis.
D. UTI/pyelonephritis.
E. Pneumonitis.
F. Extreme breast engorgement secondary to abrupt weaning causing maternal reabsorption of milk.
G. Wound infection.

PLAN

A. General interventions:
   1. Instruct on proper hygiene. Teach the client proper techniques to prevent infection (perineal area, incision site, breast).
   2. Acetaminophen (Tylenol) for fever as needed.
B. **Client teaching:**
   1. *See Client Teaching Guide for this chapter, "Endometritis."*
C. Pharmaceutical therapy:
   1. Endometritis is most often treated inpatient, but for mild cases of endometritis following a vaginal birth outpatient treatment may be appropriate. Several antibiotics are available for treatment of endometritis. Choice of antibiotics is affected by mode of delivery, severity of infection, and client allergies. Current evidence shows that intravenous clindamycin plus gentamicin is the most effective combination for treatment of endometritis.

FOLLOW-UP
A. Call or see the client in 24 to 48 hours to evaluate their status.

EMERGENT ISSUES/INSTRUCTIONS
A. Instruct the client to call immediately or go to the ED if symptoms do not resolve within 24 hours or if they worsen.

CONSULTATION/REFERRAL
A. Consult with a physician or nurse practitioner or midwife for treatment of endometritis.

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MASTITIS

DEFINITION
A. Mastitis is an infection of breast tissue with potential for abscess formation.

INCIDENCE
A. Mastitis has been estimated to occur in 2% to 10% of breastfeeding mothers. Less than 1% of these require hospitalization. Symptoms seldom appear before the end of the first week postpartum and are most often seen during the first 2 months postpartum.

PATHOGENESIS
A. During the period of lactation, the breast changes from an essentially nonfunctioning organ to a complex functioning organ of the body. The developing multiductal system becomes a rich environment for the growth of bacteria. The most common offending organism is *Staphylococcus aureus* (95%). The immediate source of the organisms that cause mastitis is almost always the nursing infant's nose and mouth.

PREDISPOSING FACTORS
A. Invasion of bacteria in the presence of breast injury, including:
- **1.** Bruising from rough manipulation (pumping) or failing to break the neonate's attachment to the areola and nipple before removing from breast.
- **2.** Prolonged breast engorgement.
- **3.** Milk stasis in a duct.
- **4.** Cracking or fissures of the nipple.
- **5.** Poor handwashing.

COMMON COMPLAINTS
A. Breast engorgement, usually bilateral.
B. Pain in the breast, usually unilateral.
C. Fever.
D. Red streak(s).
E. Flulike symptoms: body aches, headache, malaise, and chills.

OTHER SIGNS AND SYMPTOMS
A. Fever 100.0°F to 104.0°F, rapid rise.
B. Exquisitely tender breast tissue.
C. Hard mass in the breast.
D. Tachycardia and tachypnea.
E. Axillary lymphadenopathy.

SUBJECTIVE DATA
A. Elicit the onset, duration, and course of symptoms.
B. Note the frequency and length of time of the feeding or pumping.
C. Are there any red streaks on the breasts?
D. Are the nipples cracked and bleeding?
E. Quantify pain symptoms, relief measures tried, and results.
F. Review other symptoms to rule out other infections, such as wound infection, episiotomy breakdown, and urinary tract infection (UTI).

PHYSICAL EXAMINATION
A. Check temperature and blood pressure (BP), pulse, and respirations.
B. Physical exam should not be focused just on breast symptoms but should include a general ruling out of other potential problems, such as coexistent UTI or endometritis.
C. Inspect:
- **1.** Visually inspect breasts.
- **2.** Perform breast exam.
- **3.** Check episiotomy or abdominal incision to rule out infection.

D. Palpate:
- **1.** Perform breast exam. Observe breastfeeding for adequacy of latch, suck, swallow, jaw glide, and any clicking.
- **2.** Palpate lymph nodes of the neck and axilla.
- **3.** Palpate the abdomen.
- **4.** Check costovertebral angle (CVA) tenderness.

E. Auscultate:
- **1.** Auscultate the heart and lungs.

DIAGNOSTIC TESTS
A. Treatment usually initiated based on symptoms and exam. Early intervention may eliminate the need for antibiotics.
B. Complete blood count (CBC): leukocytosis in peripheral smear.
C. White blood cell (WBC), culture, and sensitivity of breast milk to identify bacteria for persistent signs of infection or if antibiotic treatment is unsuccessful.
D. Urine or wound cultures, if applicable.
E. Ultrasound considered if breast is not responding to treatment to evaluate for breast abscess.

DIFFERENTIAL DIAGNOSES
A. Mastitis: fever, chills, and malaise in conjunction with unilateral breast pain.
B. Breast engorgement: bilateral presentation of breast discomfort.
C. Breast abscess: discharge of purulent exudate from nipple, masses, or reddened areas that develop a bluish hue of the skin over the area of abscess.
D. Clogged milk duct.
E. Viral syndrome.
F. Inflammatory breast cancer.

PLAN

A. General interventions:

1. Encourage self-care and support. Advise the family to assist the client with self-care and infant care during this acute period. The client may feel extremely ill for the first 24 to 48 hours of therapy and may find it difficult to continue breastfeeding, as well as perform self-care and newborn care activities.

B. Client teaching: *See Client Teaching Guide for this chapter, "Mastitis."*

1. Advise the client to continue to breastfeed or pump to maintain milk supply.

2. Stress the importance of continuation of breastfeeding or pumping despite infection.

C. Dietary management:

1. There are no dietary restrictions.

2. Have the client increase fluid intake with increased temperature (at least 10–12 glasses a day).

3. Encourage them to eliminate caffeine, if possible, or use in moderation.

D. Pharmaceutical therapy:

1. Antibiotics:

a. Nonsevere with no methicillin-resistant *Staphylococcus aureus* (MRSA) risk factors: dicloxacillin 500 mg orally Q6H OR cephalexin 500 mg orally Q6H for 10 to 14 days to reduce relapse; however, 5 to 7 days can be used if rapid response occurs. If beta-lactam hypersensitive: clindamycin 300 mg orally Q8H.

b. Nonsevere with MRSA risk factors: clindamycin 300 mg orally Q8H. Trimethoprim-sulfamethoxazole (breastfeeding toxicity) one double-strength tablet orally Q12H.

c. Severe: vancomycin 15 to 20 mg/kg/dose every 8 to 12 hours, not to exceed 2 g per dose depending on breast milk and blood culture results.

2. Advise the client to complete the full course of antibiotics even if symptoms are improved sooner.

3. Candidal vaginitis may develop secondary to antibiotic therapy. The client should be aware of the signs, symptoms, and treatment plan if it should occur.

4. Acetaminophen (Tylenol) or ibuprofen for pain management.

5. The client may require pain medication if acetaminophen (Tylenol) or ibuprofen is not effective. Use acetaminophen with codeine phosphate (Tylenol No. 3) or other narcotic as needed for pain.

FOLLOW-UP

A. Evaluate the client in 48 hours if a breast abscess is suspected; assess need for surgical consultation.

CONSULTATION/REFERRAL

A. Consult a physician if a breast abscess is suspected, for persistent signs of infection, or if antibiotic treatment is unsuccessful. Treatment of a breast abscess may include surgical incision and drainage of the abscess.

B. Notify the pediatrician if mastitis is diagnosed.

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POSTPARTUM DEPRESSION

DEFINITION

A. Postpartum depression is a mood disorder characterized by unexplained tearfulness, sadness, irritability, and disturbances in appetite and sleep patterns, as well as an inability to care for self or baby, usually presenting within 2 weeks to 3 months postpartum.

INCIDENCE

A. Reported incidence in the United States is between 10% and 20%.

PATHOGENESIS

A. It is believed that postpartum depression may be related to psychological, physiologic, and cultural factors. The extreme hormonal changes that occur during the postpartum period may contribute. Postpartum thyroiditis is also a suspected factor. However, no confirmed biologic cause has been found. Some authorities have suggested that the mother's feeling of "loss of control" over their own life is the underlying precipitating factor.

PREDISPOSING FACTORS

A. The following may make a mother more likely to experience postpartum depression:

1. Preterm infant.

2. Multiple gestation.

3. Maternal and/or fetal complications in pregnancy.

4. History of postpartum depression or mental health conditions.

5. Social stressors: dissatisfaction in the marriage, financial difficulties, and lack of support in the home.

6. Age younger than 20 years.

7. Single parent.

8. Poor relationship with partner.

9. Evidence of significant emotional problems in the past.

10. Having experienced separation from one or both parents during childhood or adolescence.

11. Having received poor parental support and attention in childhood or having limited social support in adulthood.

12. Low self-esteem.

13. History of depression or anxiety.

COMMON COMPLAINTS

A. Insomnia.

B. Poor appetite.

C. Tearfulness.

D. Fatigue.

E. Anxiety.

F. Headaches.

G. Difficulty concentrating or confusion.

H. Feelings of excessive guilt or worthlessness.

I. Possible suicidal ideations.

OTHER SIGNS AND SYMPTOMS

A. Mood swings.

B. Despondency, social withdrawal, and feeling of inadequacy.
C. Guilt.
D. Impaired memory.
E. Ambivalence about motherhood and baby.
F. Inability to care for self and baby.
G. Poor grooming of self and/or baby.

SUBJECTIVE DATA

A. Elicit onset, duration, and course of symptoms.
B. Review the client's medical history for predisposing factors.
C. Question the client regarding their ability to care for the infant, themselves, and other family members at home.
D. Review the amount of support in the home. Is the client the primary caregiver? Are there any family members or friends who help in household management, sibling child care, and newborn care?
E. Does the client get out of bed and dress themselves daily?
F. Does the client have thoughts of harming the infant, themself, or others?
G. Review prenatal course and any complications. Review birth experience and evidence of birth trauma.

EXHIBIT 16.1 Postpartum Examination

Date: _____

History: Age: _____ G _____ PT _____ P _____ A _____ L _____

Labs: Blood type and Rh: _____ If negative, was RhoGAM workup done and RhoGAM given in the hospital, if needed? _____

Rubella: _____ If nonimmune, were they immunized before hospital discharge? _____

Discharge Hct: _____

Medical and antepartum complications: _____

Intrapartum course/complications: _____

Delivery: Date: _____ Type: _____ Sex: _____ Birth weight: _____

Apgars: 1 minute: _____5 minutes: _____ Neonatal: Gestational age: _____

Complications: _____

Feeding: Breast/bottle: _____ Current infant status: _____

Surgery: BTL: Yes/No _____

If yes, pathology report reviewed: Postpartum complications: _____

Current status: Medical problems: _____

Sexual intercourse since delivery/problems: _____

Psychosocial/postpartum depression: _____

Medications: _____

Physical exam: BP: _____ Pulse: _____ Resp: _____ Weight: _____

Breasts: _____

Pelvic: Episiotomy or laceration: _____

Abdomen: _____

Adnexa: _____

Uterus: _____

Vagina: _____

Cervix: _____

Pap smear: Date/results: _____

Plan: Current contraception and desired contraception: _____

Referrals: Labs: _____

New prescriptions: _____

Client Teaching Guides: _____

Pap smear follow-up: _____

Well woman exam with breast exam follow-up: _____

Other: _____

Signature: _____

A, abortion; BP, blood pressure; BTL, bilateral tubal ligation; G, gravida; Hct, hematocrit; L, living; P, preterm; P/T, para/term; RhoGAM, Rho(D) immune globulin.

EXHIBIT 16.2 Edinburgh Postnatal Depression Scale

Name: _____ Address: _____

_____ Your Date of Birth: _____

Baby's Date of Birth: _____ Phone: _____

As you are pregnant or have recently had a baby, we would like to know how you are feeling. Please check the answer that comes closest to how you have felt **IN THE PAST 7 DAYS**, not just how you feel today.

Here is an example.

I have felt happy:

☐ Yes, all the time	
☐ Yes, most of the time	This would mean: "I have felt happy most of the time during the past week.
☐ No, not very often	Please complete the other questions in the same way.
☐ No, not at all	

In the past 7 days:

1. I have been able to laugh and see the funny side of things.
- ☐ As much as I always could.
- ☐ Not quite so much now.
- ☐ Definitely not so much now.
- ☐ Not at all.

2. I have looked forward with enjoyment to things.
- ☐ As much as I ever did.
- ☐ Rather less than I used to.
- ☐ Definitely less than I used to.
- ☐ Hardly at all.

3. I have blamed myself unnecessarily when things went wrong.
- ☐ Yes, most of the time.
- ☐ Yes, some of the time.
- ☐ Not very often.
- ☐ No, never.

4. I have been anxious or worried for no good reason.
- ☐ No, not at all.
- ☐ Hardly ever.
- ☐ Yes, sometimes.
- ☐ Yes, very often.

5. I have felt scared or panicky for no very good reason.
- ☐ Yes, quite a lot.
- ☐ Yes, sometimes.
- ☐ No, not much.
- ☐ No, not at all.

6. Things have been getting on top of me.
- ☐ Yes, most of the time I haven't been able to cope at all.
- ☐ Yes, sometimes I haven't been coping as well as usual.
- ☐ No, most of the time I have coped quite well.
- ☐ No, I have been coping as well as ever.

7. I have been so unhappy that I have had difficulty sleeping.
- ☐ Yes, most of the time.
- ☐ Yes, sometimes.
- ☐ Not very often.
- ☐ No, not at all.

8. I have felt sad or miserable.
- ☐ Yes, most of the time.
- ☐ Yes, quite often.
- ☐ Not very often.
- ☐ No, not at all.

9. I have been so unhappy that I have been crying.
- ☐ Yes, most of the time.
- ☐ Yes, quite often.
- ☐ Only occasionally.
- ☐ No, never.

(*continued*)

EXHIBIT 16.2 Edinburgh Postnatal Depression Scale (*continued*)

10. The thought of harming myself has occurred to me.
- ☐ Yes, sometimes.
- ☐ Sometimes.
- ☐ Hardly ever.
- ☐ Never.

Administered/Reviewed by: _____ Date: _____

Instructions for using the Edinburgh Postnatal Depression Scale:
A. The mother is asked to check the response that comes closest to how they have been feeling in the previous 7 days.
B. All the items must be completed.
C. Care should be taken to avoid the possibility of the mother discussing their answers with others. (Answers come from the mother or pregnant client.)
D. The mother should complete the scale themselves, unless they have limited English or have difficulty with reading.

Scoring
QUESTIONS 1, 2, and 4 (without an*) are scored 0, 1, 2, or 3 with top box scored as 0 and the bottom box scored as 3.
QUESTIONS 3, 5–10 (marked with an*) are reverse-scored, with the top box scored as a 3 and the bottom box scored as 0.
Maximum score: 30 Possible depression: 10 or greater Always look at item 10 (suicidal thoughts)

EPDS, Edinburgh Postnatal Depression Scale.

Postpartum depression is the most common complication of childbearing. The 10-question EPDS is a valuable and efficient way of identifying clients at risk for "perinatal" depression. The EPDS is easy to administer and has proven to be an effective screening tool.

Mothers who score above 13 are likely to be suffering from a depressive illness of varying severity. The EPDS score should not override clinical judgment. A careful clinical assessment should be carried out to confirm the diagnosis. The scale indicates how the mother has felt ***during the previous week***. In doubtful cases it may be useful to repeat the tool after 2 weeks. The scale will not detect mothers with anxiety neuroses, phobias, or personality disorders.

Females with postpartum depression need not feel alone. They may find useful information on the websites of the Office on Women's Health (www.womenshealth.gov/mental-health/mental-health-conditions/postpartum-depression) and from groups such as Postpartum Support International (www.postpartum.net) and PostpartumDepression.org (www.postpartumdepression.org).

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PHYSICAL EXAMINATION

A. Check temperature (if indicated), pulse, respirations, and blood pressure.
B. Inspect:
1. Note general overall appearance, including dress, makeup, neatness of hair, tearfulness, and apathy.
2. Observe client's interaction with the baby, for example, talking to the baby and tone of voice, and eye contact.
C. Postpartum examination (Exhibit 16.1).

DIAGNOSTIC TESTS

A. The Edinburgh Postnatal Depression Scale (EPDS) should be completed at every postpartum visit (Exhibit 16.2). The EPDS is available for download at www.fresno.ucsf.edu/pediatrics/downloads/edinburghscale.pdf.
1. Scoring:
a. Add up the numbers associated with the answer that was underlined.
b. If the total is above 10, then possible depression exists; advise client to seek behavior health.
c. Always look at question 10 on suicidal thoughts; if the client answers anything other than "Never," advise behavior health appointment and assess if assistance is needed immediately.
B. A thyroid profile should be ordered.

DIFFERENTIAL DIAGNOSES

A. Postpartum depression.
B. Baby blues: tearfulness, insomnia, fatigue, headaches, poor appetite, and so forth; appearing between the birth and 14 postpartum days.
C. Postpartum psychosis: extreme emotional lability, agitation, delusions, hallucinations, and sleep disturbances.
D. Postpartum panic disorder: extreme anxiety, fear, tightness in the chest, and increased heart rate.
E. Postpartum obsessive-compulsive disorder: obsessive thoughts of harming the child, exaggerated fear of being left alone with the infant, anxiety, depression, and/or unnecessarily vigilant protectiveness of the infant.
F. Bipolar disorder.
G. Thyroid disorder.

PLAN

A. General interventions:
1. Assess all clients for mood disorders at all postpartum points of contact telephonic or in person.
2. Early assessment and treatment is very important. Symptoms that are not treated for several weeks may ▶

get progressively worse. Clients with severe depression, characterized by suicidal or homicidal ideation, aggressive behavior, delusions, hallucinations, catatonia, poor judgment, or grossly impaired functioning, are typically hospitalized.

3. Encourage involvement of the client's partner and immediate family members in the counseling sessions to assist them in learning ways to assist the client effectively.

4. Obtain serum thyroid levels.

5. Antidepressants may be indicated.

6. Psychotherapy or group care may be appropriate.

B. Client teaching:

1. Advise the client that they are not to blame for the condition, that it is not uncommon, and that successful treatment is likely.

2. Discuss participation in a support group, interpersonal psychotherapy, and cognitive behavioral therapy.

3. If antidepressants are prescribed, advise the client that the medication may take 4 to 6 weeks for peak effect. Review benefits/risks/side effects of the medication prescribed. The risk of suicide may increase after beginning antidepressants; therefore, a follow-up appointment in 1 to 2 weeks is recommended.

C. Pharmaceutical therapy:

1. Antidepressants may be ordered for those with moderate to severe symptoms of depression when physical and emotional functioning have been compromised. (Refer to the section "Depression" in Chapter 25, "Psychiatric Guidelines.")

2. Base selection of medication on whether or not the client is breastfeeding. Selective serotonin reuptake inhibitors (SSRIs) are considered first-line therapy during lactation. Sertraline (Zoloft) and paroxetine (Paxil) are generally first choice due to their low excretion in breast milk. Review risks and benefits of taking antidepressant during lactation, including the potential for relapse if not treated. Other medication treatment options during the postpartum period may include selective norepinephrine reuptake inhibitors (SNRIs), tricyclic antidepressants (TCAs), and norepinephrine-dopamine reuptake inhibitors (NDRIs). Brexanolone (Zulresso) has been approved and used for treatment of postpartum depression with good success but is extremely expensive.

FOLLOW-UP

A. If the client had risk factors for depression noted prior to delivery, a follow-up office visit 3 to 4 days after hospital discharge is suggested.

B. Frequent telephone contact, or several repeat visits, may be necessary during the course of the depression until the symptoms have improved.

C. The risk of suicide may increase after beginning antidepressants; therefore, a follow-up appointment in 1 to 2 weeks is recommended.

D. Assess the client for suicidal ideation and child neglect at every contact.

CONSULTATION/REFERRAL

A. Assess the need to refer the client to a psychiatrist, psychologist, or family counselor.

B. Refer to group therapy, interpersonal psychotherapy, and/or cognitive behavioral therapy.

C. Consult a psychiatrist about alternative treatments if no change in signs and symptoms is seen.

RESOURCES

Pacific Postpartum Support Society: www.postpartum.org.

Postpartum Support International: www.postpartum.net or 1-800-944-4773.

Postpartum Support Line: (604) 255-7999.

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SECONDARY POSTPARTUM HEMORRHAGE

DEFINITION

A. Secondary postpartum hemorrhage is defined as any significant blood loss after the first 24 hours of delivery and within 12 weeks of delivery. Postpartum hemorrhage has recently been redefined as a cumulative total of 1,000 mL or more.

INCIDENCE

Incidence of secondary postpartum hemorrhage is approximately 0.5% to 2% of females in developed countries. Peak incidence is one to two weeks postpartum.

PATHOGENESIS

A. Hemorrhage may result from retained placental fragments, subinvolution of the uterus, intrauterine infection, or inherited coagulation defects.

PREDISPOSING FACTORS

A. Abnormally adherent placenta.

B. Prolonged rupture of membranes leading to infection.

C. Overdistended uterus from multiple gestation, and polyhydramnios.

D. Hematoma.

E. Dehiscence of Cesarean scar.

F. History of postpartum hemorrhage.

COMMON COMPLAINTS

A. Heavy red bleeding or slow reddish-brown oozing.

B. Abdominal pain.

C. Loss of appetite.

D. Fatigue; cannot get enough rest and is unable to complete self-care and infant care activities.
E. Dizziness.
F. Weakness.

OTHER SIGNS AND SYMPTOMS

A. Lochia rubra is bright-red discharge immediately after delivery (1–3 days) and may contain a few small clots. A continuous trickle of bright-red blood suggests a laceration of the cervix or vagina. Saturation of one Peri-Pad in less than 15 minutes (two pads in 30 minutes, or rapid pooling of blood under the buttocks) is considered excessive bleeding and requires immediate attention.
B. Foul odor: Lochia should not be malodorous. Lochia usually has a "fleshy" odor.
C. Boggy uterus: Check the consistency of the uterus, whether it is firm or boggy. If atony is present, support the lower uterine segment, and massage the uterus or do bimanual compression.
D. Pelvic pain.
E. Tachycardia.
F. Hypotension.
G. Fever.

SUBJECTIVE DATA

A. Elicit onset, duration, and course of symptoms.
B. Elicit the amount and color of lochia, including size of blood clot(s).
C. Review symptoms of infection including fever and malodorous lochia.
D. Review labor and delivery events, including the date of delivery, mode of delivery, use of forceps or vacuum, weight of baby, manual removal of placenta, complications, and postpartum course.
E. Review pregnancy for predisposing factors such as multiple gestation and polyhydramnios (as previously noted).
F. For signs of infection ask about recent intercourse or use of tampon. Was an intrauterine device placed after delivery?

PHYSICAL EXAMINATION

A. Check temperature, blood pressure (BP), pulse, and respirations.
B. Inspect:
  1. Note color and amount of vaginal bleeding. Lochia may appear heavier when the client first stands up because the lochia pools in the vagina while they are recumbent. Once the pooled blood is discharged, lochial flow should return to normal.
  2. Inspect episiotomy or abdominal incision.
  3. Identify source of bleeding.
C. Palpate:
  1. Check for consistency of uterus, massaging uterus if boggy. Note any tenderness.
  2. Express clots, if applicable.
  3. By 2 weeks postpartum, the uterus should have involuted and once again be a "pelvic organ."
  4. Check abdominal tension.
D. Pelvic exam:
  1. Speculum exam: Assess cervical or vaginal lacerations.
  2. Bimanual exam: Rule out retroperitoneal hemorrhage.

DIAGNOSTIC TESTS

A. Complete blood count (CBC) with differential.
B. If bleeding is not under control, type and cross-match blood.
C. Coagulation test, if disseminated intravascular coagulation (DIC) is suspected. Prothrombin time, activated partial thromboplastin time, fibrinogen level.
D. Blood cultures to rule out infection.
E. Possible evaluation for a coagulation disorder such as von Willebrand disease.
F. Ultrasound.
G. Human chorionic gonadotropin (HCG).

DIFFERENTIAL DIAGNOSES

A. Late postpartum bleeding.
B. Normal postpartum bleeding.
C. Postpartum infection.

PLAN

A. General interventions:
  1. Perform uterine massage: Support the lower uterine segment during massage to prevent uterine prolapse.
  2. Give intravenous (IV) hydration for hypovolemic shock: hypotension, tachycardia, and faintness.
  3. Hospitalization is usually required for postpartum hemorrhage.
  4. Encourage breastfeeding (if applicable) to increase uterine contraction.
  5. Advise the client to rest and increase oral fluids.
B. Client teaching:
  1. Teach client how to perform fundal massage if experiencing increased bleeding.
C. Pharmaceutical therapy:
  1. Drug of choice: methylergonovine maleate (Methergine) 0.2 mg orally Q4H for six doses. **Do not give if the client is hypertensive.**
  2. For severe hemorrhage:
    a. Oxytocin (Pitocin) 10 to 20 units in 250 mL to 1,000 mL IV fluids.
    b. Methylergonovine maleate (Methergine) 0.2 mg by intramuscular injection, if the client has no history of hypertension. Advise the client to take full course of oral Methergine even if bleeding stops.
    c. **Hemabate: 250 mcg; may repeat every 15 to 90 minutes as needed.**
    d. Misoprostol (Cytotec, PGE1): 800 to 1,000 mcg, prolonged-release (PR).
    e. Continue bimanual compression and notify a physician.
  3. If infection is suspected or confirmed, antibiotics are prescribed.
D. Surgical procedures:
  1. Dilation and curettage (D&C) or suction curettage may be required if bleeding is not controlled.

FOLLOW-UP

A. Reevaluate the client 1 week from the date of discharge from the hospital.
B. Repeat hematocrit/CBC at postpartum visit.
C. The client may need iron replacement therapy, if not already prescribed. If stable at 1-week follow-up visit, have the client return in 4 to 6 weeks postpartum for routine postpartum exam.

CONSULTATION/REFERRAL

A. Immediately consult or refer the client to a physician for possible hospitalization for D&C and/or blood product replacement. ▶

B. Lactation services may be required. Postpartum hemorrhage can result in decreased milk supply.

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WOUND INFECTION

DEFINITION

A. Infection may occur at the site of the Cesarean section incision, an episiotomy, or a genital tract laceration. Most wound infections become clinically apparent after discharged from the hospital, approximately 5 to 6 days after delivery.

INCIDENCE

A. Clean wounds and grossly contaminated wounds have a risk of becoming infected postpartum. Rates of infection after Cesarean delivery range from 5 to 30 times more often than vaginal delivery. To reduce this risk, a single intravenous (IV) dose of a narrow-spectrum antibiotic (cefazolin, ampicillin) should be given within 60 minutes before making the skin incision to all females undergoing a Cesarean section. For females with a penicillin allergy, a dose of both clindamycin 900 mg and gentamycin 5 mg/kg IV can be used for prophylaxis.

PATHOGENESIS

A. A variety of organisms may be responsible. Examples include *Staphylococcus* or *Streptococcus* species and gram-negative organisms, gram-positive cocci and *Bacteroides, Clostridium* species, or *Escherichia coli*.

B. Early wound infections, occurring in the first 24 to 48 hours, are usually caused by group A or B beta-hemolytic *Streptococcus* and are characterized by high fever and cellulitis.

C. Delayed infections are more likely caused by *Staphylococcus epidermis* or *S. aureus, E. coli, Proteus mirabilis*, or cervicovaginal flora.

PREDISPOSING FACTORS

A. Obesity.
B. Anemia.
C. Malnutrition.
D. Smoking.
E. Diabetes.
F. Substance abuse.
G. Immunosuppression.
H. Poor hygiene.
I. Lower socioeconomic status.
J. Lack of preoperative prophylactic antibiotics.
K. Chorioamnionitis.
L. Blood transfusion.
M. Anticoagulation therapy.
N. Second-stage Cesarean section.
O. Subcutaneous hematoma.
P. Cardiovascular disease.
Q. Previous surgery.
R. Prior irradiation.

COMMON COMPLAINTS

A. Redness, heat, swelling, and tenderness at site.
B. Foul-smelling drainage.
C. Elevated temperature.

OTHER SIGNS AND SYMPTOMS

A. Fever and chills.
B. Edema.
C. Foul-smelling purulent discharge.
D. Leukocytosis.
E. Localized erythema, induration, and warmth at the incision site.
F. Pain at the incision site.
G. Wound separation may occur.

SUBJECTIVE DATA

A. Elicit onset, duration, and course of symptoms.

B. Review medical history (see section "Predisposing Factors"); antepartum history for complications such as diabetes; intrapartum complications for prolonged rupture of membranes, fever in labor, use of internal monitoring devices, length of labor, or frequent cervical examinations.

C. Question the client regarding hygiene at wound site since delivery, including the frequency of changing Peri-Pads, use of sitz baths, and showering.

D. Question the client regarding drainage from wound or episiotomy, noting color, amount, and odor.

E. Review signs and symptoms of breast engorgement and urinary tract infection (UTI).

F. Review vaginal delivery for third- and fourth-degree episiotomy or laceration. Although studies vary, third- and fourth-degree vaginal lacerations/episiotomies have approximately a 3% to 11% incidence, with an increased incidence of wound infection. In comparison, all types of perineal wounds have an infection rate of 0.1% to 5%.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure (BP).

B. Examination should not be limited to the incision site. A complete physical is needed to evaluate breasts, lungs, hematomas, and concurrent UTIs.

C. Inspect:
  1. Examine the incision site (episiotomy or abdomen) for drainage, redness or edema, and intactness.

D. Palpate:
  1. Perform breast examination only if indicated.
  2. Palpate suture line (episiotomy or abdomen). Probe incision with cotton-tipped swab to evaluate for hematoma, cellulitis, and/or pus.
  3. Palpate all abdominal quadrants.
  4. Palpate the vagina to rule out concealed hematoma.

E. Auscultate:
  1. Auscultate the heart and lungs.

F. Percuss:
  1. Percuss the back to assess costovertebral angle (CVA) tenderness.

DIAGNOSTIC TESTS

A. Complete blood count (CBC) with differential.
B. Blood culture (optional).
C. Culture of infected area.
D. Urinalysis, culture, and sensitivity, if indicated.

DIFFERENTIAL DIAGNOSES

A. Wound infection.

B. Impending dehiscence. If serosanguinous drainage is noted after the first 24 hours, dehiscence is possible.

C. Episiotomy/laceration breakdown.

PLAN

A. General interventions:

1. The wound may need to be opened and cleaned.

2. For an infection at a Cesarean section site, wound irrigation and dressing changes several times a day may be necessary.

3. Home health referral may be needed.

B. Client teaching: *See Client Teaching Guide for this chapter, "Wound Infection: Episiotomy and Cesarean Section."*

C. Dietary management:

1. No dietary restrictions are recommended; encourage the client to eat well-balanced meals. Increase protein in diet for wound healing.

2. Instruct the client to increase fluid intake; have them drink at least 10 to 12 glasses of liquid a day.

D. Pharmaceutical therapy:

1. Topical antiseptic and antimicrobial agents (povidone-iodine, sodium hypochlorite, hydrogen peroxide) should be avoided since they may be toxic to fibroblasts and impede wound healing.

2. Empiric treatment is started using broad-spectrum antibiotics with coverage of gram-positive cocci from the skin as well as the expected flora at the wound site:

a. Augmentin 875 mg BID for 7 to 10 days.

b. Clindamycin 450 mg Q6H for 7 to 10 days; safe for breastfeeding.

c. Cefoxitin 1 to 2 g by intramuscular injection or IV infusion every 6 to 8 hours; safe for breastfeeding.

3. Definitive antimicrobial treatment is guided by the clinical response of the client and the results of the Gram stain and wound culture and sensitivity, when available.

4. Acetaminophen (Tylenol) when required for elevated temperature.

FOLLOW-UP

A. Reevaluate the client in 48 hours to assess wound healing.

CONSULTATION/REFERRAL

A. Consult a physician for evaluation and possible surgical closure.

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POSTPARTUM EXAM

HISTORY

A. Chart review:

1. Antepartum course, including prenatal medical history and laboratory data: Pap smear, cervical cultures, maternal blood type and Rh, rubella and syphilis screen, and complete blood count (CBC).

2. Intrapartum course: length of labor, type of delivery, lacerations, and any maternal complications.

3. Neonatal course: gestational age, weight, length, cord gases, admission to normal or intensive care nursery, length of stay in the intensive care nursery, and any neonatal complications.

4. Immediate postpartum course: postpartum recovery, any postpartum complications, laboratory data, and length of hospital stay.

B. Interval history:

1. Number of weeks postpartum.

2. General maternal health and well-being, including diet or appetite, bowel and bladder function, level of activity, sleep patterns, and pain or discomfort.

3. Interval problems: calls to healthcare provider, visits to ED, fever, or illness.

4. Adjustment and role adaptation to the baby: motherhood, fatherhood, sibling rivalry, psychosocial assessment of depression, family support, housing, or financial issues. Sleep patterns should also be discussed.

5. Resumption of sexual activity: No required waiting period is recommended. Females should be encouraged to resume sexual activity when they are ready rather than a specific amount of days or weeks. Waiting for stitches to heal is recommended along with contraception use based on feeding practices to prevent unplanned pregnancies.

6. Family planning: previous method of contraception used, success of method, plans to resume contraception, and options for contraception.

7. Status of infant: breastfeeding or bottle feeding, consolability, sleep patterns, voiding, and stool patterns.

8. Establishment of healthcare follow-up: pediatrician appointment, immunizations; referral to Women, Infant, and Children (WIC), public health, and so forth, if applicable, or follow up with nurse practitioner or nurse–midwife.

C. Review of relevant systems:

1. Breasts: cracked or sore nipples, clogged ducts, engorgement, mastitis; breast care practiced.

2. Bladder function: stress incontinence, dysuria, urinary frequency, and flank pain.

3. Bowel function: constipation; discomfort, especially if the client has a history of third- or fourth-degree laceration; relief measures used and results.

4. Perineum: problems or discomfort at laceration/episiotomy site, problems with wound healing, and signs of infection.

5. Lochia: duration, type, odor, presence of clots; or resumption of menses: date, duration, and amount.

6. Abdomen: If Cesarean delivery, healing of wound, signs of infection; exercises initiated.

7. Legs: varicosities, heat, swelling, and calf tenderness.

PHYSICAL EXAMINATION

A. Blood pressure (BP), pulse, respirations, temperature (if indicated), weight.

B. Inspect:

1. Examine legs for varicosities and signs of thrombophlebitis.

2. Examine perineum, healing of episiotomy or lacerations, and abnormalities of Bartholin gland.

3. Speculum exam: Note lesions or lacerations of cervix and vagina, discharge, signs of infection; obtain Pap smear, if indicated.

C. Auscultate:
1. Auscultate the heart and lungs.
D. Palpate:
1. Palpate the abdomen for tenderness, masses, involution of uterus; examine Cesarean section and bilateral tubal ligation (if performed) incision for wound integrity and signs of infection.
2. Breast exam: Examine nipple integrity, masses, inflammation, and engorgement.
E. Bimanual exam: Check for abnormalities of cervix, uterus, adnexa; status of involution; presence of cystocele or rectocele; and vaginal muscle tone.
F. Rectovaginal exam: Check for integrity of episiotomy or laceration if indicated.

DIAGNOSTIC TESTS

A. Pap smear, if indicated.
B. CBC recommended if anemia or hemorrhage is documented or suspected.
C. 75-g 2-hour glucose tolerance test recommended if gestational diabetes is documented or suspected.
D. Other tests as indicated if there were maternal or fetal complications.

PLAN

A. General intervention:
1. This visit may be the last contact the client has with the healthcare delivery system for some time. The practitioner should evaluate any problems and provide appropriate consultations, referrals, interventions, counseling, and teaching:
B. Client teaching:
1. Explain the necessity of a yearly gynecologic exam.
2. Encourage regular aerobic, abdominal, and Kegel exercises.
3. Counsel the client on choice of contraception including failure rates with typical use:
a. Abstinence.
b. Tubal ligation/vasectomy.
c. Contraceptive implants.
d. Intrauterine devices.
e. Depo-Provera injection.
f. Oral contraceptives (OCs).
g. Barrier methods: condoms, cervical caps, diaphragm.
h. Spermicides.
i. Natural family planning, calendar method.
4. Explain the benefits of a healthy diet, especially if the client is breastfeeding.
5. Discuss breastfeeding, if applicable; answer any questions, and address concerns and review birth control options best during breastfeeding.
6. Instruct the client to communicate any breast changes to their provider and explain the importance of yearly provider-performed breast exams during their well-woman visit.
7. Explain the benefits of an interpregnancy interval of 1 to 2 years.

FOLLOW-UP

A. Administer rubella vaccination if the client has nonimmune status and administration of vaccine was missed in the hospital stay and they have not had unprotected intercourse since delivery.
B. If physical exam and laboratory and Pap tests are normal, the client does not require a physical for 1 year.
C. Establish a plan for the client to obtain Pap smear results (follow-up phone call or letter with results). See the Postpartum Examination sheet (Exhibit 16.1) to use for documentation in the client's chart.
D. Schedule primary care provider appointments to address any chronic health conditions.

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CLIENT TEACHING GUIDE

ANTEPARTUM: FIRST-TRIMESTER VAGINAL BLEEDING

PROBLEM Vaginal bleeding may occur in the first trimester of pregnancy. The amount of bleeding may range from spotting to a complete miscarriage.

CAUSE Bleeding may occur for a variety of reasons, including smoking, trauma, or abnormal fetus.

PREVENTION In most cases, the cause of vaginal bleeding may not be preventable. If bleeding is light, it may lessen or stop. You need to avoid sexual intercourse, tampons, and douches. If you smoke, it is highly recommended that you cut down or stop smoking altogether.

TREATMENT PLAN Treatment depends on the cause or suspected cause of your bleeding.

Activity:

A. Many people experience less bleeding and cramping while on limited activities. Unfortunately, activity restriction or bedrest do not prevent miscarriage.

B. Avoid sexual intercourse until at least 2 weeks after the bleeding has stopped, or until your provider tells you it is safe to resume.

C. Do not use tampons during this period of time. Use pads so that you can evaluate how much you are bleeding:

1. Scant amount: blood only on tissue when wiped or less than 1-in. stain on Peri-Pad.
2. Light amount: less than 4-in. stain on Peri-Pad.
3. Moderate amount: less than 6-in. stain on Peri-Pad.
4. Heavy amount: saturated Peri-Pad within 1 hour.

Diet: As tolerated. Eat a healthy diet with fresh vegetables, fruits, and bran cereal to avoid becoming constipated.

Medications: You may not be prescribed any medications.

You Need to Notify the Office If:

A. You develop a fever or chills with your bleeding.
B. You have a gush of blood from your vagina that is more than a period.
C. You pass blood clots or tissue from your vagina.
D. Your vaginal bleeding has a foul odor.
E. You experience abdominal pain or uterine cramping not relieved by taking acetaminophen.
F. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

ENDOMETRITIS

PROBLEM You have an infection of the inside of the uterus.

CAUSE One or more types of bacteria that invaded damaged tissue following your delivery could cause endometritis. The bacteria may be from the vagina, the bowel, or the environment.

PREVENTION

A. Use careful perineal care:
   1. Wipe from front to back after voiding.
   2. Remove Peri-Pad from front to back.
   3. Change Peri-Pad at least every 4 hours.
   4. Use a squeeze bottle filled with warm water to cleanse after each time you urinate or have a bowel movement.
   5. Use good handwashing technique after changing your pads and the baby's diaper.

TREATMENT PLAN

A. You will need to be treated with antibiotic therapy.
B. Take your temperature three times a day for the first 3 days on the antibiotics.
C. Take Tylenol or ibuprofen as needed for fever or discomfort.

Activity: It is very important for you to increase rest with an infection. Try to get a nap when the baby is sleeping. You may continue to breastfeed on some antibiotic therapy. If you do not breastfeed while you are feeling bad, pump your breast milk to keep up your milk supply but dispose of it.

Diet: Eat well-balanced meals. Drink at least 10 to 12 glasses of liquids a day.

Medications: Continue your prenatal vitamins. Take all of your antibiotics even if you start feeling better.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. Temperature that rises significantly or reaches 100.4°F.
B. Foul-smelling vaginal bleeding.
C. Increase in pain or tenderness.
D. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

GESTATIONAL DIABETES

PROBLEM Gestational diabetes develops only during pregnancy; it occurs due to the new hormones being produced. The hormonal influence makes you "insulin-resistant," meaning you still produce insulin but the hormones prevent it from working effectively.

Your blood sugar needs to be controlled so that the amount of sugar going to your baby is controlled, too. High blood sugar may cause a big baby at delivery, increases your risk of a Cesarean birth, causes the baby to have low blood sugar after delivery, increases jaundice, and causes other issues for the baby, including lung problems. It can also cause your baby to be overweight in childhood and increases the risk they will develop diabetes.

CAUSE You are producing new hormones that cause insulin resistance. The likelihood of having gestational diabetes increases with other factors, such as the mother's age, and it is more common in certain groups, such as Latin Americans, Native Americans, and African Americans.

PREVENTION Good control of your diet, exercise, and the possible use of medication and/or possibly insulin will help you to control your blood sugar during your pregnancy.

TREATMENT PLAN

A. You are asked to keep a record of your blood glucose values:
  1. You will be shown how to test your blood.
  2. You need to test your blood multiple times a day: first thing in the morning (fasting), after breakfast, after lunch, after dinner, and at bedtime.
  3. You will be given specific instructions before or after meals.
  4. Bring in or upload in your patient portal your blood sugar values every week. Your insulin may be changed weekly.
  5. The goal of your fasting blood sugar before breakfast is 60 to 95 mg/dL.
  6. Your blood sugar goal for 2 hours after meals is less than 120 mg/dL.

B. You may need to test your urine for ketones:
  1. You will be shown how to test your urine.
  2. You need to test for ketones if you are unable to eat or if you have diarrhea.
  3. You need to test for ketones if you feel like you have a urinary tract infection, sinus infection, or other kind of infection.
  4. You need to test for ketones if your blood sugar is higher than 150 mg/dL.
  5. You must follow the diet given to you by your dietitian. If you have questions or do not understand what you should be eating, contact your dietitian.

Activity: Exercise lowers blood sugar. Exercise is needed to control gestational diabetes. You need to walk at least 20 to 30 minutes a day. Try your local mall for a climate-controlled place to walk. Your heart rate should not get above 140 beats per minute.

Diet: You are placed on a _____ calorie diet. The amount of calories needs to be spread out over three meals and three snacks:

A. Breakfast, midmorning snack, lunch, midafternoon snack, dinner, and a snack at bedtime.
B. The time you eat is as important as what you eat. Try to keep on a regular schedule.

Medications: Depending on your blood sugar, you may require medication to control it. You will be instructed on how to take the medicine. If you are started on insulin, you will require extra testing for the rest of your pregnancy.

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CLIENT TEACHING GUIDE

INSULIN THERAPY DURING PREGNANCY

You Have Been Prescribed Insulin Therapy:

A. Your insulin needs may change weekly due to the change in your hormones (you become more insulin-resistant as your pregnancy progresses).

B. Insulin therapy is safe for your baby. Insulin does not cross the placenta like sugar does.

C. The insulin lowers your blood sugar and therefore controls the amount of sugar that goes to your baby.

D. You may have been prescribed Humulin insulin, which works very much like your own body's insulin.

E. Some of the insulin therapies have a mix of short-term regular (clear) insulin with intermediate-acting (cloudy) insulin:

1. You will be instructed on how to mix and give yourself your insulin.

2. The first key to insulin therapy is to be able to recognize signs of too much and too little insulin. (Table 1).

TABLE 1 SIGNS OF HIGH AND LOW BLOOD SUGAR

Blood Sugar	What to Watch for	What to Do	Causes
HYPOGLYCEMIA Low blood sugar	Excessive sweating Feeling faint Feeling shaky Headache Impaired vision Hunger Irritable feelings Personality change Trouble awakening	**Call the provider immediately if your blood sugar is below:** _____. Take glucose tablets or eat. Do not take your insulin. Do not try to force any food or liquids by mouth if the client is not conscious.	**Too much insulin** Not eating on time or enough food Unusual amounts of exercise
HYPERGLYCEMIA High blood sugar	Increased thirst Need to urinate more often Large amounts of sugar in your blood or urine Ketones in your urine Weakness and generalized aches Heavy, labored breathing with a fruity breath Nausea and vomiting	Test your blood sugar. **Call your provider immediately if your blood sugar is** _____. Test your urine for ketones. Drink extra water if able to swallow.	**Too little insulin** Eating more foods and foods not on your diet Infections and fever Stress

3. The second key is to let people know you are on insulin. You need a Medical Alert bracelet or necklace as well as information to put in your car and purse/billfold.

4. The third key is to have your baby and yourself evaluated more often when on insulin therapy:

a. You need to be seen more frequently as recommended by your healthcare provider.

b. You will have extra testing to make sure the baby is doing well and to make sure you are doing well, too.

F. You need to check your blood sugars multiple times a day. Your blood sugar targets are the following:

1. Fasting_____

2. After breakfast_____

3. After lunch_____

4. After dinner_____

5. Before going to bed _____

G. You will be instructed to check your urine for ketones when you are sick or if you have high blood sugar.

Activity: It is important to continue to exercise.

Diet: Eat a good, healthy diet. You will be instructed on how many calories to eat. Eat six smaller meals a day; with insulin, it is important to eat snacks. It is also important to try to eat at the same times each day.

(*continued*)

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CLIENT TEACHING GUIDE

INSULIN THERAPY DURING PREGNANCY (*CONTINUED*)

You Need to Notify the Office If:

A. You have moderate ketones in your urine.
B. You are unable to eat or you have loose diarrhea stool.
C. You have insulin reactions (blood sugar is below 50 mg/dL or you feel the symptoms of low blood sugar).
D. You have blood sugars higher than 175 mg/dL for two readings.
E. You have any signs of infection.
F. You have a decrease in fetal movement or do not feel your baby moving.
G. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

IRON-DEFICIENCY ANEMIA (PREGNANCY)

PROBLEM You have a "low blood count" called iron-deficiency anemia. Iron is needed for red blood formation.

CAUSE This is caused by a deficiency of iron in your diet, and it is very common. Expanded blood volume of pregnancy leads to anemia.

PREVENTION Anemia may be prevented by increasing iron in your diet and by taking extra iron tablets.

TREATMENT PLAN

A. You need to increase the iron-rich food in your diet.
B. You will be prescribed an iron supplement.
C. Antacids for indigestion and dairy products interfere with iron absorption. Do not take your iron supplements with milk or just before or after an antacid.
D. If you are pregnant, you may be eligible for the WIC program (Women, Infants, and Children), which provides supplemental foods for pregnant females and young children. Ask your healthcare provider for information about its availability in your community.

Activity: You may feel more tired than usual because of anemia. You may need to rest more than usual; however, try to continue your current exercise routine as tolerated. Alternative exercise includes walking 20 minutes a day or swimming.

Diet: You need to increase the amount of iron in your diet. Generally, the redder the meat and the greener the vegetable, the richer it is as a source of iron. You also need to make sure you have adequate intake of vitamin C (this helps increase the absorption of iron into your body). Vitamin C is found in fresh, dark-green vegetables and citrus fruits. Drink 8 to 10 glasses of liquids every day.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

Special Instructions About Iron Supplements:
A. Take the iron medication as prescribed; higher doses are not better. You may need to be taking it for a longer time. High doses of iron can be toxic to children and adults.
B. Your body only absorbs a small portion of the iron pills you take.
C. You may notice green or black bowel movements. This is normal.
D. It is best to take the iron pills on an empty stomach.
E. Try taking your iron pill with a glass of orange juice. The vitamin C in the juice helps the iron be absorbed better.
F. You may have nausea or vomiting when you take iron pills, especially during early pregnancy. If this happens, try taking the pill with food. It is better to take your iron with food than to skip your pill altogether.
G. If you are not able to tolerate the iron in the morning, try taking it in the middle of the afternoon or at bedtime.
H. You may become constipated while taking iron pills. Increase your intake of fruits, vegetables, dietary fiber, and water to avoid constipation.

You Need to Notify the Office If You:
A. Have nausea and vomiting while taking the iron supplement even after following the special instructions.
B. Become extremely constipated even after increasing the fiber and liquids in your diet.
C. Are unable to take the supplement at all.
D. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

MASTITIS

PROBLEM You have an infection in your breast tissue, not your breast milk.

CAUSE The most common organism causing mastitis is *Staphylococcus aureus*. The immediate source of the organism is almost always the nursing infant's nose and mouth. Mastitis often develops in the presence of breast injury, such as cracked nipples.

PREVENTION

A. Prevent injury to the breast:
  1. Avoid overdistension of the breasts; feed infant or use the breast pump frequently (every 2–4 hours).
  2. Avoid clogged milk ducts by applying moist heat to the breasts and massage.
  3. Avoid rough manipulation of the breast; pump carefully.
  4. Avoid cracking of nipples by proper positioning of the infant's mouth on the nipple during feeding. The baby's mouth should cover the entire areola (dark brown part of the nipple area).
  5. Read *Client Teaching Guide "Postpartum: Breast Engorgement and Sore Nipples."*

B. Personal hygiene measures:
  1. Avoid soap on the nipples; cleanse nipples with warm water only and air-dry when possible versus rubbing dry.
  2. Avoid decrusting the nipple of dried colostrum or milk.
  3. After a feeding, squeeze out a few drops of colostrum/milk and massage that into the nipple and areola. You can also use purified lanolin cream after each breastfeeding for sore, cracked nipples. (If lanolin is purified, there is no need to remove it prior to the next feeding.)
  4. Use good handwashing techniques before handling the breast and before and after breastfeeding.

TREATMENT PLAN

A. Complete course of antibiotics. Be aware that antibiotics may cause a yeast infection.

B. Continue breastfeeding even on the antibiotics. It is not uncommon for your baby to develop "thrush" (looks like white patches on your baby's mouth and tongue). You may also be prescribed Nystatin cream to apply to your breasts to help prevent thrush.

C. Apply warm soaks to your breast (*see Client Teaching Guide "Postpartum: Breast Engorgement and Sore Nipples"*). Breast massage may be needed, too.

D. Use Tylenol or ibuprofen for pain (*see Client Teaching Guide "Postpartum: Breast Engorgement and Sore Nipples"*).

Activity: Increased rest is recommended. Try to lie down for a nap when the baby goes to sleep.

Diet: There are no dietary restrictions; continue your regular diet and avoid gas-producing foods. Increase fluid intake to 10 to 12 glasses of water a day. Use caffeine in moderation (eliminate if possible).

Medications: Continue your prenatal vitamins while breastfeeding.

You Have Been Prescribed: _____

You Need to Take: _____

Take all of your antibiotics even if you feel better.

You Have Been Prescribed: Miconazole OR clotrimazole OR Mycolog cream for your breasts and nipples.

You Need to Apply It:
A. After each feeding, apply the cream to each nipple and areola of your breast.
B. Before feeding, wipe off the excess cream with a warm washcloth (do not use soap to avoid drying and cracking).

You Have Been Prescribed _____ for a yeast infection.

You Need to Use It: _____

You Need to Notify the Office If:
A. You have a temperature that does not decrease within 2 days and resolve within 4 days of taking the antibiotics.
B. You have pain that is not controlled with acetaminophen or ibuprofen.
C. Your baby develops thrush. Notify your baby's healthcare provider for medication.
D. Other: _____.

Phone: _____

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CLIENT TEACHING GUIDE

POSTPARTUM: BREAST ENGORGEMENT AND SORE NIPPLES

PROBLEM Engorgement causes swollen, tender breasts, which may have palpable nodular areas.

CAUSE Engorgement is caused by inadequate emptying of the breast. Engorgement may develop due to inadequate suckling by your baby.

PREVENTION

A. At first, nurse your baby every 2 hours.

B. Make sure your baby latches on to as much as possible of the areola (darkened area around the nipple). The baby suckling on the tip of the nipple does not provide the stimulation necessary to let down the milk and can make your nipples sore and cracked.

C. Always break the neonate's attachment of the areola and nipple before removing them from the breast. To break the neonate's attachment, place your finger in between the neonate's mouth and breast to break the suction. If your baby is not well-attached to the breast, detach the baby and make sure they open the mouth wide to accommodate most of the areola.

D. Wear a supporting nursing bra (avoid underwire bras, as they can exert pressure on certain areas of the breast and cause milk stasis, which is a good medium for bacterial growth and infection); make sure that your bra does not squeeze your breasts too tightly.

E. Making sure that the baby is properly attached to the nipple helps to avoid cracking of nipples that can predispose you to an infection of the breast called mastitis.

F. After the baby feeds, express some milk and apply it to the nipple and areola.

G. Purified lanolin can also be very helpful for sore nipples and can prevent further cracking and infection. Apply routinely after each breastfeeding session for the first several days of nursing and longer if tender or cracked nipples occur. If the lanolin is purified, there is no need to wash it off prior to feedings.

TREATMENT PLAN

A. Engorgement:

1. Treatment of engorgement includes the application of heat, breast massage, and expression of milk for comfort only.
2. A warm moist washcloth or a warm shower before massaging the breast decreases discomfort.
3. Massage breast by making several gentle but firm stroking movements with the fingertips along the swollen ducts, moving toward the nipple. This should be done around the entire breast.
4. After massaging, milk should be expressed or pumped until the breast softens enough for the baby to latch well. The baby should then be allowed to nurse from both breasts.
5. The best strategy for engorgement is frequent breastfeeding (at least every 1/2 to 2 hours until engorgement resolves).

B. Sore nipples:

1. Sore nipples are usually caused by the improper positioning of the baby on the nipple.
2. Ensure your baby is grasping the areola when sucking and not just the nipple.
3. Always break the neonate's attachment of the areola and nipple before removing them from the breast to prevent sore nipples.
4. Continuous suction pressure at the same spot of the nipple can be painful.
5. Change the position of the baby to change the "latching on" position of your baby's mouth.
6. If nipples become sore or cracked, start feeding on the less affected breast first.
7. Apply purified lanolin to nipples after each feeding.
8. Prevent mastitis with the following personal hygiene measures:
   a. Avoid using soap on nipples.
   b. Avoid decrusting the nipples of dried colostrum or milk.
   c. Change breast pads frequently.
   d. Wash hands before handling your breast and before breastfeeding.

Activity: As tolerated, extra rest is recommended after delivery.

(*continued*)

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CLIENT TEACHING GUIDE

POSTPARTUM: BREAST ENGORGEMENT AND SORE NIPPLES (*CONTINUED*)

Diet:

A. Breastfeeding mothers need extra liquids for milk production.
B. Drink 10 to 12 glasses of liquid a day.
C. Use caffeine in moderation (eliminate if possible).
D. Continue your regular diet and add about 500 extra calories per day.
E. Avoid gas-producing foods that may upset your baby's stomach.

Medications: Continue your prenatal vitamins while breastfeeding.

You Have Been Prescribed: Acetaminophen 500 mg or ibuprofen 600 mg every 3 to 4 hours for discomfort.

You Need to Notify the Office If You Have:

A. Temperature of 100.4°F or higher.
B. Pain that is not controlled with Tylenol or ibuprofen.
C. Flulike symptoms (fever, chills, malaise).
D. Red streaks on breast.
E. Headache with the symptoms listed here.
F. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

PRETERM LABOR

PROBLEM Premature contractions and early labor put your baby at risk for premature delivery. Babies born too soon are at risk for breathing problems, bleeding into their brain, infection, and bowel problems, to name a few.

CAUSE There are several predisposing factors for preterm labor (PTL), including previous premature delivery, smoking, incompetent cervix, multiple gestation (twins or triplets), and infection. In most cases, the cause of PTL is unknown.

PREVENTION You can decrease your risk for PTL by living a healthy lifestyle with a balanced diet, proper fluid consumption, and no smoking. Please review any previous PTL symptoms with your healthcare provider. Early recognition is key to success.

TREATMENT PLAN Treatment depends on the clinical picture. In general, you should remember:

A. Drink at least 8 to 10 glasses of noncaffeinated liquid a day; dehydration can increase contractions.
B. Empty your bladder every 2 to 3 hours.
C. Report any bladder infection symptoms, such as burning with urination, urinary frequency, or urgency, to your healthcare provider.
D. Avoid breast stimulation (including showers where the water stream is on your breasts); this can stimulate contractions.
E. Rest frequently. Rest means lying down on either side, not on your back.
F. Contractions and cramping happen more often in the evening and nighttime after having activity during the day.
G. Do not have intercourse or sexual stimulation without asking your nurse practitioner, certified nurse–midwife, or doctor.
H. Try to arrange for help with housework and childcare to help you decrease your physical activity and avoid lifting heavy items.
I. Take medications to stop contractions as directed.

Activity: Activity at home is based on how significant your PTL has been. You should follow the following activity guidelines:

A. Avoid strenuous physical activity and exercise, especially strength training and lifting items greater than 20 lbs.
B. Avoid working more than 40 hours per week, working night shifts, prolonged standing (more than a total of 8 hours or more than 4 continuous hours per 24-hour period), and heavy physical work, as this activity level has been variably associated with PTL and preterm birth.
C. Travel: It is unlikely that travel by car, train, or airline significantly increases the risk of PTL or preterm birth; however, you need to consider the risk of pregnancy complications away from the usual source of your medical care, as well as the availability of medical resources and your medical insurance coverage at your travel destination.

Diet: Diet as tolerated, or follow your prescribed diet. Drink 8 to 10 glasses of water each day. Avoid beverages with caffeine. Eat fresh vegetables, fruits, and bran cereal to avoid becoming constipated.

Medications: Continue taking your prenatal vitamin every day.

You Have Been Prescribed the Following: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Contractions or cramping more frequent than four in 1 hour.
B. A gush of fluid or blood from your vagina (it is normal to have spotting after vaginal exam or intercourse).
C. Pelvic pressure or low, dull backache.
D. Noticed that your baby is not moving as much as usual: less than 10 fetal movements in 2 hours after drinking and resting on your side.
E. Chest pain or difficulty breathing.
F. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

URINARY TRACT INFECTION DURING PREGNANCY: PYELONEPHRITIS

PROBLEM You have been diagnosed with an infection of the kidney (where urine is made). Bladder infections can spread to the kidney.

CAUSE Bacteria from the bladder can move up to the kidney and cause a kidney infection. Other causes are blockage in the urine system or having a catheter, or tube, in the bladder.

PREVENTION

A. Urinate frequently. Do not hold urine for long periods of time.
B. Empty your bladder as soon as you feel it is filling.
C. Urinate before and after sexual intercourse.
D. After urinating, always wipe from front to back with toilet tissue.
E. Do not wear tight underwear or pants; this can cause increased moistness and warmth in the perineal area.
F. Cotton panties are the best.
G. Wash your hands every time after going to the bathroom.

TREATMENT PLAN Antibiotics kill the bacteria that cause infection.

Activity: Rest; do not engage in strenuous physical activity.

Diet: Increase fluids; drink at least one large glass of water every hour while you are awake. Drink 100% cranberry juice to help fight and prevent urinary tract infections (UTIs). If you do not like the taste of cranberry juice, mix cranberry juice with water.

Medications: You will be prescribed antibiotics to kill the bacteria causing infection. The drugs may be changed if your urine culture results show a different bacteria. **It is very important that you finish all of your antibiotics even if you feel better. UTIs in pregnancy can lead to further complications.** You may need mild pain relievers if you have a lot of back pain. Medications such as Tylenol may be used to bring down fever. You may be prescribed a medicine to stop bladder spasm and pain.

You Have Been Prescribed an Antibiotic: _____

You Need to Take: _____

Finish all of your antibiotics even if you feel better.

You Have Been Prescribed the Following for Bladder Spasms: _____

You Need to Take: _____

This medicine will make your urine turn a different color.

You Need to Notify the Office If You Have:

A. Symptoms that worsen or do not get better during treatment.
B. New symptoms that develop during treatment, such as chest pain or shortness of breath.
C. Symptoms that return after treatment when you finish all of your antibiotics.
D. Difficulty taking your medication (you break out or vomit).
E. Other: _____

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CLIENT TEACHING GUIDE

VAGINAL BLEEDING: SECOND AND THIRD TRIMESTERS

PROBLEM Vaginal bleeding may occur during the second and third trimesters of pregnancy (more than 12 weeks). The bleeding may range from spotting of blood on your panties to bleeding like a menstrual period.

CAUSE A small amount of bloody mucous discharge or spotting may occur for about 1 day following a pelvic examination or sexual intercourse. This is normal if it is not associated with cramping or contractions.

Other causes of vaginal bleeding may be related to the location of the placenta (placenta previa) or premature separation (abruption) of the placenta from your womb. Placental abruption can be associated with cocaine use, cigarette smoking, and trauma (injuries from car wrecks or physical violence).

PREVENTION There is no known way to prevent most types of vaginal bleeding. If you have been diagnosed by ultrasound with placenta previa, you may be able to prevent bleeding by avoiding sexual intercourse and limiting your physical activity.

There is no known method of preventing placenta previa. Smoking has been associated with placental abruption and placenta previa. You should not smoke or at least you should try to cut down and stop smoking during pregnancy. When you stop smoking, it is also good for your baby's health after delivery.

TREATMENT PLAN

A. Treatment depends on the cause of your vaginal bleeding. You may be placed on limited physical activity.
B. Stop smoking. Ask your provider for a handout on tips to stop smoking.
C. You may need to modify your activity to avoid moderate or strenuous exercise, heavy lifting (greater than 20 lbs), or standing for prolonged periods (greater than 4 hours).
D. You may need to stop work.
E. You may need to arrange help for childcare, grocery shopping, and housework.

Diet: Eat fresh vegetables, fruits, and bran cereal to avoid becoming constipated. Drinking extra liquids (especially water) also helps to prevent constipation.

Medications: Continue taking your prenatal vitamins every day.

EMERGENT ISSUES/INSTRUCTIONS **Go to the ED/call 911 for:**

A. Bleeding like a period.
B. A gush of fluid or blood from your vagina.
C. Sharp, knifelike pain in your abdomen that does not go away.

You Need to Notify the Office If You Have:

A. Contractions or cramps, eight in 1 hour or four in 20 minutes.
B. Bloody, mucous discharge not associated with recent sexual intercourse or a pelvic examination.
C. Bright red or dark red vaginal spotting.
D. Pelvic pressure or low backache not relieved with emptying your bladder and resting on one side.
E. Noticed decreased movement of the baby.
F. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

WOUND INFECTION: EPISIOTOMY AND CESAREAN SECTION

PROBLEM You have an infection of your episiotomy or vaginal laceration site or Cesarean section incision.

CAUSE The cause is one or more types of bacteria that invaded the tissue following your vaginal delivery or Cesarean section. The bacteria may be from the vagina, the bowel, or the environment.

TREATMENT

A. Take your temperature if you have fever and chills.
B. Episiotomy:
  1. Wash hands before and after changing your sanitary pads and your baby's diaper.
  2. Wipe or pat dry from front to back after every urination or bowel movement.
  3. Apply and remove perineal pad from front to back.
  4. Change perineal pad at least every 4 hours and after each voiding or bowel movement.
  5. After you urinate or have a bowel movement, use a squeeze bottle to clean yourself. Position the nozzle between the legs, empty the entire bottle over the perineum, blot dry with toilet paper (do not rub), and avoid contamination from the anal area.
  6. Sitz bath: This is a shallow warm bath that helps clean and heal the perineum. Take them at least twice a day for 5 to 10 minutes each. You do not need to add soap or any medication to the water. Pat the perineum dry with a soft cloth; do not rub.
  7. Wash perineum with mild soap and warm water at least once daily.
  8. Do not insert anything into your vagina until your 6-week postpartum exam. Sex can be painful and may reopen the wound. Do not use tampons or douche.
C. Cesarean section incision:
  1. Wash hands before and after dressing change and wound care.
  2. Follow all of the previous directions for your bleeding.
  3. After showering, gently pat dry your abdomen.
  4. If wound is draining, cover it with clean dressing and call the office for instructions. Otherwise, leave it open to air.
  5. Clean incision with warm soapy water and pat dry.
  6. If your incision opens, notify your practitioner for further instructions.

Activity: Increased rest is recommended; try to lie down for a nap when the baby goes to sleep.

Diet: There are no dietary restrictions; eat well-balanced meals. Increase your fluid intake with an infection. Drink at least 10 to 12 glasses of liquid a day.

Medications: Continue your prenatal vitamins. You may take acetaminophen one to two tablets every 4 to 6 hours for your fever and/or discomfort. The maximum daily dosage for regular strength acetaminophen (325 mg) is 10 tablets in 24 hours and extra strength (500 mg) is six tablets in 24 hours.

You Have Been Prescribed the Following Antibiotic: _____

You Need to Take: _____

Take all of your antibiotics, even if you feel better, unless you have an adverse reaction to them. Then call the office.

You Need to Notify the Office If You Have:
A. Temperature that rises significantly or reaches 100.4°F.
B. Foul-smelling drainage from the incision or episiotomy/vaginal laceration site.
C. Increased pain or tenderness.
D. Separation of wound or incision.
E. An amount of vaginal bleeding that increases significantly or is heavier than a period.
F. Other: _____

Phone: _____

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CHAPTER 17

GYNECOLOGIC GUIDELINES

Rhonda Arthur and Jill C. Cash

AMENORRHEA

DEFINITION

A. Amenorrhea is the absence of menstruation when menses should occur. Amenorrhea is described as either primary or secondary based on occurrence either prior to menarche or after menarche.

1. Primary amenorrhea:
   a. Absence of menarche in ≥13-year-old females without signs of pubertal development or in ≥15-year-olds with developed secondary sexual characteristics and normal growth.
   b. Absence of menarche 3 years after thelarche or 5 years after thelarche, if that occurred before the age of 10.
2. Secondary amenorrhea:
   a. No menses for 3 or more months in a previously menstruating client or 9 months in a person who has previously experienced irregular menses.

INCIDENCE

A. Primary amenorrhea is uncommon. Amenorrhea that occurs as a result of agenesis of part of the reproductive system or a chromosomal anomaly is quite rare. Secondary amenorrhea may occur during an individual's reproductive life.

PATHOGENESIS

A. Physiologic: pregnancy, breastfeeding, and menopause.

B. Disorders of the central nervous system (hypothalamic): Hypothalamic amenorrhea, caused by a deficiency in pulsatile secretion of gonadotropin-releasing hormone (Gn-RH), is the most common cause of amenorrhea. Common factors causing hypothalamic dysfunction include a stressful lifestyle; excessive weight loss, as occurs in anorexia or bulimia; extreme exercise; medications, such as hormones, as occurs sometimes in clients using combined contraceptive methods, psychotropic drugs, or heroin; hypothyroidism; and maladies such as Crohn's disease or systemic lupus erythematosus (SLE).

C. Disorders of the outflow tract or uterine target organ: Abnormalities of this nature are uncommon. Examples include Asherman syndrome, which occurs as the result of the uterine lining being exposed to surgical instrumentation during surgical procedures such as dilation and curettage and agenesis or structural anomalies of the uterus, tubes, or vagina.

D. Disorders of the ovary: Examples include abnormal chromosomal syndromes such as Turner syndrome; conditions such as gonadal dysgenesis or agenesis, premature ovarian failure (POF); premature menopause (defined as cessation of menses before the age of 40 years); as a direct result of radiation or chemotherapy; and polycystic ovarian syndrome (PCOS).

E. Disorders of the anterior pituitary: Examples include prolactin tumors.

PREDISPOSING FACTORS

A. The disorder can affect any female between the ages of 13 and 55 years.

COMMON COMPLAINTS

A. "I haven't had a period in months." "I have periods only a few times each year."

B. "I have nipple discharge."

C. "I am 16 years old and have never had a period."

OTHER SIGNS AND SYMPTOMS

A. Absence of menses in a female individual who has previously experienced menarche, or in a person who has not experienced menarche.

B. Irregular, infrequent menses.

SUBJECTIVE DATA

A. Review the client's complete menstrual history, including age of onset (menarche), duration, frequency, regularity, and history of dysmenorrhea.

B. Review the client's pregnancy history.

C. Review the client's contraception history.

D. Note other medications the client is taking, such as hormones or psychiatric medications, as well as any recreational drug use.

E. Review general medical history including treatments received, such as chemotherapy for any prior cancers.

F. Inquire about any breast discharge.

G. Review the client's weight pattern.

H. Review sources of stress in the client's life.

I. Discuss exercise pattern and history.

PHYSICAL EXAMINATION

A. Check height, weight, body mass index (BMI), blood pressure (BP), and pulse.

B. Inspect:

1. Note overall appearance. Examine the thyroid. Inspect the breast/genitalia for Tanner staging.
2. Skin assessment: Check for central hair growth and hirsutism, which is androgen-responsive. Areas to inspect for coarse hair include the upper lip, chin, sideburns, neck, chest, lower abdomen, and perineum. ▶

The contributions of Amy Hull, Anne Moore, Brooke Faught, Cheryl A. Glass, Erin Shankel, Ginny Moore, Jordan Vaughan, Nancy Pesta Walsh, and Stefani E. Yudasz to this chapter in prior editions are acknowledged here.

C. Auscultate:
  1. Heart and lungs.
D. Palpate:
  1. Neck for thyroid enlargement.
  2. Abdomen for uterine enlargement compatible with pregnancy.
E. Pelvic examination:
  1. Inspect external genitalia. Note the pubic hair pattern for Tanner staging. Note any lesions, masses, or discharge.
  2. Speculum examination: Inspect the vagina and cervix for irregularity or lesions.
  3. Bimanual examination: Palpate for size of the uterus and for adnexal masses.

DIAGNOSTIC TESTS

A. The workup for primary amenorrhea is based on physical exam finding of presence or absence of breast development and/or uterus. Presence of a uterus and no breast development suggest lack of ovarian estrogen production. Finding of no breast development and no uterus is extremely rare and may indicate enzyme deficiency or lack of gonads. Findings of breast development and no uterus may have either androgen insensitivity syndrome or congenital absence of the uterus. Most clients with primary amenorrhea will have both normal development of breasts and a uterus. The etiology of amenorrhea in these clients is often found to be obstruction of menstrual flow related to vaginal septa or imperforate hymen.
B. Diagnostic tests to consider for primary amenorrhea with normal secondary sex characteristics:
  1. Pelvic ultrasound:
    a. Uterus present:
      i. Serum human chorionic gonadotropin (HCG).
      ii. Thyroid-stimulating hormone (TSH) to rule out thyroid dysfunction.
      iii. Prolactin: Normal is less than 20 ng/mL.
      iv. Follicle-stimulating hormone (FSH): Greater than 40 IU/mL indicates ovarian failure.
    b. Uterus absent:
      i. Genetic testing/karyotype.
      ii. Testosterone level.
C. Diagnostic tests to consider for primary amenorrhea with absence of secondary sex characteristics:
  1. FSH/Luteinizing hormone (LH).
  2. If FSH/LH elevated evaluate karyotype.
D. Diagnostic tests to consider for secondary amenorrhea:
  1. Serum HCG.
  2. TSH to rule out thyroid dysfunction.
  3. FSH: Greater than 40 IU/mL indicates ovarian failure.
  4. Prolactin: Normal is less than 20 ng/mL.

DIFFERENTIAL DIAGNOSES

A. Amenorrhea (primary or secondary).
B. Pregnancy.
C. Lactation.
D. Constitutional delay.
E. Hormonal contraception use.
F. Hypothalamic dysfunction.
G. Polycystic ovary syndrome.
H. Obesity related anovulation.
I. Thyroid disease.
J. Primary ovarian insufficiency.
K. Perimenopause.
L. Pituitary adenoma.
M. Androgen insensitivity syndrome.

PLAN

A. General interventions:
  1. If the client is pregnant, counsel regarding pregnancy and begin antepartum care.
  2. If other laboratory information points to an underlying cause for amenorrhea, treat as appropriate.
B. Client teaching: *See Client Teaching Guide for this chapter, "Amenorrhea."*
C. Pharmaceutical therapy for secondary amenorrhea:
  1. Short-term therapy: Progesterone:
    a. Norethindrone acetate (NETA) 5 mg once daily or medroxyprogesterone acetate (MPA) 10 mg each day for 10 to 12 days to offset possible adverse effects of unopposed estrogen.
    b. Withdrawal bleeding should occur within 7 to 10 days after finishing the medicine.
  2. Long-term therapy is based on the etiology:
    a. Hypothyroidism is treated with thyroid replacement.
    b. Functional hypothalamic amenorrhea requires treatment by a multidisciplinary team.
    c. Estrogen deficiency is treated with systemic combined hormonal contraceptives providing no contraindications to estrogen is present.
    d. Persistent secondary amenorrhea due to chronic anovulation requires ongoing progestin therapy to mitigate the risk of endometrial cancer. Clients may choose either cyclic or chronic endometrial suppression.
      i. Cyclic suppression: MPA 10 mg each day or NETA 5 mg once daily for the first 10 days of the cycle.
      ii. Chronic suppression: levonorgestrel (LNG) intrauterine device, a contraceptive implant, or progesterone only pills (POP). Anovulatory clients who do not have FHA may choose to use combined oral contraceptives if they have no contraindications to estrogen.

All anovulatory clients require therapeutic management. There is a risk of endometrial cancer with continued unopposed estrogen stimulation to the endometrium. There is a short latent period in progression from a normal endometrium to atypia to cancer, even in a young individual.

FOLLOW-UP

A. Reproductive age: The client should return after 6 months of treatment with progesterone or combined contraceptive products. Discontinue the hormones and assess for return of normal menses. If this does not occur, reinstitute progesterone or combined contraceptive therapy.
B. Perimenopausal: Maintain hormonal therapy. The client should return annually for preventive health visits.

CONSULTATION/REFERRAL

A. Consultation and referral for primary amenorrhea in the presence of anomalies and or abnormal lab results.
B. Referral is indicated for secondary amenorrhea in nonpregnant nonlactating females who have elevated prolactin levels, or other lab values requiring gynecologic follow-up.

INDIVIDUAL CONSIDERATIONS

A. Younger adult, mid-30s to mid-40s: Consider evaluating athletic triad of disordered eating, amenorrhea, and osteoporosis. Take time to also evaluate change in stressors and weight gain or weight loss.
B. Older adults: Irregular menses and amenorrhea are common during perimenopause. Provide anticipatory guidance ▶

and instructions regarding the need for contraceptive use until menopause is confirmed.

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BACTERIAL VAGINOSIS *(GARDNERELLA)*

DEFINITION

A. Bacterial vaginosis (BV) is an infection of the vagina caused by an alteration in the normal flora of the vagina, with an increase in anaerobes and gram-negative bacilli as well as a decrease in the *Lactobacillus* flora.

INCIDENCE

A. According to the Centers for Disease Control and Prevention (CDC), BV is the most common vaginal infection in individuals of childbearing age and is common in pregnant clients. It is not considered exclusively a sexually transmitted infection (STI).

PATHOGENESIS

A. The main etiologic agent in BV is an increase in anaerobes in the vagina. The reason this occurs is unknown. The normal lactobacilli of the vagina decrease, and vaginal pH is increased in BV. The organisms present in BV cause the level of vaginal amines to be high. These amines are volatilized when the pH is increased, causing the characteristic "fishy" odor.

B. Bacterial vaginitis is primarily polymicrobial, and the pathogens seen include *Bacteroides* species, *Peptostreptococcus* species, *Eubacterium* species, *Mobiluncus* species, *Gardnerella*, and *Mycoplasma hominis*. The incubation period is unknown.

PREDISPOSING FACTORS

A. History of STIs.
B. Multiple sexual partners.
C. Intrauterine device (IUD) use.
D. Factors that change the normal vaginal flora:
  1. Hormonal changes (menses, pregnancy).
  2. Medications: oral contraceptive use and antibiotic therapy.
  3. Foreign bodies in the vagina (tampons, IUDs), semen, and douching.

COMMON COMPLAINTS

A. Vaginal discharge (thin, white, gray, or milky).
B. Fishy vaginal odor.
C. Postcoital odor.

OTHER SIGNS AND SYMPTOMS

A. Asymptomatic.
B. Increase in odor after menses.
C. Itching and burning, occasional.

SUBJECTIVE DATA

A. Elicit onset, duration, and course of presenting symptoms.
B. Review any changes in the characteristics and color of vaginal discharge. Does the client's partner(s) have any symptoms?
C. Review any symptoms of pruritus, perineal excoriation, burning; signs of urinary tract infection (UTI).
D. Review medication and medical history.
E. Determine if the client is pregnant; note date of last menstrual period (LMP).
F. Question the client for a history of STIs or other vaginal infections.
G. Review previous infection, treatment, compliance with treatment, and results.
H. Note last intercourse date.
I. Elicit information about possible foreign body.
J. Review use of vaginal deodorants or sprays, scented toilet paper, tampons, pads, and douching habits.
K. Review change in laundry detergent, soaps, and fabric softeners.
L. Review use of tight restrictive clothing, tight jeans, and nylon panties.
M. Review history for seizures and anticoagulant therapy.

PHYSICAL EXAMINATION

A. Check temperature, if indicated, blood pressure, pulse, and respirations.
B. Inspect:
  1. Examine external vulva and introitus for discharge, irritation, fissures, lesions, rashes, and condyloma.
C. Palpate:
  1. Palpate the abdomen for masses or tenderness. Note enlarged or tender inguinal lymph nodes.
  2. Palpate the external perineal area for vulvar masses.
  3. "Milk" the urethra for discharge.
  4. Check for costovertebral angle (CVA) tenderness.
D. Pelvic examination:
  1. Inspect:
    a. Note the color, amount, and odor of discharge.
    b. Inspect the cervix:
      i. BV is a vaginosis rather than vaginitis. There is usually little or no inflammation of the vaginal epithelium associated with BV.
      ii. BV is associated with a pink, healthy cervix; "strawberry cervix" is seen with cervicitis due to *Trichomonas vaginalis* (TV).
      iii. A red, edematous, friable cervix is seen with *Chlamydia trachomatis* (CT).
  2. Speculum examination:
    a. Inspect side walls for adhering discharge.
    b. The clinical diagnosis of BV requires the presence of three of the following four signs:
      i. Homogeneous, white, adherent vaginal discharge.
      ii. Vaginal fluid pH greater than 4.5. Take smear for testing from the lateral walls of the vagina, not from the cervix, for accurate pH.
      iii. A fishy, amine-like odor from vaginal fluid before or after mixing it with 10% potassium hydroxide (positive whiff test). Semen releases the vaginal amines; therefore, there is an increase in odor after intercourse.
      iv. Presence of "clue cells" (squamous vaginal epithelial cells covered with bacteria, causing a stippled or granular appearance and ragged, "moth-eaten" borders) or coccobacilli forms, both in the fluid and adhering to the epithelial cells.
  3. Bimanual examination: Check for cervical motion tenderness (CMT) and adnexal masses. BV may be a risk factor for pelvic inflammatory disease (PID).

DIAGNOSTIC TESTS

A. Gram stain (considered the gold standard), though impractical for many outpatient practices to perform.

B. BV can be diagnosed by the use of clinical criteria; three of the following four are needed to make a clinical diagnosis (Amsel's diagnostic criteria):

1. Vaginal pH greater than 4.5; normal vaginal pH range is 4 to 4.5.

2. Clue cells on microscopic examination.

3. Homogeneous, thin white or gray discharge that coats the vaginal walls.

4. A fishy odor of the vaginal discharge before, or after, the addition of 10% potassium hydroxide (KOH) (wet prep with 10% KOH and normal saline prep); microscopic examination of vaginal secretions should always be done. Despite the availability of BV nucleic acid amplification testings (NAATs), clinical criteria remain useful in diagnosing symptomatic BV related to lower cost and more rapid diagnosis.

C. BV NAATs are available for diagnosis of symptomatic females. The test can be performed on either healthcare provider- or client-collected vaginal specimens.

DIFFERENTIAL DIAGNOSES

A. BV.
B. Vulvovaginal candidiasis.
C. Trichomoniasis.
D. Gonorrhea.
E. Chlamydia.
F. Presence of foreign body.
G. Normal physiologic discharge.

PLAN

A. General interventions:

1. Inform the client regarding other modalities for treating BV. These methods include the following:

a. Vinegar and water douches: one tablespoon of white vinegar in 1 pint of water. Douche one to two times a week.
b. *Lactobacillus* and *Acidophilus* culture four to six tablets orally daily.
c. Garlic suppositories: one peeled clove of garlic wrapped in a cloth dipped in olive oil inserted vaginally overnight and changed daily.

B. Client teaching: *See Client Teaching Guide for this chapter, "Bacterial Vaginosis."*

1. BV is *not* considered an STI.

C. Pharmaceutical therapy:

1. Drug of choice:

a. Metronidazole (Flagyl) 500 mg orally BID for 7 days. OR
b. Metronidazole gel 0.75% one applicator (5 g) vaginally at bedtime for 5 days:

i. Side effects of metronidazole include sharp, unpleasant metallic taste in the mouth, furry tongue, nausea/vomiting, loss of appetite, rash, and itching.
ii. Metronidazole may prolong prothrombin time in clients taking oral anticoagulants.

c. Clindamycin cream 2%, one full applicator (5 g) intravaginally at bedtime for 7 days.

2. Alternative regimens:

a. Clindamycin 300 mg PO BID for 7 days.
b. Clindamycin ovules 100 mg intravaginally once at bedtime for 3 days.
c. Secnidazole 2 g oral granules in a single dose (sprinkled onto applesauce or yogurt).
d. Tinidazole 2 g orally once daily for 2 days.
e. Tinidazole 1 g orally once daily for 5 days.

Clindamycin ovules and cream have an oil base that might weaken latex or rubber products (e.g., condoms and diaphragms). Use of such products within 72 hours following treatment with clindamycin ovules is not recommended.

3. Special considerations:

a. Pregnancy: BV has been associated with adverse pregnancy outcomes; therefore, all symptomatic pregnant clients at high risk for preterm birth who have confirmed BV require treatment. Support in the literature for treatment of asymptomatic pregnant individuals at high risk for preterm birth is inconsistent. Treatment of asymptomatic pregnant individuals at low risk for preterm birth also lacks consistent evidence. At this time, the evidence is insufficient to recommend routine screening for BV in asymptomatic pregnant individuals at either high or low risk for preterm birth.

i. Metronidazole 250 mg TID or 500 mg orally BID for 7 days or 0.75% metronidazole gel 5 g vaginally once daily for 5 days.
ii. Clindamycin ovules 100 mg intravaginally once at bedtime for 3 days.

FOLLOW-UP

A. Nonpregnant females: No follow-up is recommended unless indicated. Recurrence is common.

B. High risk for preterm delivery; pregnant females should be reevaluated 1 month after treatment.

C. Recommendations for treatment of BV in females infected with HIV are the same as for noninfected clients.

D. Consider treatment of the client's partner(s) in females with recurrent disease.

CONSULTATION/REFERRAL

A. Refer the client to a physician for recurrence that is unresponsive to therapies.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Clindamycin cream may be associated with increased adverse events in newborns and should not be used during the second half of pregnancy.

B. Partners:

1. Routine treatment of a client's partner(s) is not recommended at this time because it does not influence relapse or recurrence rates. BV can be transmitted between same-sex partners and they should seek evaluation if symptoms are present.

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BARTHOLIN CYST OR ABSCESS

DEFINITION

A. The Bartholin glands are small, round, nonpalpable mucous-secreting organs. They are located bilaterally in the posterolateral vaginal orifice. Obstruction of the duct causes the gland to swell with mucus and form a Bartholin cyst. The cause of obstruction is usually unknown but may be due to mechanical trauma, thickened mucus, neoplasm, stenosis of the duct, or infectious organisms not limited to sexually transmitted infections (STIs). The cyst may become infected, resulting in an abscess. Cysts develop more commonly in younger females and occurrence decreases with aging; therefore, it is important to rule out neoplasm in females over 40 experiencing Bartholin cyst.

B. The majority of individuals with Bartholin cyst are asymptomatic, but large cysts can cause pressure and interfere with walking and sexual intercourse. Abscesses generally develop rapidly over a 2- to 3-day period and are painful. Some abscesses may spontaneously rupture and often reoccur.

PREDISPOSING FACTORS

A. History of STIs.
B. Local trauma.

COMMON COMPLAINTS

A. Cysts can be asymptomatic and are found incidentally on physical exam.
B. Localized pain/irritation.
C. Dyspareunia.
D. Difficulty walking or sitting due to edema.

SUBJECTIVE DATA

A. Elicit onset, duration, and course of presenting symptoms.
B. Review any changes in the characteristics and color of vaginal discharge. Does the client's partner(s) have any symptoms?
C. Review any symptoms of pruritus, perineal excoriation, burning; signs of urinary tract infection (UTI).
D. Review the client's medication and medical history.
E. Determine if the client is pregnant; note the date of last menstrual period (LMP).
F. Question the client for a history of STIs or other vaginal infections.
G. Review previous infection, treatment, compliance with treatment, and results.
H. Note last intercourse date.

PHYSICAL EXAMINATION

A. Inspect:
  1. Examine external vulva and introitus for discharge, irritation, fissures, lesions, and rashes. Bartholin cyst will appear as a round mass, usually near the vaginal orifice causing vulvar asymmetry. Cysts are usually unilateral, tense, nontender, and without erythema. An abscess is usually unilateral, tense, painful, and erythematous.
B. Palpate:
  1. Palpate the external labia and lateral posterior introitus for enlarged cyst, mass, or tenderness; note discharge if present.

DIAGNOSTIC TESTS

A. Culture and sensitivity of purulent abscess fluid.
B. Cervical culture for STI (*Neisseria gonorrhea* and *Chlamydia trachomatis* [CT]).
C. Excisional biopsy in clients older than 40 or in any age where carcinoma is suspected.

DIFFERENTIAL DIAGNOSES

A. Bartholin cyst.
B. Bartholin abscess.
C. Neoplasm.
D. STI.
E. Sebaceous cyst.

PLAN

A. General interventions:
  1. Reassurance is indicated for clients younger than 40 with asymptomatic cysts. Incision and drainage (I&D) with culture and sensitivity is often required for symptomatic cysts and abscesses >3 cm. Because cysts and abscesses often reoccur, surgery to create a permanent opening from the duct to the exterior is often the definitive treatment. Two such surgical methods are placement of a wards catheter or marsupialization. Referral is indicated for I&D and other surgical intervention if the provider is not experienced with the procedures.
B. Client teaching:
  1. Warm sitz baths three or four times a day may encourage spontaneous rupture of abscess and provide comfort.
C. Pharmaceutical therapy:
  1. Treatment of Bartholin cysts/abscesses is primarily surgical (I&D and placement of Word catheter). Antibiotics are indicated in the case of related cellulitis or in the presence of cervicitis. First-line antibiotic choices should be effective against both aerobic and anaerobic bacteria.
    a. When antibiotics are indicated, select an antibiotic that covers *Escherichia coli* and methicillin-resistant *Staphylococcus aureus* (MRSA), such as trimethoprim 160 mg/sulfamethoxazole 800 mg BID or trimethoprim 160 mg/sulfamethoxazole 800 mg BID, plus amoxicillin/clavulanate 875 mg BID or trimethoprim 160 mg/sulfamethoxazole 800 mg BID, plus metronidazole 500 mg TID. If cervical culture is positive treat according to the latest Centers for Disease Control and Prevention (CDC) STI treatment guidelines.

FOLLOW-UP

A. Follow up with the provider if symptoms do not improve in 3 to 7 days.
B. Report to care provider if symptoms recur.

CONSULTATION/REFERRAL

A. Refer the client to a physician for recurrence that is unresponsive to therapies.
B. Clients older than 40 must be referred for surgical exploration and excision biopsy.

INDIVIDUAL CONSIDERATIONS

A. Older adults:
  1. Refer clients older than 40 years with cyst for excisional biopsy.
B. Pregnancy:
  1. Treatment with antibiotics is recommended due to the risk of complicated infection. Avoid the use of trimethoprim-sulfamethoxazole during the last trimester of pregnancy.
  2. Refer to an OB/GYN practitioner for recurrence.

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BREAST PAIN

DEFINITION

A. Benign breast disorders such as mastalgia, mastodynia, and fibrocystic breast changes are characterized by lumps or pain. The lumps may be physiologic nodularity, a ropy thickening, or distended fluid-filled cysts that are mobile. The pain may be cyclic or noncyclic, and it may be unilateral or bilateral.

INCIDENCE

A. This is a very common problem. Fifty percent or more of menstruating individuals experience breast pain. Two-thirds of breast pain are cyclic and occur in females in their 30s; one-third are noncyclic and may occur in clients at any age, but it tends to occur in individuals closer to menopause.

PATHOGENESIS

A. Dysplastic, benign histologic changes occur in the breast, such as hyperplasia of the breast epithelium, adenosis, microcysts and macrocysts, duct ectasia, and apocrine metaplasia.

PREDISPOSING FACTORS

A. Menstruation.

B. Ingesting substances containing methylxanthines (coffee, tea, chocolate, and cola drinks). Methylxanthines have been noted to contribute to breast pain by clinical observation only.

COMMON COMPLAINTS

A. "My breasts are painful, particularly just before my period."

B. "I have lumps in my breasts, and they hurt."

OTHER SIGNS AND SYMPTOMS

A. Tender breasts with palpation.

B. Ropelike masses, usually bilateral, with mobile, well-circumscribed masses that are cystic or rubbery.

SUBJECTIVE DATA

A. Elicit history of pain:

1. Note onset, duration, quality, location, and relation to menstrual cycle.

2. Ask: Is pain constant or intermittent? Sharp or dull? Focal or diffuse?

B. What has the client tried to alleviate the pain? Note what has worked, such as nonsteroidal anti-inflammatory drugs (NSAIDs), and what has been unsuccessful.

C. Note the client's family history of breast pain, lumps, fibrocystic breasts, or cancer.

D. Has there been trauma, such as being hit or having a rough sexual experience?

E. Does the client's breasts hurt during or after aerobic or nonaerobic exercise, such as running, aerobics, soccer, weightlifting, or basketball?

F. Does the client wear a good, supportive, properly fitted bra for daily use and for physical activity?

G. Has the client had any prior breast surgeries or biopsy?

H. Note medication history such as oral contraceptives, hormone therapy (HT), or phenytoin.

PHYSICAL EXAMINATION

A. Inspect:

1. Examine the breasts and note masses; dimples; changes in the skin; changes in the way the nipples are pointed while the client is in the sitting position with arms in neutral position in lap, above the head, or pressing in on hips.

B. Palpate:

1. Palpate the breasts; look for hard, fixed, or cystic masses in the breast, under the nipple, in the tail of the breast, and in the axilla. Use a standardized breast examination technique:

a. Assess the nipple for discharge.

b. Measure masses identified and use a clock face to describe their location.

c. Document the masses in the client's record, describing characteristics such as shape, size, mobility, and associated tenderness.

DIAGNOSTIC TESTS

A. Pregnancy test (as indicated).

B. If breast cancer is suspected:

1. Mammogram: If ordered, a diagnostic mammogram on the breast where the mass is palpated, rather than a screening mammogram, is appropriate. Mammograms may be difficult to interpret in individuals younger than 35 years due to the density of the breasts. Ultrasound with or without mammography may be indicated in these younger clients.

2. Ultrasonography is a first-line method to differentiate cystic masses from solid masses.

3. Fine-needle aspiration and biopsy (as indicated).

4. Excisional biopsy for solid lumps (as indicated).

DIFFERENTIAL DIAGNOSES

A. Fibrocystic breast changes with mastalgia.

B. Benign breast masses: fibroadenoma, benign phyllodes tumors, and duct ectasia.

C. Nipple discharge: duct ectasia, prolactin-secreting pituitary tumors.

D. Extramammary pain: costal chondritis, chest wall muscle pain, neuralgia, herpes zoster infection, and fibromyalgia.

E. Heart: angina pectoris.

F. Gastrointestinal (GI): gastroesophageal reflux disease.

G. Psychologic: anxiety and depression.

PLAN

A. General interventions:

1. Once malignancy is excluded, reassurance is an effective intervention in the majority of females with breast pain and normal findings. Use the term *fibrocystic changes* rather than *fibrocystic disease* to stress the functional nature of the problem. Stress that the pain is real but not a disease state.

B. Client teaching: *See Client Teaching Guide for this chapter, "Fibrocystic Breast Changes and Breast Pain":*

1. Encourage breast self-awareness to facilitate the client in becoming very familiar with the "usual" state of their own tissue so that if something new appears they may be more likely to recognize and report any changes to the healthcare provider. Evidence does not show that regular self-exams reduce deaths from breast cancer and may expose the client to increased false-positives and additional interventions.

2. "Lumpiness" that varies with the menstrual cycle is not abnormal. Breasts may normally be of different sizes. It is a change that is significant.

3. Evaluate medications as a potential cause/aggravator of pain. Consider changing dose or discontinuing HT for clients on HT with mastalgia, and continuous hormonal contraceptive use (skipping the placebo week) in clients currently using hormonal methods.

4. Symptomatic measures to relieve discomfort:

a. Good supporting bra, properly fitted. Adolescents whose breasts are maturing and perimenopausal females whose bodies are changing are two groups who often wear improperly fitted bras.

b. Local heat or ice application (whichever works best).

C. Diet:

1. Elimination of methylxanthines is a good idea, but the relationship of methylxanthines to breast pain is unproven in research studies. Sodium intake restriction has also been advocated but has not been supported by research.

2. Decreased caffeine use is commonly recommended; however, it has not been associated with a decrease in breast pain or tenderness.

D. Pharmaceutical therapy:

1. Topical nonsteroidal anti-inflammatory gel can be used for local mastalgia.

2. Antiestrogen treatment:

a. Tamoxifen citrate 10 mg/d for 3 months for severe breast pain.

b. Danazol (Danocrine) 200 mg daily for 6 months. *Note*: Doses less than 400 mg daily may not inhibit ovulation. The client must use a barrier contraceptive or intrauterine device (IUD) contraceptive measure. Although the side effect profile is significant, long-term symptomatic relief and histologic changes may be achieved.

3. Vitamins:

a. Vitamin E 200 IU BID supplementation has support in the literature for treating mastalgia.

4. Herbs:

a. Flaxseed 25 mg daily may have some benefit in the treatment of cyclic mastalgia.

b. Evening primrose oil (EPO) 1 to 3 g daily in divided doses has had mixed results.

FOLLOW-UP

A. Clients with fibrocystic changes need to be seen after 1 to 2 months of pharmacologic therapy to assess for complications and efficacy.

B. If a mass or abnormality is identified, published guidelines for diagnostic workup of breast masses should be followed to determine follow-up.

CONSULTATION/REFERRAL

A. Consult or refer the client to a physician when breast masses are identified.

B. Consult with a physician and refer the client to a surgeon if findings include a suspicious mammographic study, an abnormal needle biopsy, or a solid mass per ultrasonogram.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Consider plugged duct or mastitis with treatment as indicated.

B. Adults:

1. Organizations that formulate guidelines for female care disagree on breast cancer screening initiation, frequency, and discontinuation. The American Cancer Society (ACS) guidelines indicate that mammography screening for females at average risk are as follows:

a. **Females 40 to 44** of age should receive counseling on the risks and benefits of mammography and have the choice to initiate annual breast cancer screening with mammograms if they wish to do so.

b. **Females 45 to 54** of age should be counseled to obtain annual mammograms.

c. **Females 55 and older** can move to mammograms every 2 years or may continue annual screening. Screening should continue as long as a female is in good health and is expected to live 10 more years or longer.

d. **All females** should be informed of the benefits, limitations, and potential harms linked to breast cancer screening.

2. Care should be individualized considering potential risks, benefits, and limitations of screening. High-risk females may benefit from additional screening, including earlier initiation of screening and additional screening modalities such as ultrasound, tomosynthesis, and MRI.

3. Tomosynthesis has been proven to be a valuable screening modality. The addition of tomosynthesis to digital mammography for screening has been associated with an increased cancer detection rate, with a concomitant reduction in recall rate, for females with both dense and nondense breast tissue.

4. When clinical breast examination, mammography, and needle-aspiration biopsy are used, breast cancer detection rates are 93% to 100%.

C. Geriatrics:

1. Breast pain should carry heightened suspicion for potential existing breast cancer.

D. Partners:

1. Pain may inhibit sexual activity involving the breast.

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CERVICAL CANCER SCREENING GUIDELINES AND INTERPRETATION

INCIDENCE

A. In the United States, approximately 14,480 new cases of cervical cancer are diagnosed annually. Among these cases, approximately 4,290 deaths will occur. Cervical cancer is the seventh most common cancer in females. Since the 1950s, there has been a 70% reduction in the incidence of cervical cancer due to the use of Pap smear screening.

DEFINITION

A. The Pap test is a sampling of exfoliated cells collected from the cervix for cytologic evaluation. This screening test is designed to increase detection and treatment of precancerous and early cancerous cervical lesions, and to decrease morbidity and mortality from cases of invasive cervical cancer. Human papillomavirus (HPV) testing is another cervical cancer screening test. The HPV test looks for high-risk HPV (hRHPV). The HPV test can be used alone or at the same time as a Pap test. This is called cotesting.

RISKS

A. HPV increases the risk for cancer, including cervical, vulvar, vaginal, penile, and anal cancer, as well as cancer of the oropharynx. Most cervical cancers are caused by high-risk type HPV.

B. The following are risk factors for the development of cervical cancer:

1. HIV/immunosuppression.
2. Smoking.
3. Oral contraceptive use for 5 or more years.
4. Multiparity (three or more children).
5. Having several sexual partners.
6. History of sexually transmitted infection (STI), in particular HPV.
7. Diethylstilbestrol (DES) exposure in utero.

Sexually transmitted agents, particularly the HPV strains 16, 18, 31, 33, 39, and 42, are strongly associated with the development of cervical cancer. Virtually all cervical cancers are associated with hrHPV infection. HPV types 16 and 18 cause 70% of all cervical cancers.

SCREENING RECOMMENDATIONS

A. The American College of Obstetricians and Gynecologist (ACOG), the American Society for Colposcopy and Cervical Pathology (ASCCP), and the Society of Gynecologic Oncology (SGO) endorse the U.S. Preventive Services Task Force (USPSTF) cervical cancer screening recommendations.

1. Begin screening at age 21.
2. Age 21 to 29: screening with cytology alone from every 3 years. (ACOG, ASCCP, and SGO advise that primary hrHPV testing every 5 years can be considered as an alternative to cytology-only screening in people aged 25–30.)
3. Age 30 to 65: either cytology alone every 3 years; the Food and Drug Administration (FDA) approved primary hrHPV alone every 5 years, or cotesting (hrHPV and cytology) every 5 years.
4. Age greater than 65: no screening after adequate prior negative screening results (three consecutive negative cytology results, two consecutive cotesting results, or two consecutive negative hrHPV results within 10 years prior to stopping screening).
5. Hysterectomy with removal of cervix: no screening in individuals who do not have history of high-grade cervical precancerous lesions or cervical cancer.
6. Risk factors such as HIV infection, compromised immune system, in utero DES exposure, previous treatment of high-grade precancerous lesion or cervical cancer should receive individualized care.

B. The American Cancer Society (ASC) cervical cancer screening recommendations:

1. Individuals with a cervix begin screening at age 25 with primary HPV testing every 5 years.
2. If primary HPV testing is not available, then individuals aged 25 to 65 should be screened with cotesting every 5 years alone or with cytology every 3 years.
3. Individuals aged greater than 65 who have no history of cervical intraepithelial neoplasia (CIN) grade 2 or more severe disease in the last 25 years and who have had adequate documented negative prior screening in the prior 10 years discontinue screening.
4. These recommendations are for persons initiating cervical cancer screening or had all normal screening results in the past or have been returned to routine screening based on risk management consensus guidelines.
5. The recommendations do not apply to individuals at increased risk for cervical cancer due to solid organ transplant, stem cell transplant, HIV, or immunosuppression, or in utero DES exposure.

PAP TEST

A. The Pap test should include sampling from both the ectocervix and the endocervix to be considered "adequate for interpretation." The ectocervix is the cervical portion extending outward from the external cervical os. The endocervix extends upward from the external os to the internal os, where the cervical epithelium meets the uterine endometrium.

B. Cervical epithelium is composed of squamous and columnar cells. Squamous epithelium, appearing smooth and pink, lines the vagina and continues upward to cover variable amounts of the ectocervix. Columnar epithelium, darker red and more granular in appearance, lines the endometrium and continues downward to the cervix, lining the endocervical canal. The boundary between squamous and columnar epithelium is called the squamocolumnar junction (SCJ) and may occur anywhere on the ectocervix or endocervix.

C. The SCJ may regress at various times as a result of hormonal variation, particularly with menopause and during pregnancy. As new cells are formed and moved from the internal to the external cervix, those cells that formerly made up the SCJ are moved farther out to the perimeter of the os on the ectocervix. The area between the original and new SCJ is called the transformation zone. When columnar epithelium is visible on the ectocervix, appearing as a granular, red area, it is referred to as ectropion or ectopian. This condition is often seen in pregnancy and with combined oral contraceptive use.

D. Cervical cancer is a progressive disease with a number of histologically definable stages. Invasive cancer of the cervix and its precursors are detectable by cytology before they become symptomatic and before gross clinical signs appear. When symptoms are present, they usually include postcoital spotting, intermenstrual bleeding, and increased menstrual bleeding. Clients with invasive cancer may experience serosanguineous, or yellowish, vaginal discharge, which may be foul-smelling and intermixed with blood. ▶

E. Advanced disease may cause urinary or rectal symptoms, including bleeding. On speculum examination, advanced lesions appear as necrotic ulcers; with invasive disease, they may extend upward or protrude into the vagina.

BETHESDA SYSTEM (INTERPRETATION)

A. The 2014 Bethesda system is the most current classification system used to interpret cytologic findings. The article "The Pap Test and Bethesda 2014" by Naya and Wilbur published in *Acta Cytologica* is available for download at www.karger.com/Article/Pdf/381842.

1. Specimen type:
   a. Conventional slide method versus liquid-based preparation.
2. Adequacy of the specimen:
   a. Satisfactory for evaluation.
   b. Presence or absence of endocervical or transformational zone components.
   c. Quality indicators such as obscuring blood or inflammation.
   d. Unsatisfactory for evaluation and specific reason.
3. General categorization:
   a. Negative for intraepithelial lesion or malignancy.
   b. Others, such as endometrial cells in clients older than 45 years.
   c. Epithelial cell abnormality: See "Interpretation/result."

If an infection is indicated as a Pap test finding, evaluate the client and treat them accordingly. Pap tests are not diagnostic of vaginal or cervical infection. Institute therapy for infections after confirmation by wet prep and/or cultures and guided by cytologic reading. For example, if Candida is identified on Pap test results, evaluate the client in the office, confirm the finding, and treat the client with appropriate antifungal therapy.

4. Interpretation/result:
   a. Negative for intraepithelial lesion or malignancy (optional to report):
      i. Nonneoplastic cellular variations.
      ii. Reactive cellular changes associated with:
         1) Inflammation.
         2) Radiation.
         3) Intrauterine device (IUD).
   b. Organisms:
      i. Trichomonas. vaginalis.
      ii. Fungal organisms.
      iii. Shift in flora suggestive of bacterial vaginosis (BV).
      iv. Cellular changes consistent with herpes simplex virus (HSV).
      v. Cellular changes consistent with cytomegalovirus.
   c. Other: endometrial cells (in a client 45 years or older).
5. Epithelial cell abnormalities:
   a. Squamous cell abnormalities:
      i. Atypical squamous cells of undetermined significance (ASCUS): indicates slight cellular abnormality, without clear cause (e.g.,may be due to infection or cervical trauma).
      ii. Atypical squamous cells, cannot exclude high grade (ASC-H): This result notes that there are abnormal cells present that have some high-grade characteristics. An ASC-H Pap result is of higher severity than an ASCUS Pap.
      iii. Low-grade squamous intraepithelial lesion (LSIL): indicates the effect of HPV, mild dysplasia, or cervical intraepithelial neoplasia (CIN) I.
      iv. High-grade squamous intraepithelial lesions (HSIL): moderate and severe dysplasia, carcinoma in situ or CIN II, and CIN III.
      v. Squamous cell carcinoma.
   b. Glandular cell:
      i. Atypical glandular cells (AGC):
         1) Endocervical cells (not otherwise specified [NOS] or specify in comments).
         2) Endometrial (NOS or specify in comments).
         3) Glandular (NOS or specify in comments).
      ii. Atypical:
         1) Endocervical cells favor neoplastic variations.
         2) Glandular cells favor neoplastic variations.
      iii. Endocervical adenocarcinoma in situ.
      iv. Adenocarcinoma:
         1) Endocervical.
         2) Endometrial.
         3) Extrauterine.
         4) NOS.
6. Other malignant neoplasms: Specify.
7. Adjunctive testing.
8. Computer-assisted interpretation of cervical pathology.
9. Educational notes and comments appended to cytology report (optional).

MANAGEMENT OF ABNORMAL PAP TESTS

A. The 2019 American Society for Colposcopy and Cervical Pathology (ASCCP) Risked-Based Management Consensus Guidelines for Abnormal Cervical Cancer Screening Test and Cancer Precursors shifted recommendations to address individual client risk, not results. The recommendations are based on the individual's risk of CIN 3+ and are determined by a combination of client history and current lab results.

B. See and download the 2019 ASCCP algorithms for complete management options at www.asccp.org/guidelines.

ASCCP has a mobile device application. Look for the mobile application "ASCCP Management Guidelines" on iTunes.

C. Refer for treatment as indicated.

ADDITIONAL CONSIDERATIONS

A. According to the U.S. Preventive Services Task Force, ASCCP, and American College of Obstetricians and Gynecologists, screening guidelines apply to all people with a cervix, including females, transgender males, and individuals who have undergone supracervical hysterectomy. Begin cervical cancer screening at age 21 years. Individuals younger than 21 years should not be screened regardless of the age of sexual debut. Pap alone should be performed every 3 years for clients aged 21 to 29. Annual screening is not indicated for any individual.

B. Individuals who have a high-risk medical history (immunocompromised, HIV-positive, DES-exposed in utero, or ▶

a history of cervical cancer) are not included in the routine guidelines for screening and need individual risk-based plans.

C. The Advisory Committee on Immunization Practices (AICP) recommends routine vaccination of females and males aged 11 to 12 years. The series can be started as young as 9 years of age. AICP recommends HPV vaccination for everyone through age 26. HPV vaccination is not recommended for everyone older than 26, but some adults aged 27 to 45 may decide to get vaccinated based on shared decision making with a health care provider.

D. Client education regarding the prevention of cervical cancer, by avoiding exposure to HPV, should include reduction or elimination of high-risk activities. These high-risk activities include having sexual intercourse at an early age (younger than 18 years of age), having multiple sexual partners (defined as greater than five), and having partners with multiple partners. Use of condoms can reduce the risk of HPV as well as other STIs. Smoking cessation can also reduce the risk of cervical cancer. Identification and treatment of precancerous lesions can reduce the risk of invasive cervical cancer, so screening according to ASCCP guidelines should be recommended.

TREATMENT MODALITIES

A. Treatment is instituted based on the severity of the lesion and the presence of pathology within the columnar epithelium of the endocervix. Treatment options include:

1. Observation and repeat cytology.
2. Cryotherapy.
3. Loop excision of the transformation zone.
4. Laser of the transformation zone.
5. Cold-knife conization.

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CERVICITIS

DEFINITION

A. Cervicitis is acute or chronic inflammation of the cervix that is visible to the examiner.

INCIDENCE

A. Incidence is unknown due to multiple etiologies.

PATHOGENESIS

A. Acute cervicitis is primarily due to infection from the following organisms:

1. Bacteria:
   a. *Chlamydia trachomatis (CT).*
   b. *Neisseria gonorrhoeae.*
   c. *Mycoplasma.*
   d. *Ureaplasma.*
2. Viruses:
   a. Herpes simplex virus (HSV) type 2.
   b. Human papillomavirus (HPV).
3. *Trichomonas vaginalis* (TV).

B. Chronic cervicitis is primarily due to the following:

1. Trauma occurring during childbirth or instrumentation.
2. Infection.
3. Presence of foreign bodies (e.g., intrauterine devices [IUDs]).

PREDISPOSING FACTORS

A. Vaginal delivery.

B. Cervical procedures: laser, loop, or other excision procedures.

C. IUD.

D. Sexually transmitted infections (STIs).

COMMON COMPLAINTS

A. Copious mucopurulent vaginal discharge.

B. Postcoital bleeding.

OTHER SIGNS AND SYMPTOMS

A. Asymptomatic; may be found on routine gynecologic exam.

B. Thick, yellow vaginal discharge.

C. Dysuria.

D. Dyspareunia.

E. Vulvovaginal irritation or pruritus.

SUBJECTIVE DATA

A. Determine onset, duration, and course of symptoms. Is there any dyspareunia, pelvic pain, fever, or urinary symptoms?

B. Determine characteristics of the vaginal discharge.

C. Review the client's history of STIs.

D. Review the client's sexual history to include number of partners and partner symptoms (if any), use of sex toys, and sexual lifestyle.

E. Note last Pap smear and results. Has the client ever had an abnormal Pap, and if so how was it treated?

F. Note date of last menstrual period (LMP), use of contraception, and type(s) of contraception.

G. If the client has recently been pregnant, review their records for cervical cerclage, vaginal delivery with cervical laceration, or other complications.

PHYSICAL EXAMINATION

A. Check temperature, pulse, and respirations.
B. Inspect:
1. Observe generally for discomfort before, during, and after exam:
a. Observe the external vulva for Bartholin gland enlargement (Bartholin gland abscess is due primarily to infection by CT), lesions, irritation, fissures, and condyloma.
b. Note color, amount, and odor of vaginal discharge.
C. Palpate:
1. Back: Note costovertebral angle (CVA) tenderness.
2. Abdomen: Palpate for enlarged or tender inguinal lymph nodes.
D. Pelvic examination.
1. Speculum examination: Inspect cervix for inflammation and ectropion:
a. Inspect cervix for signs of inflammation. Be aware that cervical ectropion can be noted in approximately 20% of healthy young females (especially in teens and women using combined hormonal contraception). It represents columnar epithelium that is found farther out on the ectocervix, causing the cervix to appear granular and red. Presence of cervical erosion can appear similar to normal ectropy (or ectopy, as it is also known). Ectropy is uniform, whereas inflammation may appear with edges that are more irregular and may bleed to the touch of a cotton swab. A "strawberry cervix" (petechiae) is highly suggestive of trichomoniasis.
b. Check cervix for friability and bleeding when the cervix is touched with a cotton-tipped swab.
c. Assess the vagina and cervix for leukoplakia, lesions, polyps, and discharge. Assess vaginal walls for discharge and rugae.
d. Vesicular or ulcerated cervical lesions warrant testing for HSV, syphilis, and/or chancroid.
2. Bimanual examination: Check cervical motion tenderness (CMT); adnexal masses; uterine size, consistency, and tenderness:
a. Milk urethra for discharge.
b. Palpate Bartholin's glands.

DIAGNOSTIC TESTS

A. Wet prep (to assess for BV and trichomoniasis).
B. Symptomatic clients with cervicitis and negative wet mount should have a nucleic acid amplification testing (NAAT), culture, or other Food and Drug Administration (FDA) cleared diagnostic test for TV.
C. Cervical cultures for gonorrhea and chlamydia.
D. Consider testing for syphilis (rapid plasma reagin [RPR] or Venereal Disease Research Laboratory [VDRL] test).
E. Herpes simplex culture, if indicated.
F. Pap test and/or HPV testing as indicated by guidelines.
G. Urine culture and sensitivity, if indicated.
H. White blood cell (WBC) count, if indicated.

DIFFERENTIAL DIAGNOSES

A. Cervicitis.
B. CT.
C. GC.
D. Bartholin gland abscess.
E. Cervical neoplasm.
F. Cervical polyps.
G. HSV type 2.
H. Urinary tract infection (UTI).
I. Cervical ulceration, or erosion, from trauma: fingernail, cervical biopsy, postpartum, or sex toys.
J. Pelvic inflammatory disease (PID).

PLAN

A. General interventions:
1. Presumptive treatment for *C. trachomatis* and *N. gonorrhoeae* should be provided for females at increased risk (e.g., those <25 years, those with any recent change of sex partner, those not in a mutually monogamous relationship, or those with a sex partner who has a current, or recent, STI), especially if follow-up cannot be obtained. Trichomoniasis and BV should be treated if detected.
B. Client teaching: *See Client Teaching Guide for this chapter, "Cervicitis."*
1. Encourage clients to obtain routine care and Pap test evaluations per guidelines.
2. Clients should have no sexual intercourse for 1 week during treatment; they should avoid reinfection by abstaining from intercourse until sexual partners are adequately treated.
3. Avoid tampons and douches until antibiotics are completed.
C. Pharmaceutical therapy:
1. Drug of choice for cervicitis: doxycycline 100 mg orally BID for 7 days; azithromycin (Zithromax) 1 g orally in a single dose. Consider concurrent treatment for gonococcal infections if the client is at risk or lives in a community with a high prevalence of gonorrhea. (Partner treatment is determined by cause.)
2. Drug of choice for gonorrhea: ceftriaxone (Rocephin) 500 mg intramuscular (IM) injection (for clients weighing >150 kg) plus a single dose of azithromycin (Zithromax) 1 g orally. Requires partner treatment.
3. Drug of choice for HSV: Acyclovir 400 mg TID for 7 to 10 days for initial outbreak; acyclovir 400 mg TID for recurrent infections; acyclovir 400 mg BID for suppression.
4. Drug of choice for trichomoniasis in females: metronidazole (Flagyl) 500 mg BID for 7 days or tinidazole (Tindamax) 2 g orally in a single dose. Requires partner treatment.
5. Drug of choice for trichomoniasis in males: metronidazole (Flagyl) 2 g orally in a single dose or tinidazole (Tindamax) 2 g orally in a single dose.
6. Drug of choice for UTI: See the "Urinary Tract Infection (Acute Cystitis)" section in Chapter 15.

FOLLOW-UP

A. Clients treated for gonorrhea, chlamydia, or trichomoniasis should be retested 3 months after treatment.
B. Follow up with Pap and/or HPV testing as indicated by guidelines.

CONSULTATION/REFERRAL

A. Refer the client to a physician for suspected neoplasm and for cervicitis unresponsive to treatment.
B. If the cervix has a suspicious lesion, the client should be referred for colposcopy and/or biopsy regardless of cytology results. On physical examination, the cervix may be edematous and erythematous and may show exposed columnar epithelium. It may be friable. Reddened areas of the cervix may be seen around the cervical os. The irregularity and friability sometimes differentiate them from eversion; other times, colposcopy is required to make the distinction.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Cervical inflammation is common in early pregnancy. If an STI is diagnosed, nonteratogenic pharmacologic therapies must be implemented.

B. Partners:

1. A positive STI result warrants treatment of each sexual partner in accordance with Centers for Disease Control and Prevention (CDC) STI treatment guidelines.

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CONTRACEPTION

DEFINITION

A. Contraception is the intentional prevention of pregnancy by either or both sexual partners. Contraception can be mechanical, chemical, or surgical and is either reversible or nonreversible. Considerations in counseling regarding contraceptive choices include cost, efficacy, safety, and personal considerations such as personal belief systems and ability to use selected method.

B. The Centers for Disease Control and Prevention (CDC) U.S. Medical Eligibility Criteria for Contraceptive Use is a document to assist healthcare providers in counseling individuals and assists healthcare providers in determining safe and effective contraceptive methods individualized to client preferences and individual health issues. The CDC summary can be especially valuable for the busy clinician. Find the link to the summary charts at www.cdc.gov/reproductivehealth/contraception/mmwr/mec/summary.html.

INCIDENCE

A. Clients frequently visit primary care providers to request contraception and family planning education. There are 61.5 million females in the United States of childbearing age. Ninety-nine percent of females aged 15 to 44 years have used at least one contraceptive method. Unfortunately, approximately 45% of all pregnancies in the United States are unintended. At greatest risk for unintended pregnancy are females at both ends of the reproductive age spectrum: teenagers and perimenopausal individuals. Consistent use of a reliable and effective contraceptive method can greatly reduce the unintended pregnancy rate. Easy access and education regarding contraceptive use is a keystone in the prevention of unintended pregnancy.

SUBJECTIVE DATA

A. Review the client's complete menstrual history, including age of onset, duration, frequency, regularity, and degree of discomfort with menses. Review the first day of last menstrual period (LMP).

B. Review the client's pregnancy history.

C. Ascertain pregnancy intention: "Would you like to become pregnant in the next year?"

D. Review the client's contraception and history and satisfaction with method.

E. Review the client's sexual history.

F. Note other medications the client is taking, including over-the-counter (OTC) medications and supplements.

G. Ask the client whether they have had a major medical disease, including hypertension, cardiovascular incident, thromboembolic disease, diabetes, migraine headaches, gallbladder disease, or liver disease.

H. Review the client's substance abuse/use history.

I. Review the client's childhood illness and immunization record.

J. Note any allergies.

K. Review any pertinent family medical history, particularly asking about family history of cardiovascular events, myocardial infarctions (MIs) prior to age 50, and thromboembolic disease.

PHYSICAL EXAMINATION

A. Check height, weight, blood pressure (BP), pulse, and body mass index (BMI).

B. Inspect:

1. Note overall appearance. Look at the neck (thyroid). Inspect the breast/genitalia for Tanner staging. Tanner's Sexual Maturity Stages descriptions are located at childgrowthfoundation.org/wp-content/uploads/2018/05/Puberty-and-the-Tanner-Stages.pdf.

2. Skin assessment: Check for central hair growth, which is androgen-responsive. Areas to inspect for coarse hair include the upper lip, chin, sideburns, neck, chest, lower abdomen, and perineum.

C. Palpate:

1. Palpate the neck for thyroid enlargement.

2. Palpate the abdomen for enlarged organs or uterine enlargement compatible with pregnancy.

D. Auscultate:

1. Auscultate the heart and lungs.

2. If pregnancy is suspected, consider auscultating for fetal heart tones.

E. Breast examination:

1. Palpate for masses.

2. Assess for nipple discharge.

F. Pelvic examination:

1. Inspect external genitalia. Note pubic hair pattern for Tanner staging. Note any lesions, masses, or discharge.

2. Speculum examination: Inspect vagina and cervix. Note any vaginal discharge. Obtain Pap smear and cervical/vaginal cultures according to Pap smear guidelines.

3. Bimanual examination: Palpate the cervix and check for cervical motion tenderness (CMT). Palpate the size of the uterus and assess for adnexal masses.

4. Consider rectal examination as indicated.

DIAGNOSTIC TESTS

A. Urine: pregnancy test as indicated/urinalysis as indicated.

B. Serum: complete blood count (CBC) if indicated by history and/or symptomology.

C. Pap smear according to the American Society for Clinical Pathology (ASCP) guidelines.

D. Vaginal/cervical cultures testing for sexually transmitted infections (STIs) as indicated.

PLAN

A. General interventions:

1. Review all methods of contraception available with the client and partner, if available.

2. Consider all aspects of the client's history and make recommendations as appropriate. ▶

B. Client teaching: *See Client Teaching Guide for this chapter, "Contraception: How to Take Birth Control Pills (for a 28-Day Cycle)."*

1. Review the anatomy and physiology of the menstrual cycle and reproduction with all clients.

2. Review the risks, benefits, costs, use, and efficacy of contraceptive methods. Review perfect use versus typical use of method selected.

3. Review STI prevention and limitations of STI prevention as related to each method.

4. Assist the client in selecting the most appropriate method of contraception with regard to cost, efficacy, health status of the client, ability to use correctly and consistently, and the client's personal values.

5. Warning signs and information on when to call the care provider should be provided to all clients.

6. All individuals of childbearing age should be educated on the availability and proper use of emergency contraceptives.

7. Provide all clients information on the prevention of STIs.

METHODS OF CONTRACEPTION

A. Abstinence: refraining from sexual intercourse:

1. Advantages: easily accessible and inexpensive. Perfect use offers protection against STIs and pregnancy.

2. Disadvantages: user-dependent.

B. Barrier methods:

1. Male condom:

a. Advantages: Male condoms are easily accessible (OTC with no prescription needed) and relatively inexpensive. Condoms do not require daily intervention and offer some protection against STIs.

b. Disadvantages: Male condoms are technique-dependent for efficacy. Breakage and spillage can occur. Some condoms are made from latex, and those with latex allergies need to be aware and carefully check the label for latex content. Nonlatex condoms are available. Male condoms are intended for onetime use only.

c. Efficacy with perfect use of male condoms: Approximately 2 in 100 individuals will become pregnant each year. With typical use of male condoms, approximately 13 in 100 individuals will become pregnant each year.

2. Female condom:

a. Advantages: Female condoms are easily accessible (OTC with no prescription needed) and relatively inexpensive. Condoms do not require daily intervention and offer some protection against STIs.

b. Disadvantages: Female condoms are technique-dependent for efficacy. Slippage and spillage can occur. The female condom is intended for onetime use only and may be inserted up to 8 hours prior to intercourse.

c. Efficacy with perfect use of the female condom: Approximately 5 in 100 individuals will become pregnant each year. With typical use of the female condom, approximately 21 in 100 individuals will become pregnant each year.

3. Diaphragm:

a. Advantages: Diaphragms are nonhormonal and can be used for years with proper care. May be inserted up to 6 hours prior to intercourse.

b. Disadvantages: Diaphragms must be properly fitted by an experienced healthcare provider and are user-controlled. Placement is crucial to contraceptive benefit, and spermicide must be used. Must be removed within 24 hours due to risk of toxic shock syndrome (TSS). The client must have fit checked after childbirth and weight gain or loss. Urinary tract infections (UTIs) may be more frequent in diaphragm users, and some clients may experience sensitivity or allergy to spermicide. Avoid use during menses.

c. Efficacy with perfect use of the diaphragm: 6 in 100 individuals will become pregnant each year. With typical use of the diaphragm, 16 in 100 individuals will become pregnant each year.

4. Cervical cap:

a. Advantages: Cervical caps are nonhormonal and can be used for years with proper care. The cervical cap may be inserted and left in place up to 48 hours.

b. Disadvantages: Cervical caps must be properly fitted by an experienced healthcare provider and are user–controlled. Placement is crucial to contraceptive benefit, and spermicide must be used. It must be removed within 48 hours due to risk of TSS. The FemCap is made of latex and is not appropriate for latex-allergic clients. Some females may experience sensitivity or allergy to spermicide. Avoid use during menses.

c. Efficacy with use of the cervical cap is similar to the diaphragm.

5. Vaginal sponge:

a. Advantages: The vaginal sponge is a nonhormonal OTC polyurethane sponge that releases the spermicide nonoxynol-9. It can be used for multiple acts of intercourse over 24 hours.

b. Disadvantages: The vaginal sponge is user-controlled. Some clients may experience sensitivity or allergy to the spermicide. Avoid use during menses.

c. Efficacy with use perfects use of the vaginal sponge: Among parous individuals, 20 in 100 will become pregnant each year, and 9 nulliparous individuals will become pregnant each year. With typical use, 27 in 100 parous individuals and 14 nulliparous individuals will become pregnant each year.

C. Surgical:

1. Male sterilization:

a. Advantages: Sterilization is a very effective form of contraception. User does not have to remember to do anything prior to intercourse, and it is not user-dependent. Sterilization is permanent.

b. Disadvantages: Sterilization involves a surgical procedure. Insurance may not cover the cost of the procedure. A backup method of contraception is needed for 3 months after procedure.

c. Efficacy with perfect use of male sterilization: 0.1 in 100 individuals will become pregnant each year. With typical use of male sterilization, 0.15 in 100 individuals will become pregnant each year.

2. Female sterilization is the second most often used contraceptive method in the United States:

a. Advantages: Sterilization is a very effective form of contraception. User does not have to remember to do anything prior to intercourse, and it is not user-dependent. Sterilization is permanent.

b. Disadvantages: Sterilization involves a surgical procedure. If pregnancy does occur, there is a higher incidence of ectopic pregnancy. Insurance may not cover the cost of the procedure.

TABLE 17.1 TYPES OF IUDS

Copper IUD	Available Since	Years Effective	Use and FDA Approval	Possible Side Effects
Copper IUD (ParaGard)	1988	10 y	Approved only in parous females, but available to all females regardless of parity Can be used as emergency contraception when inserted within 5 days	Abnormal menstrual bleeding Higher frequency or intensity of cramps/pain
Hormonal LNG-IUDs	**Available Since**	**Years Effective**	**Use and FDA Approval**	**Possible Side Effects**
Mirena	2001	5 y	Approved only in parous females, but available to females regardless of parity	Intermenstrual spotting in the early months Reduces menstrual blood loss significantly
Skylar	2013	3 y	Approved for females regardless of parity	Hormone-related: headaches, nausea, breast tenderness, depression, cyst formation
Liletta	2015	3 y	Approved for females regardless of parity	
Kyleena	2016	5 y	Approved for females regardless of parity	

FDA, Food and Drug Administration; IUD, intrauterine device; LNG-IUD, levonorgestrel intrauterine device.

Source: From Henry J. Kaiser Family Foundation (2016, November 7). Intrauterine devices (IUDs): Access for women in the U.S. Retrieved from https://www.kff.org/womens-health-policy/fact-sheet/intrauterine-devices-iuds-access-for-women-in-the-u-s.

c. Efficacy with both perfect and typical use of female sterilization: 0.5 in 100 individuals will become pregnant each year.

D. Intrauterine device (IUD; Table 17.1):

1. Hormonal levonorgestrel (LNG)-medicated IUD:

a. Advantages: It is a very effective form of contraception. LNG-IUDs may be left in place for 3 to 5 years. User does not have to remember to use prior to intercourse. LNG-IUDs may reduce menstrual flow.

b. Disadvantages: Risks of any IUD include uterine perforation, increased spontaneous abortion, ectopic pregnancy, and pelvic pain and infection. Hormonal IUDs must be inserted by a qualified healthcare professional. IUD may be spontaneously expelled.

c. Efficacy with both perfect and typical use of the Mirena: 8 out of 1,000 (0.8%) become pregnant over 5 years using the IUD.

2. Nonhormonal (ParaGard):

a. Advantages: ParaGard is a very effective form of contraception. ParaGard may be left in place for 10 years. User does not have to remember to use prior to intercourse.

b. Disadvantages: Risks of any IUD include uterine perforation, increased spontaneous abortion, ectopic pregnancy, and pelvic pain and infection. ParaGard must be inserted by a qualified healthcare professional. IUD may be spontaneously expelled.

c. Efficacy with perfect use of the ParaGard: 0.6 in 100 individuals will become pregnant each year. With typical use of the ParaGard, 0.8 in 100 individuals will become pregnant each year.

Individuals who are not appropriate candidates for an IUD include those with recent pelvic infections, anatomic uterine abnormalities, and pregnancy. Caution should be exercised when considering an IUD in those who have multiple sexual partners; pelvic inflammatory disease (PID); immunosuppression; undiagnosed, irregular, or heavy menstrual bleeding; abnormal Pap smear; and difficulty obtaining follow-up care.

E. Pharmaceutical therapy:

1. Progestin-only pills (POPs; also known as mini pill):

a. Advantages: The POP is a safe hormonal alternative for clients who cannot take estrogen. It is preferred to combined oral contraceptives (COCs) for lactating individuals as it is not as likely to reduce milk supply. POPs are rapidly reversible and controlled by the individual taking them.

b. Disadvantages: The POP cannot be taken if the client has any contraindications to progestin use. POPs are less effective than COCs and must be taken daily at the same time, requiring strict adherence to regimen. May cause menstrual changes or follicular cysts. Does not consistently suppress ovulation but increases cervical mucus and thins endometrium.

c. Efficacy with perfect use of POPs: 0.3 in 100 individuals per year will become pregnant. With typical use, 9 in 100 per year will become pregnant.

2. Injection of Depo-Provera long-acting depot medroxyprogesterone acetate (DMPA):

a. Advantages: It is easy to use. The user only has to remember the injection every 3 months. May decrease vaginal bleeding. DMPA is a safe hormonal alternative for clients who cannot take estrogen.

b. Disadvantages: Clients cannot use DMPA if they have any contraindications to progestin use; it may cause amenorrhea or irregular vaginal bleeding in such clients. There is a risk of weight gain with DMPA use. Use of this method requires routine (13-week) visits to the provider's office for intramuscular or subcutaneous injections. DMPA does not provide protection against STIs. DMPA is associated with reversible decreased bone mineral density.

c. Efficacy with perfect use of DMPA: 0.2 in 100 individuals per year will become pregnant. With typical use, 6 in 100 per year will become pregnant.

d. DPMA should be administered during the first 5 to 7 days of the menstrual cycle or postpartum before resumption of intercourse (preferably, after lactation has been established). DMPA may be given at any time during the menstrual cycle if you can be reasonably certain the client is not pregnant. In the event of starting DMPA later than the first 5 to 7 days of the beginning of the menstrual cycle with reasonable certainty of no pregnancy, ▶

administer a pregnancy test and have the client use condoms for at least 1 week after injection. The Centers for Disease Control and Prevention (CDC) recommends up to a 2-week (15 weeks) grace period for administration DMPA. If a female is 15 or more weeks from previous injection, exclude pregnancy, offer emergency contraception (except ulipristal acetate) if needed, and have the client use condoms for at least 1 week after injection.

3. Contraceptive implant (Nexplanon): long-acting reversible etonogestrel implant:

a. A single-rod subdermal radiopaque implant

b. Advantages: Progestin implant is a safe hormonal alternative for females who cannot take estrogen. This is a long-acting (3 years) reversible contraceptive.

c. Disadvantages: Clients cannot use Nexplanon if they have any contraindications to progestin use. Possible insertion and removal complications exist.

d. The manufacturer strongly recommends that care providers who wish to insert and/or remove Nexplanon participate in training sessions. Only clinicians who have completed the training program are eligible to purchase the product. The link to request this training is www.nexplanon-usa.com/en/hcp/services -and-support/request-training/index.asp.

e. **Absolute contraindications to progestogen therapy:**

i. Active thrombophlebitis or thromboembolic disorders.

ii. Acute liver disease, liver tumors (benign or malignant).

iii. Known or suspected cancer of the breast, history of breast cancer, or other progestin-sensitive cancer.

iv. Pregnancy.

v. Undiagnosed, abnormal vaginal bleeding.

f. Efficacy: 0.1% of individuals will become pregnant each year.

4. Combined estrogen/progesterone contraceptives: Combined estrogen/progesterone contraceptives come in three delivery methods: oral pills, a transdermal patch, and a vaginal ring. Advantages and disadvantages and efficacy are similar regarding hormones, but there are some differences in delivery method.

a. Advantages: Combined contraceptives are easy to use, convenient, rapidly reversible, and controlled by the individual taking them. The transdermal patch is only changed weekly, and the vaginal ring is left in place for 3 weeks. In addition to predictable menses, combined contraceptives decrease menstrual flow and length of menses.

b. Disadvantages: Dependent on user, and oral pills must be taken daily. Exposure to hormones may not be suitable for certain females based on health status and risk. See absolute and relative contraindications that are not appropriate for some females in a prescriber's reference guide. Smoking in conjunction with use of combined contraceptive use increases cardiovascular risk and should be considered. Does not protect against STIs. Other medications, such as anticonvulsants and antibiotics, may interfere with the effectiveness of combined hormonal contraceptives and should be considered in prescribing.

c. Efficacy with perfect use of combined hormonal contraceptive: 0.3 in 100 individuals per year will become pregnant. With typical use, 9 in 100 individuals per year will become pregnant.

d. Prescribing considerations: Oral contraceptives come in combination extended cycle, combination monophasic, combination biphasic, combination triphasic, and progestin-only formulations. Side effects can be managed in consideration of pill composition (see prescribing reference guides such as the Monthly Prescribing Reference at www.empr.com). General considerations for pill selection include age, health history/status, and the client's preference. Because POPs are highly sensitive to consistency in timing, reserve prescriptions of them for clients who have contraindications to estrogen. Alternatives for COCs should be considered in individuals older than 35 who smoke due to increased risks of thrombolytic events. In asymptomatic adolescents, it is acceptable to prescribe COCs without an initial pelvic exam. For adolescents or anyone who may have difficulty remembering to take a daily pill, consideration should be given to prescription of the vaginal ring, patch, or other methods. See the office's prescriber's reference for complete information on safety side effects and contraindications.

e. **Absolute contraindications to estrogen therapy (ET):**

i. Acute liver disease.

ii. Cerebral vascular or coronary artery disease, myocardial infarction (MI), or stroke.

iii. History of or active thrombophlebitis or thromboembolic disorders.

iv. History of uterine or ovarian cancer.

v. Known or suspected cancer of the breast.

vi. Known or suspected estrogen-dependent neoplasm.

vii. Pregnancy.

viii. Undiagnosed, abnormal vaginal bleeding.

f. **Relative contraindications to ET:**

i. Active gallbladder disease.

ii. Familial hyperlipidemia.

iii. Smoker older than 35.

iv. Use with caution in clients with migraine with aura due to increased risk for ischemic stroke.

5. Spermicide (foam, film, gel, tablets, and suppositories):

a. Advantages: Spermicide is a nonhormonal OTC preparation and contains nonoxynol-9. It is inexpensive and easily accessible.

b. Disadvantages: Spermicide is user-controlled and if not used consistently will lead to contraception failure. Some may experience sensitivity or allergy to spermicide. Spermicide has a high failure rate.

c. Efficacy with perfect use of spermicide: 16 in 100 individuals will become pregnant each year. With typical use of spermicide, 21 of 100 will become pregnant each year.

F. Natural family planning (NFP):

1. Advantages: NFP is nonhormonal. It is inexpensive and easily accessible.

2. Disadvantages: NFP is user-controlled and depends on regularity of cycle and avoidance of intercourse. Can be complex for user and has a high failure rate. Does not protect against STIs.

3. Efficacy with use: Depending on fertility awareness method selected, efficacy ranges from 0.4% to 5% unintended pregnancies the first year with perfect use and 2% to 23% unintended pregnancies with typical use.

Additional information, training, and client teaching may be found at the following websites: Association of Reproductive Health Professionals: www.arhp.org; *Institute for Reproductive Health:* www.irh.org; *Planned Parenthood: www.plannedparenthood.org.*

G. Withdrawal:

1. Advantages: Withdrawal is nonhormonal, inexpensive, and easily accessible.

2. Disadvantages: Withdrawal is user-controlled. It does not protect against STIs.

3. Efficacy with use: With perfect use of the withdrawal method, 4 in 100 individuals will become pregnant the first year. Typical use of withdrawal method will result in 20 in 100 becoming pregnant the first year.

FOLLOW-UP

A. The client should return in 3 months of initiation of oral contraceptives, ring, and patch to assess BP, use, side effects, and satisfaction. Then yearly visits are recommended for health maintenance.

B. Clients on Depo-Provera injections should return every 3 months for follow-up injection and weight evaluation and then yearly for health maintenance.

C. Clients with a diaphragm should return for refitting with change in weight of 10 lbs (lost or gained) or when in a postpartum state.

CONSULTATION/REFERRAL

A. If the contraceptive method selected is one the practitioner is not experienced in providing (diaphragm, implant, IUD, surgical sterilization), refer to an experienced appropriate provider.

INDIVIDUAL CONSIDERATIONS

A. The risk of pregnancy is decreased but still needs to be considered for clients over 40 years of age. Midlife individuals who do become pregnant have increased risks, including venous thromboembolism (VTE) and hypertensive disorders:

1. Intrauterine contraception is one of the most effective and is safe to use in midlife individuals with few exceptions.

2. Progestin-only contraception is also a safe option because it is not associated with an increased risk of cardiovascular complications.

3. Combined (estrogen-progesterone) contraception can be safely used by midlife individuals who do not have cardiovascular risk factors (smokers, hypertension, and diabetes).

4. The U.S. Selected Practice Recommendation (U.S. SPR) guideline for contraceptive use states that contraception can be used by midlife females without medical contraindications until the age of menopause, at which time they may consider transition to systemic hormone therapy (HT).

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DYSMENORRHEA

DEFINITION

A. Dysmenorrhea is painful uterine cramping felt primarily in the lower abdomen but also in the lower back and upper thighs:

1. Primary dysmenorrhea: not associated with pelvic pathology; usually associated with ovulatory cycles. Occurs first day or two of the menstrual period. Usually worse the first day. Affects teens and individuals in their 20s. Often associated with prostaglandin-induced symptoms of diarrhea, nausea, vomiting, and/or headache.

2. Secondary dysmenorrhea: painful uterine contractions due to a pathologic etiology, such as endometriosis or pelvic inflammatory disease (PID). Occurs individuals primarily after menstruation has been established for several years, occurring in their 20s, 30s, and 40s. This type of pain is usually a progressive pain that intensifies over time.

INCIDENCE

A. Primary dysmenorrhea is very common, affecting 50% to 90% of young females to some extent at some time. It is the leading cause of absenteeism from work or school in females younger than 30. A smaller but significant group, perhaps 10%, miss days of school or work each month due to the incapacitating pain. The incidence of endometriosis is 8% to 30% of individuals of reproductive age.

PATHOGENESIS

A. Primary dysmenorrhea is due to myometrial contractions that are caused by prostaglandins in the secretory endometrium. The prostaglandins cause uterine ischemia through platelet aggregation, vasoconstriction, and dysrhythmic contractions.

B. Secondary dysmenorrhea is associated with pathologic conditions such as endometriosis, cervical stenosis, tumors, adhesions, adenomyosis, myomas, polyps, infection (PID), intrauterine device (IUD) malposition, retained products of conception, or nongynecologic causes. The pain of secondary dysmenorrhea may also be unrelated to menses.

C. In endometriosis, there are islands of endometrium found on peritoneal surfaces of the bladder, broad ligaments, fallopian tubes, ovaries, bowel, and cul-de-sac, as well as distant sites on the abdominal wall, vagina, lung, or other sites.

PREDISPOSING FACTORS

A. Female.

B. Reproductive age.

C. Ovulatory menstrual cycles.

D. Cervical stenosis, possibly.

COMMON COMPLAINTS

A. "I have painful periods."

B. "My menstrual cramps are terrible, particularly the first day of my cycle."

C. "My cramps are so bad I feel sick to my stomach and have diarrhea."

OTHER SIGNS AND SYMPTOMS

A. History of menstrual cramps just prior to the onset of the menstrual period and for the first 24 to 48 hours.

B. Pain beginning earlier; associated with intercourse, defecation, and urination; and lasting throughout the ▶

menstrual period is usually associated with endometriosis or adenomyosis.
C. Acute pain may be associated with infection (PID) or ectopic pregnancy.

SUBJECTIVE DATA

A. Obtain a complete menstrual history: age at menarche; frequency, duration, and regularity of periods; amount of flow, measured in the number of perineal pads or tampons used.
B. Ask the client about the location of the pain; note radiation and associated symptoms such as nausea, vomiting, or diarrhea.
C. Is pain rhythmic or spasmodic?
D. How old was the client when the pain began? Primary dysmenorrhea usually begins 2 to 3 years after menarche, with the onset of ovulatory cycles.
E. Inquire about the type of contraception used.
F. Obtain obstetric history and last menstrual period (LMP).
G. Is the pain related to the menstrual period, or does it occur prior to or independent of the menstrual period?
H. Does the client have dyspareunia?
I. Do they have urinary tract infection (UTI) symptoms?
J. What treatments have been tried and which were effective?

PHYSICAL EXAMINATION

A. Check height and weight to calculate body mass index (BMI), temperature (if indicated), blood pressure (BP), and pulse.
B. Inspect:
1. Examine the general body habitus for the client's adipose distribution on the buttocks and thighs.
2. Note breast development.
3. Observe the abdomen for distension.
4. Inspect the external genitalia for pubic hair pattern, lesions, discharge, and odor.
C. Palpate and percuss:
1. Abdomen for masses or tenderness.
D. Auscultate:
1. Heart, lungs, and abdomen for bowel sounds.
E. Pelvic examination:
1. Palpate the external genitalia for masses or areas of tenderness.
2. Speculum examination:
a. Inspect the cervix and vagina for discharge, lesions, ectropion, cervical erosion, and IUD string.
3. Bimanual examination:
a. Palpate the vagina and cul-de-sac areas for tenderness or masses.
b. Check:
i. Cervical position, mobility, and pain with mobility.
ii. Uterine size, mobility, shape, regularity, masses, position, and tenderness.
iii. Adnexa for masses (cystic or solid) and tenderness.
4. A normal pelvic examination is a significant finding in primary dysmenorrhea and often in endometriosis.

DIAGNOSTIC TESTS

A. Perform stat quantitative serum pregnancy test if suggested by history/physical exam.
B. Consider pelvic ultrasonography to rule out pelvic pathology.
C. Laboratory studies: urinalysis, hemoglobin, hematocrit, and white blood cell (WBC).
D. Consider vaginal and cervical cultures for *Chlamydia trachomatis* (CT) and gonorrhea (GC), if infection is suspected.
E. Pap and/or human papillomavirus (HPV) test as indicated per guidelines.
F. Urinalysis if indicated.

DIFFERENTIAL DIAGNOSES

A. Dysmenorrhea: The client's history and a normal pelvic examination are used to diagnose primary dysmenorrhea.
B. Complication of pregnancy: missed or incomplete abortion or ectopic pregnancy.
C. Endometriosis or adenomyosis.
D. Ruptured/ovarian cyst.
E. Infection: endometritis, salpingitis, PID, or pelvic adhesions.
F. Fibroid tumor.
G. Adhesions.
H. UTI.
I. Bowel disease: irritable bowel disease, inflammatory or diverticular bowel disease.

PLAN

A. General interventions:
1. Support client concerns and identify reality of discomfort. Identify primary source of pain if other diagnoses exist.
B. Client teaching: *See Client Teaching Guides for this chapter, "Dysmenorrhea (Painful Menstrual Cramps or Periods)" and "Contraception: How to Take Birth Control Pills (for a 28-Day Cycle)."*
1. Educate the client about the physiology of menstruation.
2. Teach the client that endometriosis is one of the leading causes of infertility. Discuss the consequences that may occur with chronic pelvic pain/endometriosis, which include compromised fertility issues.
3. Encourage activity and exercise, such as walking or swimming.
4. Advise warm baths or heating pads to help relieve some pain.
C. Pharmaceutical therapy:
1. Nonsteroidal anti-inflammatory drugs (NSAIDs):
a. They inhibit prostaglandin synthesis in the endometrium, thus decreasing uterine cramping while providing an analgesic effect.
b. The drugs should be started as the menstrual period begins. It is no longer considered the standard of care to begin the drugs a few days prior to onset of the menstrual period.
c. Prostaglandin inhibitors relieve dysmenorrhea in 80% of clients.
d. Take NSAIDs with food to avoid gastrointestinal (GI) upset and irritation.
2. Medications of choice for dysmenorrhea:
a. NSAIDs available over-the-counter (OTC):
i. Arylacetic acid derivatives: naproxen sodium 440 to 550 mg initially, then 220 to 550 mg Q12H as needed.
ii. Propionic acid derivatives: ibuprofen 800 mg initially, followed by 400 to 800 mg Q8H as needed.
iii. Anthranilic acid derivatives: mefenamic acid 500 mg initial dose, then 250 mg Q6H. The treatment maximum is 2 to 3 days.
iv. Cyclooxygenase-2 specific inhibitor: celecoxib 400 mg initially, followed by 200 mg Q12H as needed for treatment in clients 18 and older.

b. Hormonal control:
i. Oral contraceptive pills: Any combination pill is efficacious. Consider extended-cycle dosing to prevent having monthly periods.
ii. NuvaRing.
iii. Depo-Provera.
iv. Mirena IUD.
v. Nexplanon.

3. Medications of choice for endometriosis (refer to the "Endometriosis" section of this chapter for a detailed review):
a. NSAIDs.
b. Combined hormonal contraception.
c. With physician consultation or referral, danazol (Danocrine).
d. With physician consultation or referral, gonadotropin-releasing hormone (Gn-RH) agonists such as nafarelin (Synarel), leuprolide acetate (Lupron, Lupron Depot), goserelin acetate (Zoladex), and elagolix (Orilissa).
e. Levonorgestrel intrauterine system (LNG-IUS; Mirena, Skylar, Kyleena, and Liletta): Note: **Only trained healthcare providers who carefully review the selected device's manufacturer's package insert for indications and use as well as specific insertion instructions should place IUDs.**

4. Antibiotics as indicated for sexually transmitted infection (STI) and/or PID:
a. Refer to Chapter 18, "Sexually Transmitted Infections Guidelines".
b. Refer to "Pelvic Inflammatory Disease" section of this chapter

D. Vigorous exercise more than three times weekly has been associated with a decrease in symptomology.

FOLLOW-UP

A. Have the client return in 3 months. Encourage the client to give the treatment 3 months to determine effectiveness.

CONSULTATION/REFERRAL

A. Consult with a physician if dysmenorrhea does not respond to NSAIDs or oral contraceptives for further workup to determine the source of the pain.
B. Consider consultation with OB/GYN for laparoscopy or hysteroscopy to diagnose endometriosis; however, empirical treatment is recommended.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Uterine contractions in pregnancy could be preterm labor.

B. Adolescents:
1. Remember that endometriosis can occur in this age group. It is not an extremely rare finding.

C. Adults:
1. Endometriosis can be a disabling condition interfering with work and sexual relationships. It may continue into the perimenopausal period.

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EMERGENCY CONTRACEPTION

DEFINITION

A. Emergency contraception (EC) is a prospective method of pregnancy prevention when unprotected intercourse occurs (e.g., condom breakage, skipped oral contraceptive pills).

INCIDENCE

A. Approximately 45% of the annual pregnancies in the United States are unintended. One in nine sexually active females report using EC, with the highest use rate among females aged 20 to 24 years.

PATHOPHYSIOLOGY

A. Hormones in oral contraceptive pills temporarily disrupt ovarian hormone production and cause an absent or dysfunctional luteal phase hormone pattern. This results in an out-of-phase endometrium that is unsuitable for implantation. Hormone disruption may likewise interfere with fertilization and cause disordered tubal transport. Hormones or minerals (copper) in an intrauterine device (IUD) as well as an inflammatory response occur, which makes the endometrium unsuitable for implantation and interferes with fertilization and transport.

PREDISPOSING FACTORS

A. Rape.
B. Failure of other means of birth control, including broken condom, dislodged diaphragm or cervical cap, expelled IUD, lost or forgotten pills.
C. Unprotected intercourse.

COMMON COMPLAINTS

A. "I'm worried that I might get pregnant because the condom broke."
B. "My diaphragm slipped."
C. "I went on vacation and forgot my pills."

OTHER SIGNS AND SYMPTOMS

A. Unprotected intercourse.

SUBJECTIVE DATA

A. Elicit a menstrual history. When was the client's last menstrual period (LMP)? Are their periods regular?
B. What form of contraception was used, if any? What was the date and time of the unprotected encounter? (e.g., how many hours ago?)
C. Has the client experienced any early signs of pregnancy? If so, discuss.
D. Ask about early symptoms of pregnancy such as frequency of urination, nausea, breast tenderness, and late or missed period.
E. Ask the client about their feelings or plans if they should get pregnant.

PHYSICAL EXAMINATION

A. Physical examination is not required but may be useful if health status is unclear, if sexually transmitted infection (STI) testing is indicated, or client desires well-woman exam. If so, proceed with physical exam as indicated. ▶

B. Check blood pressure (BP), pulse, weight and body mass index BMI.

C. Inspect:

1. Eternal genitalia for lesions; note the client's pubic hair pattern.

D. Pelvic exam:

1. Speculum examination: Observe the cervix for a bluish color (Chadwick sign). Observe for vaginal discharge; note its color and odor.

2. Bimanual examination: Palpate the cervix for softening associated with early pregnancy. Palpate uterine size, contours, and position.

3. Abdomen. If the uterus is enlarged and is measured to be greater than 11 weeks' gestation, attempt to hear fetal heart tones with fetal Doppler.

DIAGNOSTIC TESTS

A. Pregnancy test: urine human chorionic gonadotropin (HCG if the first day of the client's last menses was 4 or more weeks prior or if menstrual history is uncertain. Serum HCG can detect pregnancy sooner than urine testing can.

DIFFERENTIAL DIAGNOSES

A. Pregnancy.

B. Abnormal uterine bleeding (AUB).

C. Amenorrhea from anovulation.

D. Polycystic ovarian syndrome.

E. Perimenopause.

PLAN

A. General interventions:

1. Review the client's medical history, contraceptive history, date of LMP, estimated date of ovulation, date of unprotected intercourse, and number of hours since the first and most recent unprotected intercourse.

2. Discuss the likely risk of pregnancy.

3. Explore the client's feeling about continuing pregnancy.

4. Decide whether a physical exam and pregnancy test are needed if there is a possibility of a pregnancy from the previous month.

B. Client teaching: *See Client Teaching Guide for this chapter, "Emergency Contraception pills."*

1. Discuss options, risks, failure rates, necessary follow-up, alteration of menstrual period, and warning signs of complications.

2. Discuss interim plan for contraception.

3. Advise the client to take oral contraceptive pills as prescribed or have IUD inserted within 96 hours of unprotected intercourse.

4. Treatment is most effective if taken within 12 to 24 hours for progestin estrogen methods and within 120 hours for progestin antagonist/antagonist.

5. Treatment is *not effective* in an already established pregnancy.

6. Educate the client about the possibility of menstrual cycle disturbance with the next menstrual period.

7. If menstrual bleeding does not begin within 3 weeks, evaluate for possible pregnancy.

8. EC is not associated with an increased incidence of abnormal outcome of pregnancy should pregnancy not be averted. EC does not always work.

9. This is *not* to be used as a primary contraceptive method.

10. Have prescription or pack of pills available for an emergency situation.

11. The IUD should be used only for females at low risk for pelvic inflammatory disease (PID) and when the client intends to continue use of the IUD for contraception.

C. Pharmaceutical therapy: Choosing the best method for EC should be based on day and time of unprotected intercourse; BMI; breastfeeding; and recent (within 5 days) use of pill, patch, or ring.

1. Copper IUD: The copper IUD is the most effective form of EC, with nearly 100% reported efficacy. It can be used up to 120 hours after unprotected intercourse.

a. A copper IUD (ParaGard) must be inserted within 5 to 7 days after ovulation in a cycle when unprotected intercourse has occurred. The advantage is that the IUD may be left in place for continuing contraception for 10 years.

b. Mechanism of action: The IUD leads to endometrial changes that prohibit implantation. Its main mechanism of action is to prevent fertilization through the effect of copper ions on sperm function (copper causes sperm decapitation). If fertilization has already occurred, copper ions affect the endometrium, rendering it unreceptive to implantation.

2. EC oral formulations:

a. Ella (ulipristal acetate) is a progesterone agonist/antagonist that is available only by prescription. It cannot be used in breastfeeding females and may be less effective in females with a BMI greater than 35. However, the efficacy of ulipristal acetate may be higher in overweight and obese females than progestin-only EC products like Plan B One Step, My Way, and Next Choice. EC efficacy tends to decline as BMI increases. For females who have a BMI of 26 or greater and who use progestin-only EC, pregnancy rates are similar to females who do not use EC at all. Ella's efficacy appears to decline in females when the BMI is 35 or greater.

i. One tablet is taken orally as soon as possible after unprotected intercourse within 120 hours (5 days).

ii. Common side effects include headaches, abdominal pain, and nausea. Less common side effects include dysmenorrhea, fatigue, and dizziness.

iii. Repeated use of Ella within the same menstrual cycle is not recommended.

iv. If vomiting occurs within 3 hours of taking a dose, take another dose.

v. Because Ella and the progestin component of hormonal contraception bind to receptor sites, females wishing to use hormonal contraception after use of Ella should not do so until 5 days after Ella use, but should be counseled on alternative nonhormonal pregnancy prevention methods.

b. Levonorgestrel EC, commonly called the "morning after pill," is available under multiple brand names such as Plan B One-Step, My Way, and Next Choice; it is available in one dose (1.5 mg tablet of levonorgestrel). These progestin-only ECs are available unrestricted as OTC medicines. They are less effective for females with a BMI greater than 25 and may not be effective for females with a BMI greater than 30.

TABLE 17.2 EMERGENCY CONTRACEPTION

Antiprogestin Emergency Contraception Pill Directions for Antiprogestin Pills: Take one pill within 120 hours.			
Brand		**Number of Pills Per Dose**	
Ella		One white pill	
Progestin-Only Emergency Contraceptive Pill Directions for Progestin-Only Pills: Take one dose within 72 hours.			
Brand	**Number of Pills Per Dose**	**Ethinylestradiol (mcg)/Dose**	**Levonorgestrel (mg)/Dose**
Plan B one-step	One white pill	0	1.5
Plan B two dose	Two white pills	0	0.75
Next choice	Two peach pills	0	1.5

i. Levonorgestrel 1.5 mg tablet should be taken as soon as possible after unprotected sex (no later than 72 hours).

c. Using a standard packet of combined hormonal oral contraceptive pills: two doses of a combination of ethinyl estradiol and norgestrel or levonorgestrel, 12 hours apart. Table 17.2 provides the equivalent dosing that may be used as an emergency contraceptive.

i. Method must be used within 72 hours of unprotected intercourse. Treatment is most effective if taken within 12 to 24 hours.

ii. Side effects of nausea and vomiting with EC are common. Take each dose with food. Take the antiemetic meclizine (sold as Dramamine II or Bonine) 30 minutes before the dose of medication.

iii. If vomiting occurs within 1 hour of taking a dose, take another dose.

iv. Educate the client about common side effects such as breast tenderness, abdominal pain, headache, and dizziness.

3. Provide information about all contraceptive methods and to initiate a regular method of contraception when possible.

FOLLOW-UP

A. Have the client return in 4 weeks if they do not have a menstrual period. Ideally, the client should begin a reliable method of contraception the day after completing the EC dosing.

CONSULTATION/REFERRAL

A. Consult a specialist if there is no withdrawal bleed within 4 weeks.

B. Additional information sources on EC for clients:

1. Not 2 Late is operated by the Office of Population Research at Princeton University and the Association of Reproductive Health Professionals: 888-NOT-2-LATE.

2. Website:

a. Planned Parenthood: www.plannedparenthood.org/learn/morning-after-pill-emergency-contraception.

SPECIAL CONSIDERATIONS

A. Pregnancy:

1. There is no increased incidence of anomalies if pregnancy does occur after the use of EC.

2. Perimenopause:

a. The chance of pregnancy still exists so the recommendation is for using progestin-only emergency contraception method such as Plan B.

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ENDOMETRIOSIS

DEFINITION

A. Endometriosis is ectopic endometrial tissue that exhibits hormonal responsiveness but is located outside the uterine cavity. Bleeding from this ectopic endometrial tissue causes pelvic inflammation and scarring, resulting in chronic pelvic pain and infertility. Endometrial lesions have been found in the vagina, gastrointestinal (GI) tract (especially the sigmoid colon), thoracic cavity, limbs, and gallbladder.

INCIDENCE

A. The true incidence of endometriosis is unknown. The incidence is thought to affect about 10% of the general female population. Endometriosis is the leading cause of secondary dysmenorrhea in adolescents. Endometriosis in adolescents is considered chronic and may progress if left untreated. Positive first-degree family history increases the risk up to sixfold. A concordance among twin sisters has been observed.

PATHOGENESIS

A. Retrograde menstruation is the most popular theory for the etiology of endometriosis. Menses are suspected of "flowing backward" through the fallopian tubes, resulting in the "seeding" of endometrial tissue outside the uterus.

PREDISPOSING FACTORS

A. Positive family history, mother and/or sister.

B. History of progressive dysmenorrhea.

C. History of prolonged uninterrupted menstrual cycles; first pregnancy at a late age; nulliparity.

D. Limited or no prior use of hormonal contraceptives.

E. Uterine abnormalities.

COMMON COMPLAINTS

A. Pain prior to period, pain with menstrual periods that increases over time (dysmenorrheal).
B. Pain with intercourse (dyspareunia).
C. Pain with bowel movements, may include constipation from the fear or pain of having a bowel movement (dyschezia).
D. Spotting and bleeding.

OTHER SIGNS AND SYMPTOMS

A. Dyspareunia and/or pain that radiates to the thigh and back.
B. Chronic, noncyclic pelvic pain.
C. Abnormal vaginal bleeding: premenstrual spotting and dysfunctional uterine bleeding (DUB).
D. Other bowel symptoms: diarrhea and rectal bleeding.
E. Urinary symptoms: dysuria, urgency, and hematuria.

SUBJECTIVE DATA

A. Review the onset, duration, and course of complaints.
B. Obtain a menstrual history: interval and duration of menstrual cycles and history of dysmenorrhea.
C. Review for a history of nonmenstrual pelvic pain/cramping.
D. Obtain a contraceptive history.
E. Question the client regarding change in bowel patterns or habits or pain with defecation.
F. Obtain a sexual history, including incidence of dyspareunia.
G. Note client parity and/or history of infertility.
H. Obtain a family history, asking about similar complaints in female relatives

PHYSICAL EXAMINATION

A. Temperature (if indicated), pulse, respirations, blood pressure (BP), and height and weight to calculate body mass index (BMI).
B. Inspect:
  1. Note general appearance for discomfort before, during, and after examination.
  2. Perform detailed external genitalia exam.
C. Auscultate:
  1. Abdomen for bowel sounds in all quadrants.
D. Palpate:
  1. Palpate abdomen for masses.
  2. Check for suprapubic tenderness.
  3. Back: Check for costovertebral angle (CVA) tenderness.
E. Pelvic examination:
  1. Speculum examination: Inspect the cervix for cervicitis; friability; and discharge color, odor, and amount. Note any cutaneous lesions of the vagina, or cervix that resembles "powder burn or chocolate spots."
  2. Bimanual examination: Check for cervical motion tenderness (CMT), adnexal masses; check uterine size, consistency, position, and mobility.
  3. Rectovaginal examination: The clinician may elect to perform a rectovaginal exam, though it is not required for diagnosis. A rectal examination is done because the uterus is often fixed in a retroverted position due to endometriosis. The endometrial nodules present on the posterior uterine wall, cul-de-sac, and uterosacral ligament may be distinguished better rectally. If this exam performed, the uterosacral ligaments are gently palpated for pain and nodularity. The rectum is evaluated for masses and polyps.

The most common indicator for endometriosis is a fixed retroverted uterus with nodularity. Palpation of endometrial implants may result in exquisite pain for the client.

DIAGNOSTIC TESTS

A. There are no specific diagnostic tests for endometriosis. Definitive diagnosis may be done by laparoscopy with tissue biopsy. Presumptive diagnosis is based on clinical signs and symptoms with imaging; this approach is usually appropriate as a conservative first step. If the client is unresponsive to treatment or if higher-risk therapies (e.g., donazol) are being considered, laparoscopy should be performed first to confirm diagnosis.
B. Serum beta human chorionic gonadotropin (HCG) to rule out ectopic pregnancy.
C. Complete blood count (CBC) to rule out infection.
D. Cervical culture for *Chlamydia trachomatis* (CT) and gonorrhea (GC) to rule out sexually transmitted infections (STIs) and pelvic inflammatory disease (PID).
E. Urine culture, if indicated.
F. Transvaginal ultrasonography to rule out cysts and masses.
G. GI series or barium enema, if indicated.

DIFFERENTIAL DIAGNOSES

A. Endometriosis.
B. Dysmenorrhea.
C. Ovarian cysts.
D. PID.
E. Premenstrual syndrome (PMS).
F. Mittelschmerz.
G. Trauma.
H. Appendicitis.
I. Pregnancy: normal, missed abortion, or ectopic.
J. GI: diverticular disease, inflammatory bowel disease (Crohn's disease [CD] or irritable bowel syndrome [IBS]).
K. Genitourinary: interstitial cystitis (IC) or urinary tract infection (UTI).

PLAN

A. General interventions:
  1. The client may be managed empirically if they are suspected of having endometriosis with pharmacologic and nonpharmacologic therapies.
  2. The American Society of Reproductive Medicine Practice Committee states that "endometriosis should be viewed as a chronic disease that requires a lifelong management plan with the goal of maximizing medical treatment and avoiding repeated surgical procedures."
B. Client teaching:
  1. Treatment goals include prevention of disease progression, alleviation of pain, and preservation of fertility. Treatment options include the following:
    a. Observation alone.
    b. Medical therapy or pharmacologic therapy.
    c. Referral or consultation for laparoscopic therapy, including laser vaporization and removal of adhesions, if other treatment strategies are unsuccessful.
  2. Continuation or recurrence of pelvic pain may necessitate assisting the client to manage their chronic pelvic pain and dysmenorrhea with nonsteroidal anti-inflammatory ▶

drugs (NSAIDs), hormonal options, and/or other nonnarcotic chronic pain therapies, such as visualization and biofeedback.

3. Hysterectomy and bilateral salpingo-oophorectomy are the only definitive cures for clients who do not wish to preserve their reproductive capacity. This should be considered only as a last resort for failed conservative treatment.

C. Pharmaceutical therapy:

1. Mild endometriosis (which does not cause frequent interference with work or school):

a. NSAIDs and combined hormonal contraception are considered the first-line therapy. If the client experiences pain during the week of withdrawal bleeding, they may take active pills/use ring continuously, omitting the placebo days. Avoid cyclooxygenase 2 (COX-2) inhibitors and hormonal contraceptives in clients who are trying to conceive.

b. Medroxyprogesterone acetate (MPA) 104 mg subcutaneously every 3 months.

c. MPA 150 mg intramuscularly (IM) every 3 months.

d. Levonorgestrel IUS.

e. Etonogestrel-releasing implant.

2. Moderate to severe complaints:

a. Gonadotropin-releasing hormone (Gn-RH) agonist:

i. Elagolix (Orilissa): First Food and Drug Administration (FDA)-approved oral treatment for the management of moderate to severe pain associated with endometriosis was released in 2018. Initiation: 150 mg/d delivers a dose-dependent partial suppression of estradiol production.

1) Maximum treatment duration with no comorbid conditions is 24 months. The maximum treatment duration with moderate hepatic impairment (Child–Pugh class B) is 6 months.

2) Maximum treatment duration with moderate hepatic impairment (Child–Pugh class B) is 6 months.

3) No dosage adjustment is required in females with mild hepatic impairment (Child–Pugh class A).

4) Elagolix 200 mg BID (400 mg total daily) delivers almost full suppression of estradiol production and may be used with coexisting dyspareunia:

5) Maximum treatment duration is 6 months.

6) Elagolix 200 mg BID is not recommended for moderate hepatic impairment.

7) The most common adverse reactions (>5%) in clinical trials included hot flashes and night sweats, headache, nausea, insomnia, amenorrhea, anxiety, arthralgia, depression-related adverse reactions, and mood changes:

8) Elagolix causes a dose-dependent decrease in bone mineral density (BMD), which is greater with increasing duration of use and may not be completely reversible after stopping treatment.

9) Contraindications to elagolix use:

a) Pregnancy.

b) Known osteoporosis.

c) Severe hepatic impairment.

d) Concomitant use of strong organic anion transporting polypeptide (OATP) 1B1 inhibitors (e.g., cyclosporine and gemfibrozil).

ii. Leuprolide acetate (Lupron) 3.75 mg by IM injection every month or 11.25 mg IM injection every 3 months.

b. Synthetic gonadotrophin releasing hormone:

i. Nafarelin (Synarel) nasal spray 200 mcg BID.

c. Hormones:

i. Consider adding oral norethindrone acetate 5 mg/d to minimize hypoestrogenic symptoms. This is called "add-back therapy."

Use of Gn-RH agonist, which acts to suppress ovulation, can result in side effects, including hot flashes, mood changes, and other menopausal symptoms. Use is restricted to 6 months (or 12 months if using add-back therapy) to avoid decrease in bone density. Expense of this therapy may preclude its use.

ii. Danazol 100 to 400 mg BID for up to 6 months:

1) Use of danazol, which acts to produce anovulation and hypogonadotropism, can result in androgenic side effects, including acne, hirsutism, weight gain, and voice changes that may not be reversible.

2) Other side effects, which are reversible, include decreased breast size, atrophic vaginitis, dyspareunia, hot flashes, and emotional liability.

3) Confirmation of diagnosis with laparoscopy is recommended prior to initiation of this treatment due to side effect profile.

iii. Progestin therapy:

1) Levonorgestrel intrauterine system (LNG-IUS), etonogestrel implant, or medroxyprogesterone acetate (as noted earlier).

FOLLOW-UP

A. Clients must return monthly while receiving Gn-RH agonist or danazol therapies to assess for symptom relief and side effect profile.

B. Clients with known endometriosis and a desire to conceive should be referred to a clinician specializing in infertility.

CONSULTATION/REFERRAL

A. The workup, evaluation, and medications for endometriosis are expensive. Consider referral to a women's health specialist for initial management. A prudent approach is recommended with a conservative treatment option; evaluate the results before trying another.

B. Refer the client for a surgical consultation for definitive diagnosis. Endometriosis may be suspected based on symptoms and physical examination. It cannot, however, be confirmed unless actually visualized by laparoscopy.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Infertility may be a presenting symptom. Treatment focus may be on endometriosis abatement and fertility support.

RESOURCE

Endometriosis Association: endometriosisassn.org

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FEMALE SEXUAL DYSFUNCTION

DEFINITION

A. Any persistent problem with desire, sexual response, or function, which may affect the client and their relationship and occurs for at least 6 months. It is classified into subtypes:

1. Desire disorder: lack of interest or desire (most common).

2. Arousal disorder: inability to become aroused during sexual activity; absent or reduced genital sensations.

3. Orgasm disorder: delay, absence, or decreased intensity of orgasm:

a. Primary: The client has never had an orgasm.

b. Secondary: The client has achieved orgasm in the past, but is unable to achieve orgasm at the time of presentation.

4. Pain disorder: genitopelvic pain/penetration disorder (formerly dyspareunia and vaginismus). This is described as pelvic or vulvovaginal pain during vaginal penetration, anxiety, and/or fear related to the thought of vaginal penetration, or marked tightening of pelvic floor muscles (PFMs) during vaginal penetration.

INCIDENCE

A. Female sexual dysfunction affects an estimated 43% of U.S. females. Prevalence rate changes related to age groups. Approximately 10% of females aged 18 to 44, 15% of females 45 to 64 years, and 9% of females aged 65 to 85 years experience female sexual dysfunction.

PATHOGENESIS

A. Multiple models exist to describe the phases of normal sexual function:

1. Masters and Johnson: consists of the stages of excitement, plateau, orgasm, and resolution.

2. Kaplan and Leif: consists of desire, excitement, and orgasm.

3. Basson: consists of emotional intimacy, sexual stimuli, psychological factors, and relationship satisfaction.

B. Sexual dysfunction includes various biological, psychological, and social components:

1. Biological factors include aging, medical conditions such as diabetes and hypertension, and declining testosterone or estrogen.

2. Psychological factors include depression or anxiety, history of sexual abuse, childhood trauma, personality disorders, body image disorders, and perceived stress.

3. Social factors include cultural or religious values, relationship issues, career issues, financial hardship, and household responsibilities.

C. Diagnosis is made based on the *Diagnostic and Statistical Manual of Mental Disorders* Fifth Edition (*DSM-5*; 2013) and requires the following: Symptoms must be present for a minimum of 6 months, a female experiences personal distress, symptoms are not a result of substance or medication use or medical conditions, and symptoms are not related to a nonsexual medical disorder.

D. Orgasm disorders can be caused by neurologic and/or vascular disease such as spinal cord injury, diabetes, or multiple sclerosis.

PREDISPOSING FACTORS

A. Emotional:

1. Depression.
2. Mood disorders.
3. Poor body image.
4. Low self-esteem.
5. Fatigue.
6. Stress.

B. Poor health status:

1. Spinal cord injury.
2. Diabetes.
3. Premature ovarian failure (POF).
4. Trauma.

C. Partner relationship issues.

D. Medications that are associated with low sexual desire in females:

1. Serotonin-specific reuptake inhibitors (SSRIs).
2. Anxiolytics.
3. Antihistamines.
4. Anticholinergics.
5. Calcium channel blockers.
6. Angiotensin-converting enzyme (ACE) inhibitors.
7. Oral contraceptives.
8. Anticonvulsants.
9. Opiates.
10. Illicit drugs.

E. History of sexual abuse may contribute to low desire.

F. Advancing age: menopausal females affected more frequently. Low sexual desire may result from decreasing hormone levels, specifically testosterone and estrogen levels. Low estrogen levels are also linked to vulvovaginal atrophy (VVA) and dyspareunia, both of which may decrease sexual desire in females.

COMMON COMPLAINTS

A. Females:

1. Absence of orgasm.
2. Pain during vaginal penetration.
3. Difficulty relaxing PFMs to allow vaginal penetration.

B. Females and males:

1. Lack of interest or desire (most common).
2. Inability to become aroused.
3. Pain with intercourse.

C. Males: Common complaints for males are specifically discussed in Chapter 15, "Genitourinary Guidelines," in the sections "Sexual Dysfunction, Male:Erectile Dysfunction" and "Sexual Dysfunction, Male: Premature Ejaculation."

OTHER SIGNS AND SYMPTOMS

A. Vaginal discharge.

B. Vulvar itching.

C. Vulvar pain, described as stinging, burning, irritation, raw sensation.

SUBJECTIVE DATA

A. Include full medical, gynecologic, and sexual history, using open-ended questions:

1. Assess the client for signs of depression, anxiety, and sexual concerns.
2. Review the medication list with the client. Specifically ask about the types of medications that may contribute to low sexual desire:
    a. SSRIs.
    b. Anxiolytics.
    c. Antihistamines.
    d. Anticholinergics.
    e. Calcium channel blockers.
    f. ACE inhibitors.
    g. Oral contraceptives.
    h. Anticonvulsants.
    i. Opiates.
    j. Illicit drugs.
3. Assess for medical conditions that contribute to sexual dysfunction, which may include the following:
    a. Chronic diseases (cardiovascular disease, diabetes, kidney or liver failure).
    b. Neurologic disorders.
    c. Hormone imbalances.
    d. Alcoholism.
    e. Illicit drug use.
    f. Evaluate for causes of pelvic pain: vaginal dryness, vaginal discharge, or vaginal infectious causes (such as sexually transmitted infections [STIs], yeast infections, or pelvic inflammatory disease [PID]).
4. Evaluate for history of previous pelvic disorders or surgery (fibroids, endometriosis, malignancy, uterine/bladder prolapse, or episiotomy).
5. Evaluate for the degree of distress that this may cause the client.

B. The use of self-report screening tools is recommended to identify clients with low sexual desire. These tools may include the following:

1. Decreased Sexual Desire Screener: found at Decreased Sexual Desire Screener (DSDS; herhsdd.com).
2. Brief Sexual Symptom Checklist for Women: may be downloaded from www.researchgate.net/figure/The-modified-Brief-Sexual-Symptom-Checklist-for-Women-BSSC-W_fig1_277610474.
3. Female Sexual Function Index: found at Female Sexual Function Index (FSFI) Questionnaire Calculator (thecalculator.co).

PHYSICAL EXAMINATION

A. Vital signs: Check blood pressure (BP), pulse, and respirations.

B. Inspect:

1. Thyroid for presence of nodules.
2. Breasts for presence of nipple discharge if history warrants.
3. Skin for hirsutism, acne, alopecia, and truncal obesity may indicate hyperandrogenism.

C. Palpate:

1. Thyroid for presence of nodules.
2. Breasts for presence of nipple discharge if history warrants.

D. Pelvic Examination:

1. A gynecologic exam focused on areas of concern identified in the history.
    a. Inspect:
        i. Examine external genitalia for erythema, lesions, atrophy, or unusual discharge.
        ii. Inspect for vulvar dermatoses, such as lichen sclerosus or lichen planus. Lichen sclerosus may look like white skin discolorations or wrinkled patches of skin. Severe cases may have bleeding or ulcerated lesions. Lichen planus may look like purple-colored lesions or bumps with flat tops. Thin white lines or blisters may appear over the lesions.
        iii. Inspect for pelvic floor prolapse and PFM contraction.
    b. Palpate:
        i. External genitalia for presence of pain.
    c. Speculum examination:
        i. Assess for atrophy (common in postmenopausal individuals, PFM, strength, masses, prolapse, and deep pelvic pain.
    d. Bimanual examination:
        i. Check for cervical motion tenderness (CMT), uterine size, and position.
        ii. Check adnexa for masses or tenderness.

DIAGNOSTIC TESTS

A. Laboratory tests should be performed as indicated by the history and physical examination for any medical conditions that may contribute to low desire.

B. Pap and HPV testing as indicated by guidelines, STI testing, wet prep as indicated.

C. Serum testing: CBC, vitamin D, thyroid function tests, prolactin level if underlying disease is suspected.

D. Consider hormone testing for clients if there is a suspicion of gonadal dysfunction (POF or polycystic ovarian syndrome).

E. Transvaginal ultrasound may be warranted if pelvic pain is elicited upon examination.

DIFFERENTIAL DIAGNOSES

A. Arousal disorder.

B. Desire disorder.

C. Orgasm disorder.

D. Pain disorder.

E. Vulvar dermatoses, such as lichen planus or lichen sclerosis.

F. Vaginal atrophy.

G. Infectious issues, such as STIs, candidiasis, or bacterial vaginosis (BV).

H. Depression or anxiety.

I. Cardiovascular disease.

J. Diabetes.

K. Thyroid disorder.

L. Neurologic disorder.

M. Hormonal imbalance.

PLAN

A. General interventions:

1. Set realistic goals for treatment.
2. Empower clients to take an active role in treatment plan. Encourage discussion about anatomy and sexual function, allowing the client to ask questions as they are comfortable.
3. Office-based therapy may be useful for providers who wish to include this in their practice. The Permission, Limited Information, Specific Suggestion, Intensive Therapy (PLISSIT) model is one office-based counseling model detailed as follows:
    a. Permission: Clients are given permission for full discussion of the topic. ▶

b. Limited information: The provider gives educational information on sexual function and sexual dysfunction in the form of handouts or videos.
c. Specific suggestion: The provider gives very specific advice tailored to each client and their presenting issues.
d. Intensive therapy: The provider makes a referral for individual or couples therapy.

B. Client teaching:

1. Educate about normal anatomic and sexual functions.

2. Encourage healthy lifestyle behaviors of diet, exercise, avoiding tobacco, and minimizing stress.

3. Educate clients about the use of vaginal lubricants to assist with vaginal dryness or dyspareunia: KY Jelly or Astroglide: Dose as needed with intercourse; can also try natural remedies at home lubricants such as egg whites, coconut oil, or olive oil.

C. Pharmaceutical therapy is considered when nonpharmacologic interventions are not successful:

1. Vaginal estrogen for treatment of vaginal atrophy:
a. Vagifem: 10 mcg, one tablet in vagina nightly for 2 weeks, then twice a week for maintenance.
b. Estring: one ring, placed intravaginally every 3 months.
c. Premarin: 0.625 mg/g: Insert 0.5 g/d for 21 days on, 7 days off. May adjust dose based upon client response.
d. Estrace: 100 mcg/g: Insert 2 to 4 g in vagina nightly for 2 weeks, then gradually taper to half the initial dose for 1 to 2 weeks, then a maintenance dose of 1 g one to three times a week for maintenance. Taper or discontinue at 3- to 6-month intervals.

2. Ospemifene:
a. A nonestrogen compound for treatment of moderate to severe dyspareunia related to vaginal atrophy in postmenopausal clients.
b. Dosage: 60 mg once/d.
c. If using ospemifene, prescribe opposing progestin in clients with intact uterus.
d. Ospemifene is contraindicated in clients with undiagnosed abnormal vaginal bleeding; those with history of venous thromboembolism, pulmonary embolism, or breast cancer; and females of childbearing age.

3. Prasterone (Intrarosa):
a. A steroid indicated for treatment of moderate to severe dyspareunia due to menopause.
b. Dosage: one vaginal insert once daily at bedtime, using the provided applicator.
c. Contraindicated in clients with breast cancer, dysfunctional uterine bleeding, endometrial cancer, ovarian cancer, or vaginal bleeding, dysfunctional uterine bleeding of undetermined origin, pregnancy, and breastfeeding.

4. Flibanserin:
a. Multifunctional serotonin agonist and antagonist. Indicated in the treatment of hypoactive sexual desire (HSSD) in premenopausal clients.
b. Dosage 100 mg taken at bedtime (maximum benefit may take several weeks to occur).
c. Boxed warning: "Patients should wait 2 hours after alcohol consumption and that severe hypotension may occur with the use of moderate or strong CYP3A4 inhibitors."
d. Stop treatment if no benefit observed at 8 weeks.

5. Bremalanotide:
a. Nonselective melanotropin receptor agonist analog indicated in the treatment of HSSD in menopausal clients.
b. Dosage: 1.75 mg injected subcutaneously as needed 45 minutes prior to sexual activity.
c. Contraindicated in clients with uncontrolled hypertension or known cardiovascular disease.

FOLLOW-UP

A. As determined by history and physical examination findings and diagnoses made. Clients placed on medications should follow up 1 month after initiating and have routine follow-up at 3- to 6-month intervals to determine if continuation of the medication is necessary.

B. Postmenopausal clients with vaginal bleeding should follow up immediately.

CONSULTATION/REFERRAL

A. Refer to any therapist who specializes in sexual problems: Counseling may include sex therapy or cognitive behavioral therapy (CBT).

B. May refer to pelvic floor therapist for treatment of genitopelvic pain disorders, once other medical conditions have been treated.

INDIVIDUAL CONSIDERATIONS

A. Use of opposing progestin in clients with an intact uterus is not necessary for all forms of vaginal estrogens. Vagifem, for example, does not have to be opposed with a progestin. Clinicians prescribing hormonal therapy should be well informed and educated on which particular formulations require opposition therapy. Additionally, estrogen should always be initiated at the lowest possible effective dose and given for the shortest duration of time. Hormone therapy (HT) should be very individualized to the client, taking into consideration their symptoms, quality of life, and personal preference. The decision to initiate, continue, and discontinue HT is one made in partnership with the individual client.

B. Clients with a history of breast cancer: Although risk is low, consult with the client's oncologist before initiating vaginal estrogen. Clients with a history of breast cancer may be appropriate candidates for vaginal ET.

C. If using ospemifene (Osphena), use opposing progestin in clients with intact uterus. Contraindicated in clients with undiagnosed abnormal vaginal bleeding, history of venous thromboembolism (VTE), pulmonary embolism (PE), breast cancer, and females of childbearing age.

RESOURCES

American Association of Sexuality Educators, Counselors, and Therapists
35 E Wacker Drive
Suite 850
(202) 449-1099
Email: info@aasect.org
www.aasect.org

International Society for the Study of Women's Sexual Health
14305 Southcross Dr
Suite 100
Burnsville, MN 55306
(952) 683 9025
Email: info@isswsh.org
www.isswsh.org

North American Menopause Society
30050 Chagrin Blvd, Suite 120 W- Pepper Pike

OH,44124
(440) 442-7550
Email: info@menopause.org
Society for Sex Therapy and Research
8120 Lehigh Ave, Suite 100
Morton Grove, IL 60053
(847) 647-8832
Email: info@sstarnet.org

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GENITO-PELVIC PAIN/PENETRATION DISORDER (DYSPAREUNIA)

DEFINITION

A. Genito-pelvic pain/penetration disorder (GPPPD) previously termed dyspareunia and/or vaginismus. GPPPD is defined as one of more of the following persistent or recurrent symptoms for 6 months: marked vulvovaginal or pelvic pain during penetrative intercourse or penetrative attempts, marked fear or anxiety about vulvovaginal or pelvic pain related to penetration, and or marked tensing of the pelvic floor muscles (PFMs) during attempted vaginal penetration genital or pelvic discomfort associated with sexual intercourse (upon entry or deep penetration) that interferes with sexual satisfaction. Pain may be superficial (also known as introital), relating to vulvar and vaginal pain, or it may be deep, relating to deep, pelvic pain. Vaginismus is the involuntary (often painful) contraction of the PFMs in response to pressure or attempted penetration.

INCIDENCE

A. The estimated prevalence of GPPPD in the United States ranges from 3% to 25%.

PATHOGENESIS

A. Both physical and psychosocial etiologies have been identified.

1. Physical causes:
- a. Vulvovaginal anomalies:
  - i. Thick hymen.
  - ii. Short vagina.
  - iii. Vaginal agenesis.
  - iv. Vaginal septum.
- b. Organic dyspareunia:
  - i. Episiotomy scars.
  - ii. Bartholin gland cyst.
  - iii. Vulvar dystrophy.
  - iv. Inflammation or infection, STI.
  - v. Vulvovaginal cancer.
  - vi. Pelvic disease:
    - 1) Pelvic inflammatory disease(PID).
    - 2) Uterine or ovarian tumors.
    - 3) Adenomyosis.
    - 4) Pelvic scarring or adhesions versus endometriosis.
  - vii. Genital mutilation.
- c. Musculoskeletal anomalies:
  - i. Disc disease.
  - ii. Myofascial pain.
  - iii. Coccygodynia.
- d. Extensive prolapse or organ displacement.
- e. Urethral syndrome or other urinary tract disorders.
- f. Vulvodynia.
- g. Hormonal factors:
  - i. Hypoestrogenemia causing atrophic vaginitis.
  - ii. Breastfeeding causing atrophic vaginitis.
  - iii. Menopause causing atrophic vaginitis.

2. Psychosocial causes:
- a. Childhood molestation.
- b. Fear of pain, infection, or pregnancy.
- c. Pelvic congestion syndrome.
- d. Poor partner communication.
- e. History of sexual assault, including date rape.
- f. Previous trauma during intercourse.
- g. Domestic violence.
- h. Strict religious orthodoxy.
- i. Poor sexual education; negative attitudes toward sex.

COMMON COMPLAINTS

A. Irritation and/or burning with intercourse.
B. Lack of vaginal lubrication.
C. Pain and or burning upon vulvar or vaginal contact.
D. Pain with deep penetration.
E. Postcoital bleeding.

SUBJECTIVE DATA

A. Review the onset, duration, and course of presenting symptoms, including the precise location and timing of pain during intercourse.

B. Review the client's medical or surgical history for physical causes (see the "Pathogenesis" section).

C. Ask: How often does pain occur (with every act of intercourse, near menses, or in certain sexual positions)? What relief measures have been tried? Is there improvement with using extra lubrication? How much relief was obtained with each measure?

D. Obtain a complete sexual history including the following:

1. Sexual practices.
2. Sexual satisfaction; ability to achieve orgasm.
3. Perception of partner satisfaction.
4. Age at first coitus.
5. History of sexual abuse, molestation, assault.
6. Perceptions regarding sexuality.
7. Current number of sexual partners and preferences.
8. Time spent in foreplay.
9. History of recent pregnancy/birth and breastfeeding.
10. Age at onset of puberty, date of last menses, and cycle history.
11. Current method of birth control and satisfaction with method; previous methods and why they were discontinued.
12. Presence of vaginal discharge, odor, dysuria, or other physical symptoms occurring both before and after intercourse.
13. Medications, including prescription and OTC drugs.
14. Can the client insert a tampon without pain?

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure (BP).

B. Inspect:

1. Observe generally for discomfort before, during, and after the examination.

C. Auscultate:

1. The abdomen for bowel sounds in all quadrants; auscultation of the abdomen should precede any palpation or percussion due to the changes in intensity and frequency of sounds after manipulation.

D. Palpate:

1. Palpate the abdomen for masses; check for suprapubic tenderness.

2. Examine the back; assess range of motion. Observe for evidence of disc disease, myofascial pain, and coccygodynia. Palpate for CVAT.

E. Pelvic examination:

1. Inspect: Perform a perineal examination for atrophic vaginitis. Atrophic vaginitis presents as red, shiny, smooth vaginal tissue (loss of rugae); vaginal thinning; and decreased elasticity of vaginal tissues. Vulvar inflammation may be present.

2. Evaluate the client for vulvovaginitis. Perform a vulvar examination for Bartholin gland enlargement, fissures, condyloma, and herpes simplex infection. Inspect for anatomic variants, including narrowed introitus, congenital vaginal malformation (septum), and pelvic relaxation (cystocele and rectocele).

3. Q-tip test for vulvar vestibulitis. (Using a moistened cotton Q-tip, apply gentle pressure on the inner thigh, labia majora, labia minora, clitoris, and perineum.)

4. Speculum examination: Inspect for cervicitis, friability, and discharge. If the client can insert a tampon without pain, a mechanical obstruction is unlikely.

5. Bimanual examination: Check cervical motion tenderness (CMT); adnexal masses; and uterine size, consistency, and position.

6. Rectovaginal examination: Palpate uterosacral ligaments for pain and nodularity and other signs of PID and endometriosis. In cases of rectal trauma, cultures may be needed to rule out STIs.

DIAGNOSTIC TESTS

A. Laboratory testing is typically not necessary in the initial evaluation unless an undiagnosed medical etiology is suspected.

B. Wet prep to rule out candidiasis, trichomoniasis, and BV.

C. Cervical cultures for chlamydia, gonorrhea.

D. Viral cultures of lesions, if any.

E. Urine culture, if applicable.

F. Pelvic ultrasonography, if indicated to rule out pelvic mass or evaluate endometriosis.

DIFFERENTIAL DIAGNOSES

A. Genito-pelvic pain penetration disorder.

B. See the "Pathogenesis" section.

PLAN

A. General interventions:

1. Perform a detailed physical examination after taking a thorough history.

2. Clients should be encouraged to involve their partner(s) in assessment, diagnosis, and treatment of dyspareunia.

3. A secure, trusting relationship must be established with the care provider before many clients feel comfortable discussing sexuality issues. Continuity with one provider is essential.

4. Clients with dyspareunia should be evaluated for multiple etiologies. Treat underlying pathologies such as musculoskeletal anomalies, pelvic infection, urinary tract infection (UTI), STIs, hormonal deficiencies, and GI etiologies (see specific chapters for treatment plans and drug therapy).

B. Client teaching: *See Client Teaching Guide for this chapter, "Dyspareunia (Pain With Intercourse)."*

C. Pharmaceutical therapy:

1. Refer to specific chapters for therapies related to etiology.

2. Low-dose vaginal estrogen therapy is used in the treatment of genitourinary syndrome of menopause.

3. Lubricants and moisturizers may reduce pain during intercourse (level B evidence).

4. Vulvodynia: Consider the use of topical agents applied to the vulva or vestibule.

a. Lidocaine (Xylocaine) 2% or 5% gel applied to the vulva, vestibule, and fourchette, both nightly and prior to intercourse (level C evidence).

D. Psychological and psychosexual therapy:

1. Cognitive behavioral therapy (CBT) as indicated.

2. Pelvic floor physical therapy as indicated.

FOLLOW-UP

A. Refer to follow-up plans for specific etiology.

CONSULTATION/REFERRAL

A. Refer the client to an OB/GYN physician for removal of cysts and endometriomas. Laparoscopy is indicated if endometriosis, adhesions, or an adnexal mass is suspected.

B. Refer the client to an OB/GYN physician for vulvovaginal anomalies, including thickened hymen, shortened vagina, or vaginal agenesis.

C. Refer the client to a clinician specializing in sexual therapy for continued issues of sexual dysfunction without an identifiable physical cause.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy or postpartum:

1. Sexual intercourse may continue throughout pregnancy unless there is pain, bleeding, preterm labor, or premature rupture of the membranes. Alternative sexual positions may need to be used to facilitate client comfort, particularly in the third trimester. Sexual intercourse may resume in the postpartum period when the bleeding has decreased or stopped, and any laceration or episiotomy is healed.

2. Breastfeeding causes hormonal changes that may result in atrophic vaginitis, as well as a vaginal environment that is similar to that of menopause. Vaginal lubrication production may also be diminished in the breastfeeding client; counseling the client that extra lubrication may be necessary for enhanced comfort with intercourse is helpful.

B. Partners:

1. Encourage the client to have partner(s) participate in sexual health counseling.

C. Postmenopausal:

1. This population may experience dyspareunia due to vulvar and vaginal atrophy from withdrawl of estrogen.in the tissues. Similar counseling as the postpartum clients is recommended for treatment. Partners need to understand the thinness due to atrophy of the vagina tissue.

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GENITOURINARY SYNDROME OF MENOPAUSE

DEFINITION

A. In 2014, the International Society for the Study of Women's Sexual Health (ISSWSH) and the North American Menopause Society (NAMS) reached a terminology consensus on the change in language to genitourinary syndrome of menopause (GSM) for vulvovaginal atrophy (VVA). GSM is medically more accurate, and all-encompassing. GSM is defined as a collection of symptoms and signs associated with a decrease in estrogen and other sex steroids involving changes to the labia majora/minora, clitoris, vestibule/introitus, vagina, urethra, and bladder.

INCIDENCE

A. GSM is very common and is thought to affect about 50% of postmenopausal individuals, ranging from mild to debilitating. Symptoms of GSM are also present in preadolescence, postpartum, and during breastfeeding.

B. Unlike the low-estrogen state in postmenopausal period, vasomotor symptoms often improve over time. GSM, however, is chronic and progressive and encompasses premenopausal to the elderly female population. Clients do not need to display all of the symptoms for the diagnosis of GSM. Symptoms include the following:

1. Dryness, burning sensations, and irritation in the genital area.

2. Dyspareunia.

3. Urinary frequency, pain with urination, urgency, nocturia, urinary incontinence (UI), or recurrent urinary tract infections (UTIs).

PATHOGENESIS

A. Decrease in estrogen and other sex hormones.

1. Lack of sufficient estrogen promotes an increase in vaginal pH that supports the development of bacterial infections. Estrogen loss also results in a decrease in vaginal glycogen and a thin-walled epithelium, promoting friability and inflammation.

2. The female genital tract and the lower urinary tract share a common embryonic origin. As such, estrogen plays an important role in the function of the lower urinary tract.

B. Anatomic changes occur, including reduced collagen, decreased elastin, and fewer blood vessels.

C. Physiologic changes result in reduced vaginal blood flow, resulting in decreased lubrication, flexibility, and elasticity of the vaginal vault and increased vaginal pH.

PREDISPOSING FACTORS

A. Preadolescence.
B. Postpartum.
C. Breastfeeding.
D. Perimenopause.
E. Postmenopause.
F. Ovarian failure.
G. Medications:

1. Aromatase inhibitors.

2. Antidepressants.

3. Anticholinergics.

COMMON COMPLAINTS

A. Vaginal dryness, irritation, and/or bleeding.
B. Dyspareunia.
C. Dysuria.

OTHER SIGNS AND SYMPTOMS

A. Postcoital bleeding.
B. Thin vaginal discharge.
C. Vaginal itching.

SUBJECTIVE DATA

Because clients may be reluctant to volunteer information about GSM symptoms, ask specific questions about the presence, severity of symptoms, and impact on their quality of life and sexual function.

A. Question the client regarding onset, duration, and course of symptoms.

B. Is this a new problem? If so, review the use of a new soap, laundry detergent, or hygiene products.

C. Describe the color, amount, and odor of vaginal discharge or bleeding.

D. Determine existence of coexisting vasomotor symptoms, such as hot flashes.

E. Are they experiencing any or all of the symptoms of GSM, dysuria, urinary frequency, vulvar dryness and itching, or dyspareunia? With dyspareunia, ask the client if discomfort occurs with foreplay or pain occurs with deep penetration, or both.

F. Determine if the client is breastfeeding and for what length of time.

G. Ask the client the date of their last menses and if they are having irregular cycles. Determine if the client had a hysterectomy with oophorectomy or ovarian failure.

H. Review the number of the client's sexual partners and any new sexual practices.

I. Review the client's current medications, including antidepressants and medications with anticholinergic side effects.

J. Explore whether they are currently taking or whether they have stopped hormone therapy (HT).

K. Has the client tried any self-help measures? Was there any relief?

L. When was their last Pap smear and what were the results?

M. Review all medications, including over-the-counter (OTC) and herbal products.

N. Review their medical history, specifically for diabetes, urinary system dysfunction, autoimmune disorders, and breast cancer.

PHYSICAL EXAMINATION

A. Check temperature (if infection is suspected), blood pressure (BP), pulse, respirations, height, and weight to calculate body mass index (BMI).

B. Inspect:

1. Observe the client generally for discomfort before, during, and after pelvic examination.

C. Palpate:

1. Back: Check for costovertebral angle (CVA) tenderness.

2. Abdomen: Note suprapubic tenderness.

D. Pelvic examination:

1. Inspect:

▶

a. Examine external genitalia for friability, erythema, lesions, condyloma, and amount and color of discharge.
b. Sparse and brittle pubic hair, shrinking of the labia minora, and inflammation of the vulva may be noted in menopausal clients.
c. The vulva may appear erythematous, and there may be labial edema.
d. Excoriation may be present if the client has complained of pruritus.

2. Palpate:
a. "Milk" urethra for discharge to rule out infection.

3. Speculum examination:
a. Check rugae, friability of vaginal epithelium, and color and amount of discharge; evaluate cervix for lesions, friability, and erythema.
b. Typical atrophic symptoms on inspection: thin, friable vaginal epithelium; decreased or absent vaginal rugae; scant vaginal discharge.
c. A pediatric speculum may be needed for geriatric females due to a narrowed introitus.
d. If the speculum examination cannot be completed, it should be delayed until HT has had adequate time to alter the introitus.

4. Bimanual examination:
a. Check for cervical motion tenderness (CMT), uterine size, and position (if no hysterectomy).
b. Check adnexa for masses.

DIAGNOSTIC TESTS

A. Serum hormone levels should only be measured when clinically indicated. There is no laboratory blood test that will confirm or negate the diagnosis of GSM. There is no cut-off serum concentration for any sex steroid below which most clients will experience GSM symptoms.

B. Vaginal pH: Measure the pH of the vaginal vault secretions:
1. pH of a healthy vagina ranges from 3.5 to 5.0.
2. Normal pH in premenopausal clients is 4 to 4.5.
3. pH may reach levels of 5.5 or higher in clients with GSM and can be considered an indicator of vaginal atrophy due to hormonal deficiency.

C. Wet prep:
1. Multiple white blood cells (WBCs) indicate inflammation, may show increased bacteria, and may have decreased lactobacillus, suggesting atrophic vaginitis.
2. Test should be negative for *Trichomonas*. Bacterial vaginosis (BV): Whiff test should be negative.

D. Pap smear with maturation index. (Vaginal wall maturation index evaluation is controversial.)

E. Cultures for gonorrhea (GC) and *Chlamydia trachomatis* (CT), if applicable.

F. Urine culture, if applicable.

G. Ultrasound for uterine lining thickness (<4 or 5 mm suggests loss of estrogenic stimulation).

H. Endometrial biopsy, if indicated.

I. A vulvar biopsy may be indicated.

Postmenopausal vaginal bleeding, however scant or infrequent, must be thoroughly investigated to rule out the possibility of endometrial hyperplasia or endometrial cancer.

DIFFERENTIAL DIAGNOSES

A. GSM:
1. Preadolescence.
2. Breastfeeding.
3. Perimenopausal.
4. Postmenopausal.

B. Ovarian failure.

C. Trauma.

D. Foreign body in the vagina.

E. UTI.

F. Vaginitis from infective cause: fungus, bacteria, or virus.

G. Contact irritation: latex (condom), spermicide, lubricant.

H. Dermatologic conditions of the vulva (e.g., lichen sclerosus or planus, eczema).

I. Vulvodynia/vaginismus.

J. Autoimmune disorders.

K. Chronic pelvic pain.

PLAN

A. General interventions:
1. Treat any underlying infections (GC, CT, vaginitis) as diagnosed.

B. Client teaching: *See Client Teaching Guide for this chapter, "Genitourinary Syndrome (Atrophic Vaginitis)."*
1. Clients should be reassured that this problem is physical, not emotional, and that about half of females experience these same symptoms.
2. Treatment should be individualized therapy dependent on the client's symptoms, sexual function, relationships, and quality of life.
3. Discuss the benefits of regular sexual activity to decrease problems of genitourinary symptoms. An important reason for decreased sexual activity is unavailability of a partner. Masturbation also facilitates the natural resumption of the production of lubricating secretions by the body. Decline in sexuality is influenced by culture and attitudes as well as physical problems.
4. Symptomatic relief of dryness during sexual activity may be obtained with the use of water-soluble lubricants and adequate foreplay. Lubricants are used to reduce friction and dyspareunia related to vaginal dryness. There are many commercially available lubricants. Coconut, olive, or sweet almond oils are natural alternatives.
5. Vaginal moisturizer for relief of symptoms.
6. Discuss pregnancy prevention and inform that perimenopausal symptoms do not ensure lack of fertility.
7. Refer for pelvic floor therapy for training and relaxation to help reduce symptoms.
8. Discuss the use of vaginal dilators for the vaginal introitus.
9. Pessary.
10. Fractional CO_2 laser therapy.

C. Pharmaceutical therapy:
1. Calamine lotion may be applied externally for local symptomatic relief.
2. Estrogen therapy (ET): An individualized approach should be taken with all hormone therapies (HTs). Discuss risks versus benefits with the client and consider client goals and preferences. Benefits of ET appear to outweigh risks in most clients younger than 60 or within 10 years of menopause:
a. Vaginal hormonal therapy (preferred over systemic hormone replacement if symptoms are confined to the vagina):

Absolute contraindications to use of ET also apply to use of topical estrogen (breast cancer, active liver disease, history of recent thromboembolic event). Vaginal estrogen creams are systemically absorbed. As ▶

with use of oral and transdermal estrogen, a progestin must be administered to females who have an intact uterus, secondary to the risk of endometrial hyperplasia or cancer.

i. Conjugated estrogen (Premarin) cream 0.625 mg/g; use 0.5- to 1.0-g applicator inserted intravaginally at bedtime every night for 1 to 2 weeks, then every other night for 1 to 2 weeks, then as needed. **Not for daily use if the client has an intact uterus.**

ii. Estradiol (Estrace) 0.1 mg/g, one-half (2 g) to one (4 g) applicator inserted intravaginally at bedtime every night for 1 to 2 weeks. When vaginal mucosa is restored, maintenance dose is one-quarter applicator (1 g) one to three times weekly in a cyclic regimen. **Not for daily use if the client has an intact uterus.**

iii. Estradiol acetate transvaginal ring (Femring) 0.5 mg a day to 0.1 mg a day. Insert one ring per vagina and replace every 90 days.

iv. Estradiol (Estring) 7.5 mcg/24 hours, insert one ring per vagina and replace every 90 days.

v. Estradiol hemihydrate (Vagifem) vaginal tablets 10 mg one tablet each day for 14 days, then one tablet twice weekly for 10 weeks.

b. Oral estrogen replacement therapy:

i. Conjugated estrogen (Premarin) 0.625 mg orally every day from days 1 through 28 of month. Plus medroxyprogesterone acetate (Provera) 5 mg orally on days 15 through 28.

ii. See section "Menopause" for other regimens of HT.

For long-term ET, consider use of oral or patch methods of delivery if the client shows additional symptoms of hypoestrogenemia (e.g., hot flashes, night sweats).

c. Ospemifene (Osphena) is an estrogen agonist/antagonist that was approved by the Food and Drug Administration (FDA) in 2013 for treatment of moderate to severe dyspareunia secondary to vulvar and vaginal atrophy due to menopause:

i. Dosage 60 mg/d with food.

ii. Use for the shortest duration consistent with treatment goals and risk for the individual client.

iii. Contraindications to use of ospemifene:

1) Undiagnosed abnormal genital bleeding.

2) Known or suspected estrogen-dependent neoplasia.

3) History of or active deep vein thrombosis (DVT) or pulmonary embolism (PE).

4) History of or active arterial thromboembolic disease (i.e., stroke, myocardial infarction [MI]).

5) **Ospemifene is a category X associated with embryo-fetal harm and/or death. It should not be prescribed for clients who are or may become pregnant.**

d. Intravaginal DHEA (prasterone) has been approved by the FDA for the management of moderate to severe dyspareunia due to menopause.

Ospemifene has estrogenic agonistic effects on the endometrium. As with use of oral, vaginal, and transdermal estrogen, a progestin must be administered to clients who have an intact uterus, secondary to the risk of endometrial hyperplasia or cancer.

FOLLOW-UP

A. Breastfeeding clients should be reevaluated following weaning, especially if symptoms persist (e.g., alternate etiology is suspected).

B. Postmenopausal clients should be evaluated for additional etiologies (e.g., endometrial hyperplasia) if vaginal bleeding persists beyond 3 to 6 months following treatment.

C. The client should return to clinic 1 to 2 months after beginning oral or vaginal drug therapy; the client then needs to be seen in 3 to 6 months to check side effects, BP, and response to therapy.

D. Perform Pap smears and physical examination per client health history or risks and symptoms.

CONSULTATION/REFERRAL

A. If bleeding is a symptom in a postmenopausal client, the practitioner *must* rule out bleeding of uterine origin. If there is *any* doubt, consultation for endometrial biopsy or dilatation and curettage (D&C) must be obtained.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Breastfeeding clients have amelioration of symptoms as weaning progresses unless an alternate etiology exists.

B. Postmenopausal clients:

1. Evaluate the clients for other risks of hypoestrogenemia, such as cardiovascular disease and osteoporosis. Continuous systemic ET may be indicated.

2. Evaluate need for continuous systemic HT.

3. Vaginitis in the postmenopausal client is rarely due to any of the organisms responsible for vaginitis in the premenopausal client (unless they has new sexual partners). Candidiasis, trichomoniasis, and BV are uncommon after the menstruating years.

C. Geriatrics:

1. Physical examination in the elderly:

a. The pelvic examination may be more difficult related to the lack of hip mobility, arthritis, or spinal deformities.

b. The examination may need to be performed on the client turned to their left side.

c. Allow extra time to explain each step of the examination and for positioning. An assistant can help the client onto the exam table and may help gently support the client's legs if necessary.

d. Expected age-related changes include thinning pubic hair and decreased elasticity of the introitus.

e. Bluish swelling may be varicosities.

2. UTIs dramatically increase in elderly females. UI, anatomic changes such as a cystocele, increased residual urine, and diabetes are risk factors for recurrent UTIs in older clients.

3. Clients who use pads or adult diapers should be informed of risks associated with infrequent changing. Due to the high cost of absorbent pads and undergarments, infrequent changing is common and contact dermatitis and candidal rashes may develop. If these occur, ▶

clients should be encouraged to change pads/garments more frequently and avoid wearing absorbent garments overnight, and instead should use mattress/bedding pads.

4. For the older adult with existing GSM-associated urinary symptoms, nurse practitioners should practice special consideration of the following geriatric syndromes:

a. Delirium is a common early sign of acute illness or infection in the elderly. It is more common in clients with existing cognitive impairment. Dehydration is more common in the elderly. Signs of dehydration include confusion, muscle weakness, fever, dizziness, poor skin turgor, hypotension, and tachycardia. Diminished thirst mechanism and decreased body water exacerbate dehydration:

i. At a minimum, clients and their family should be asked if the client has experienced episodes of altered mental status.

ii. The Brief Confusion Assessment Method (bCAM) is a well-documented assessment for delirium. The bCAM is available at eddelirium.org/wp-content/uploads/2016/05/bCAM-Training-Manual-Version-1.0-10-15-2015.pdf.

b. Falls: Consistently assess for falls. Discuss the risk of dizziness or light-headedness when standing. Many falls occur en route to the bathroom when the client hurries and may not be able to remove their pants and undergarments in time:

i. At the minimum, older adults should be asked how many times they have fallen since their last visit. A reported fall should trigger questions about whether the fall led to injury, a visit to urgent care or hospital ED, or a hospital admission.

ii. Safety measures to reduce falls in the home include removing small area carpets, keeping night lights along the path to the bathroom, and installing a toilet seat or nearby grab bars. *See Client Teaching Guide for Chapter 3, "Safety Issues: Fall Prevention."*

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IMPAIRED FERTILITY

DEFINITION

A. Infertility is defined as the inability of a couple to conceive within 12 months of unprotected intercourse. Many clinicians use a 6-month time frame if the individual is 35 years of age and older due to the age-related decline in fertility. Expedited evaluation may also be considered in situations including oligomenorrhea or amenorrhea, known tubal or uterine disease, endometriosis, or suspected male subfertility.

B. A female who has never been pregnant or a male who has never initiated a pregnancy is said to have "primary infertility."

C. If a previous pregnancy has been achieved and the couple is unable to conceive a subsequent pregnancy, the term "secondary infertility" is applied.

INCIDENCE

A. Infertility affects up to 15% of couples. The most common cause of female infertility is ovulatory dysfunction. Other common causes include blocked fallopian tubes caused by pelvic inflammatory disease (PID) or endometriosis. The most common cause of male factor infertility is oligospermia (few sperm seen in the ejaculate) or azoospermia (no sperm seen in the ejaculate).

PATHOGENESIS

A. Infertility may occur in the male or female. Every effort should be made to evaluate both partners at the same time. For female–male couples, male factor infertility has a prevalence of 40% to 50%. Given this, a basic history and medical evaluation of the male partner are warranted. Unexplained infertility occurs in up to 30% of infertile couples.

1. Infrequent intercourse.

2. Medical (Table 17.3).

PREDISPOSING FACTORS

A. Predisposing factors depend on the etiology.

COMMON COMPLAINTS

A. The common complaint is an inability to achieve pregnancy despite frequent acts of intercourse.

OTHER SIGNS AND SYMPTOMS

A. Dependent on the pathogenesis and history.

SUBJECTIVE DATA

A. Obtain a complete health history, including the following:

1. Age of both partners.

2. General health of both partners: past surgeries, previous hospitalizations, serious illnesses or injuries, PID, or exposure to sexually transmitted infections (STIs).

3. Complete pregnancy history of the female:

a. Complications during pregnancy, including placenta previa accreta, gestational diabetes, preterm labor, or cerclage for incompetent cervix.

b. Number of pregnancies, Cesarean/vaginal deliveries, miscarriages, dilatation and curettages (D&Cs), abortions.

4. Family history of birth defects, developmental delay, early menopause, or reproductive problems.

5. Length of infertility, including prior workup, if any.

6. Coital history:

a. Frequency and timing.

b. Able to ejaculate in the vagina.

c. Use of lubricants (some may be spermicidal).

d. Postcoital habits: douching or voiding.

B. Obtain a complete menstrual history, including the following:

1. Age at puberty.

2. Cycle length and characteristics.

3. Dysmenorrhea.

4. Molimina.

5. Date of last missed period (LMP).

C. Obtain a complete gynecologic history, including the following:

1. Previous contraceptive use.

2. Medical and surgical interventions:

a. D&C.

b. Laparoscopy or endometriosis.

3. Anomalies.

TABLE 17.3 PATHOGENESIS OF INFERTILITY

Male Pathogenesis	Female Pathogenesis
Inadequate sperm production • Azoospermia from: ■ Cancer therapy ■ Adult mumps ■ Sertoli-cell-only syndrome ■ Hypogonadism ■ Retrograde ejaculation • Oligospermia from: ■ Varicocele ■ Small testicular size Reproductive tract anomaly • Blocked vas deferens • Varicocele • Congenital obstruction of epididymis • Absence of the vas deferens (cystic fibrosis) Klinefelter syndrome Physical and chemical agent exposure • Coal tar • Radiation Health conditions/disorders • Diabetes • Low serum testosterone • Pituitary tumors • Hyperprolactinemia • Hypertension Testicular infection (epididymitis) Injury to reproductive organs/tract Nerve damage/neurologic disease: spinal cord injury Impotence/erectile difficulty: performance anxiety Premature ejaculation Early withdrawal Lifestyle factors • Drugs • Smoking • Alcohol • Malnutrition Antispermatozoa antibodies Medications • Antihypertensives • Antidepressants • Antipsychotics • Antiulcer agents/antacids • Muscle relaxants • Testosterone • Anabolic steroid use	Advanced maternal age Disorder of ovulation/hypothalamic dysfunction • Anovulation • Amenorrhea • Polycystic ovary triad ■ Acne ■ Obesity ■ Hirsutism • Premature ovarian failure ■ Autoimmune ■ Idiopathic ■ Cancer therapy • Luteal phase insufficiency • Hyperprolactinemia Ovarian factors • Cysts or tumor • Irradiation Tubal disorders/damage/blockage • PID • Chlamydia trachomatis • Postpartum infection • Pelvic trauma (motor vehicle accident) • Inflammatory bowel disease • Endometriosis • Pelvic adhesions • Idiopathic Uterine pathology • Congenital anomalies: duplication • Septate • Fibroids • Polyps • Asherman syndrome • Synechiae Cervical factors • Anatomic abnormalities (hood) • Previous cervical surgery (e.g., conization, which leads to mucus depletion) • Hostile cervical mucus • Presence of sperm antibodies in the cervix • Infections Lifestyle factors • Drugs • Smoking • Alcohol consumption Vaginal factors • Intact hymen • Septum • Absent vagina • Infection ■ Trichomonas ■ Candida ■ Chlamydia ■ Mycoplasma ■ Bacterial vaginosis

(*continued*)

TABLE 17.3 PATHOGENESIS OF INFERTILITY (*CONTINUED*)

Male Pathogenesis	Female Pathogenesis
	▪ Gonorrhea ▪ Streptococci Medications: oral contraceptives Medical problems • Lupus • Hypothyroidism • Diabetes • Antiphospholipid syndrome • Blood clotting disorders (factor V, PAI-1)

PAI-1, plasminogen activator inhibitor-1; PID, pelvic inflammatory disease.

D. Take a complete nutritional and exercise history; note eating disorders:
1. Anorexia nervosa.
2. Bulimia.
E. Review female and male reproductive tract infections and treatments for past and present partners.
F. Review social habits for the male and female:
1. Smoking: How much, how often, how long.
2. Drugs: How much, how often, how long for each drug.
3. Alcohol: How much, how often, how long.
4. Use of saunas or hot tubs.
5. Exercise, including cycling.
G. Take a complete medication history, specifically review for:
1. Antihypertensives.
2. Antidepressants.
3. Antipsychotics.
4. Muscle relaxants.
5. Testosterone or anabolic steroid use for the male partner.
H. Review for exposure to toxic chemicals, radiation, or known teratogens:
1. Military/war exposure.
2. Employment exposure.
3. Pesticides, nail salons.
I. Inquire about diethylstilbestrol (DES) exposure in utero (for either partner).
J. Review for symptoms of thyroid dysfunction:
1. Weight gain or loss.
2. Change of bowel habits.
3. Intolerance to heat or cold.
4. Appetite changes.
K. Review for systemic diseases:
1. Cardiac.
2. Thyroid disease.
3. Diabetes.
4. Androgen excess in the female.
L. Assess the psychosocial context of the infertility, including personal, emotional, and economic factors; family pressures for children; expectations; timing of pregnancy; consideration of adoption; and stress from failure to conceive.

OTHER SIGNS AND SYMPTOMS

A. See "Pathogenesis" section and information in "Subjective Data" section regarding medical history.
B. History of not being able to get pregnant over the past 6 to 12 months.

PHYSICAL EXAMINATION: MALE

A. Obtain height, weight, and body mass index (BMI). Check temperature, pulse, respirations, and blood pressure (BP).
B. Inspect:
1. Note general signs and appearance of under androgenization: decreased body hair, gynecomastia, and eunuchoid proportions.
2. Examine the penis, making note of the location of the urethral meatus; observe urethra for discharge.
C. Percuss:
1. Check deep tendon reflexes (DTRs) for signs of hypothyroidism.
D. Palpate:
1. Neck: Examine thyroid.
2. Genitals: Palpate and measure the testes, the presence and consistency of both vasa and epididymides, and the presence of a varicocele.
E. Rectal examination:
1. Check prostate and seminal vesicles for tenderness and other signs of infection.

Valsalva maneuver, performed while the client stands, helps to reveal small varicocele. Varicocele feels like "a bag of worms" with bluish discoloration visible through the scrotum. Approximately 23% to 30% of infertile males have varicocele (usually present on the left side). No treatment is necessary if the semen analysis is normal.

PHYSICAL EXAMINATION: FEMALE

A. Check temperature, pulse, respirations, and BP. Obtain height, weight, and BMI.
B. Inspect:
1. Examine breasts for presence of nipple discharge.
2. Note general signs, and of androgen excess.

Polycystic ovarian syndrome (PCOS) triad includes acne, obesity, and hirsutism.

C. Auscultate:
1. Abdomen for bowel sounds in all quadrants. Auscultation of the abdomen should precede any palpation or percussion due to the changes in intensity and frequency of sounds after manipulation.
D. Palpate:
1. Neck: Examine the thyroid.
2. Abdomen: Check for tenderness and masses.
3. Back: Check costovertebral angle (CVA) tenderness.

E. Percuss:
1. Percuss: Check DTRs.
F. Pelvic examination:
1. Perform detailed external peritoneal exam for signs of infection; lesions; or anomalies of clitoris, labia, Skene gland, Bartholin gland, vulva, and perineum.
2. Speculum examination:
a. Observe length of vagina, position and characteristics of cervix, and any anomalies.
b. Observe the characteristics of cervical mucus: thin and watery or thick and cloudy; odor; evidence of infection.
3. Bimanual examination: Check uterine size, consistency, contour, mobility, cervical motion tenderness (CMT), and adnexal masses.

A fixed, immobile uterus determined on bimanual exam could indicate the presence of pelvic adhesions from surgery or past pelvic infection.

4. Rectovaginal examination: Palpate uterosacral ligaments for pain and nodularity; evaluate masses and polyps of rectum.

DIAGNOSTIC TESTS

A. Male factor: semen analysis. A semen analysis is a necessary part of the couple's infertility evaluation. Semen analysis should be done early in the infertility evaluation. Typically, metabolic causes are ruled out first in each partner, which would include lab work for both partners (including the semen analysis). If the first evaluation is abnormal, it should be repeated one time. According to the World Health Organization accepted reference range for semen analysis, normal semen analysis includes the following:
1. Volume: 1.5 mL.
2. pH ≥ 7.2.
3. Sperm concentration (10^6 per mL): 15.
4. Sperm count total sperm number (10^6 per ejaculate): 39.
5. Total motility: greater than 40%.
6. Sperm agglutination: absent.
7. Sperm morphology: 4% normal forms.
B. Female (ovarian) factor results are considered in the context of the client's age:
1. Assess ovarian reserve:
a. Antimullerian hormone (AMH; value less than 1 ng/mL) may indicate diminished ovarian reserve.
b. FSH: Measured between cycle days 2 to 5. A high FSH, greater than 10 mIU/mL, may indicate diminished ovarian reserve.
c. Ultrasound for antral follicle count. Less than 5 to 7 may indicate diminished ovarian reserve.
2. Assess for ovulatory dysfunction:
a. Menstrual history may be adequate to assess for ovulatory dysfunction.
b. Serum progesterone measured midway through luteal phase: Luteal progesterone greater than 3 ng/mL indicates ovulation but does not indicate the quality of the luteal phase.
c. Urinary luteinizing hormone (LH) for surge.
d. Thyroid-stimulating hormone (TSH).
3. Serum prolactin for females with irregular menses or other signs of hyperprolactinemia.

When nipple discharge is present, check serum prolactin and TSH to rule out hyperprolactinemia, a pituitary tumor, and thyroid dysfunction.

C. Pelvic or uterine factor:
1. Hysterosalpingogram (HSG); also done for tubal factor HSG (performed in radiology): evaluates tubal patency and rules out uterine anomalies. The HSG should be scheduled for the interval between cessation of menstrual flow and ovulation to avoid retrograde flow of menstrual tissue into the tubes and the abdominal cavity.
2. Laparoscopy (not routinely done for unexplained fertility) if endometriosis is suspected as the cause. Endometrial lesions can be lasered during the procedure; thus, while it is diagnostic, it is also a treatment. Performed by a gynecologist, it is usually done in the first 2 weeks of the menstrual cycle to ensure that the client is not pregnant. Direct visualization of the pelvic organs provides data about degree of adhesion formation, presence of endometriosis or fibroids, and possibility of surgical repair of damaged tubes.
3. Sonohysterography for assessment of uterine structure or abnormalities.
D. Other tests:
1. Pap and human papillomavirus, as indicated by guidelines.
2. Cultures for gonorrhea and chlamydia as indicated.
3. Pregnancy test if amenorrhea is present.
4. Wet mount as indicated.

DIFFERENTIAL DIAGNOSES

A. Infertility.
B. Sexual dysfunction.
C. Hypothyroidism.
D. Hypothalamic dysfunction: amenorrhea.
E. Hyperprolactinemia.
F. Menopause.
G. Ovarian failure.
H. PCOS.
I. Asherman syndrome.
J. Tubal occlusion.
K. Antisperm antibodies.
L. Endometriosis.
M. Oligo-ovulation.
N. Uterine anomalies: fibroids, synechiae, and septa.
O. Pelvic adhesions.

PLAN

A. General interventions:
1. Semen analysis is one of the first steps in an infertility workup. The male should have abstinence of 2 to 5 days prior to analysis. If the first evaluation is abnormal, it should be repeated in 3 months. If still abnormal, consider referral.
2. The male physical examination may be deferred if the semen analysis is normal.
3. The male should always be evaluated early on in the process due to the ease and low cost of an analysis.
4. Female evaluation:
a. The first step is to document ovulation. This may be done through a thorough menstrual history. In ovulatory clients, menstrual cycles are regular and predictable, occurring in intervals of 21 to 35 days and accompanied by a consistent pattern of flow characteristics and molimina symptoms.
b. Serial BBT- Basal body temperature measurements provide a simple and inexpensive method for evaluating ovulatory function. This is no longer considered the best method in evaluating ovulatory function. Some clients' temperatures dip just before the day of ovulation ▶

and then rise. Ovulation and the development of the corpus luteum manifest as an increase of BBT by 0.6° to 1° above the client's baseline temperature. The period of highest fertility spans the 7 days prior to the midcycle rise. The BBT cannot reliably define the time of ovulation and can be tedious for clients.

c. Urinary LH kits (ovulation predictor kits) can identify the midcycle LH surge that precedes ovulations by 1 to 2 days. Clients should begin testing around 5 days prior to ovulation. In a 28-day cycle, this would correlate to cycle day 10. The test is most reliable if performed midday or in the evening and predicts the days of greatest fertility: the day of the LH surge and the following day.

d. Endometrial biopsy, or sampling of the uterine lining late in the luteal phase: Histology can determine endometrial development, which implies ovulation. However, this test cannot distinguish between fertile and infertile individuals. It is no longer recommended in the evaluation of ovulatory function.

e. Serum progesterone: When drawn at the appropriate time of the cycle, it is a reliable indication of ovulation. This should be obtained 1 week prior to expected menses. A progesterone level greater than 3 ng/mL provides evidence of recent ovulation. Higher thresholds at midluteal phase have been used as a measure of quality of luteal function. These levels are ideally greater than 10 ng/mL.

f. Transvaginal ultrasound: A transvaginal pelvic ultrasound can evaluate the number and size of the developing follicles. This provides evidence of follicular growth, collapse of preovulatory follicle, corpus luteal cysts, and luteinization of the endometrial lining. Because of the cost associated with ultrasounds, this should be reserved for clients who are not able to use simpler testing.

g. Evaluation of uterus and fallopian tubes: HSG, performed in radiology, evaluates tubal patency and rules out uterine anomalies. The HSG should be scheduled for the interval between cessation of menstrual flow and ovulation to avoid retrograde flow of menstrual tissue into the tubes and the abdominal cavity.

h. Laparoscopy/hysteroscopy: Diagnostic if used as the final screening examination for infertility. Performed by a gynecologist, it is usually done in the first 2 weeks of the menstrual cycle to ensure that the client is not pregnant. Direct visualization of the pelvic organs provides data about the degree of adhesion formation, presence of endometriosis or fibroids, and the possibility of surgical repair of damaged tubes. Hysteroscopy may be used to visualize intrauterine pathology including septums, polyps, or fibroids.

B. Client teaching:

1. Infertile couples often require extensive counseling, including grief counseling, for failure to achieve pregnancy.

2. When couples do not meet the definition of infertility, efforts should be made to maximize fertility:

a. The fertile window spans the 6-day interval ending on the day of ovulation. Intercourse is most likely to result in pregnancy when it occurs within the 3-day interval ending on the day of ovulation. Couples having intercourse every other day midcycle will maximize their fecundability.

b. Being overweight or underweight, consuming alcohol and caffeine, smoking, and exposure to toxins are all lifestyle factors that affect fertility.

c. Time to conception increases with age. Efforts should be made to achieve pregnancy before their mid-30s.

C. Pharmaceutical therapy:

1. Treatment depends on causative factor(s). These may include oral agents, injectable gonadotropins, or in vitro fertilization.

2. Prescription medications must be supervised by physician and/or specialist due to possible complications such as ovarian hyperstimulation.

FOLLOW-UP

A. Follow-up depends on causative factor(s).

CONSULTATION/REFERRAL

A. Consultation and referral should be considered when pregnancy is not achieved within a reasonable time frame and for specialized testing, surgery, and assisted reproductive therapy.

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MENOPAUSE

DEFINITION

A. Physiologic or natural menopause is the cessation of menses for 12 consecutive months due to the loss of ovarian follicular activity. Natural or physiologic menopause is a retrospective diagnosis recognized 12 months after the final menses. Natural menopause is generally experienced by females between 45 and 55 years of age, with the average age being 51 years.

B. Natural menopause before the age of 40 is considered premature.

C. Premature ovarian failure (POF) is the full or intermittent loss of ovarian function before the age of 40. POF is thought to be caused by genetics, autoimmune disorders, or surgical or chemical interventions.

D. Induced menopause is the abrupt cessation of menses related to chemical or surgical interventions.

E. Perimenopause is caused by fluctuations in ovarian function in the years preceding menopause. The average onset is usually in an individual's 40s but may occur earlier. Due to fluctuations in ovarian function, pregnancy may still occur and unintended pregnancy should be avoided. Perimenopausal symptoms often past several years, with the average duration being 5 years.

INCIDENCE

A. Approximately 2 million individuals reach menopause each year in the United States.

PATHOGENESIS

A. Physiologic menopause is due to failure of ovarian follicular development and ovarian hormone depletion. The major endocrine changes include the decreasing negative feedback on the hypothalamic–pituitary system with increasing follicle-stimulating hormone (FSH) and luteinizing hormone (LH). When the ovaries cease to produce estrogen, they become unable to respond to FSH, resulting in the cessation of ovulation and menstruation.

COMMON SYMPTOMS

A. Insomnia.
B. Absence of menses.
C. Urogenital atrophy:
  1. Vaginal dryness.
  2. Dyspareunia.
  3. Dysuria/frequency.
D. Vasomotor symptoms such as hot flashes/night sweats.
E. All postmenopausal spotting/bleeding should be evaluated for pathologic causes.

SUBJECTIVE DATA

A. Determine onset, duration, and course of presenting symptoms.
B. Obtain complete medical history, including medications, and assess for risk of osteoporosis, cardiovascular disease, and breast and endometrial cancer.
C. Obtain complete gynecologic history, including menarche, interval and duration of menstrual cycles, history of dysmenorrhea, and pregnancy history. Question the client regarding sexual history and contraceptive use (condoms, pills, diaphragm, intrauterine devices [IUDs]).
D. What is the client's current menstrual pattern? Is pregnancy a possibility?
E. Review associated symptoms (hot flashes, insomnia, genitourinary symptoms), onset, timing, duration, and impact on daily life.
F. Assess for mood swings and dysphoria.

PHYSICAL EXAMINATION

A. Check blood pressure (BP), pulse, and respirations.
B. Observe general overall appearance and obtain height, weight, and body mass index (BMI).
C. Auscultate:
  1. Heart, lungs, and abdomen.
D. Percuss:
  1. Abdomen for organomegaly.
E. Palpate:
  1. Palpate thyroid gland.
  2. Perform clinical breast exam, including supra/infraclavicular and axillary lymph node assessment.
  3. Palpate groin for lymphadenopathy.
  4. Palpate the abdomen for masses.
F. Pelvic examination:
  1. Inspect external genitalia for fissures, lesions (external genital warts, genital ulcers), and atrophy.
  2. Palpate for tenderness, cysts, lesions; "milk" the urethra for discharge.
  3. Speculum examination:
    a. Inspect vagina for presence of discharge (amount, color, odor), rugae, foreign bodies, signs of trauma, inflammation.
    b. Inspect cervix for presence of inflammation, friability, discharge.
    c. Perform vaginal/cervical cultures and Pap test as indicated.
  4. Bimanual examination if indicated (pain, bleeding):
    a. Check cervical motion tenderness (CMT); evaluate the size, contour, mobility, and tenderness of uterus. An enlarged or irregular uterus requires additional evaluation. **Over time it is normal for the postmenopausal uterus to decrease in size.**
    b. Palpate the adnexa for tenderness and masses. **Ovaries should not be palpable in postmenopausal clients and require further evaluation if masses or ovaries are appreciated.**
  5. Rectovaginal examination: A rectal exam to evaluate for masses, hemorrhoids, or polyps as indicated in clients older than 50 years. The U.S. Preventive Services Task Force (USPSTF) recommends offering colorectal screening starting at age 45. A onetime hemoccult has very low sensitivity/specificity to evaluate for blood. Instead of a hemoccult, screening for colorectal cancer should be conducted using a colorectal cancer screening test and intervals as recommended by the USPSTF.

DIAGNOSTIC TESTS

A. No laboratory tests are diagnostic of menopause.
B. Consider thyroid-stimulating hormone (TSH).
C. Consider qualitative beta human chorionic gonadotropin (HCG), if amenorrhea is sudden.
D. Complete blood count (CBC) if excessive vaginal bleeding.
E. Obtain Pap smear as indicated per guidelines.
F. Endometrial biopsy as indicated for postmenopausal spotting or bleeding.
G. Transvaginal ultrasonography for enlarged or irregular uterus or inadequate exam due to body habitus.
H. FSH greater than 40 IU/L is consistent with menopause; however, fluctuations in FSH and 17–2 estradiol (E_2) may make use of these markers unreliable, such that they are no longer recommended for determining menopausal status.
I. Shared decision-making regarding colorectal screening test.
J. Additional screening as indicated, such as mammogram, cholesterol, and bone mineral density (BMD).

DIFFERENTIAL DIAGNOSES

A. Menopause.
B. Perimenopause.
C. Anemia.
D. Leukemia or other cancer.
E. Menstrual irregularity for any cause of secondary amenorrhea.
F. Pregnancy.
G. Psychosomatic illness.
H. Thyroid disorders.

PLAN

A. Client teaching:
  1. Provide reassurance as to the cause of the absence of menses.
  2. Discuss common symptoms of menopause.
  3. Provide education regarding healthy lifestyle changes: regular exercise, weight control, smoking cessation, limiting use of drugs and alcohol, and stress reduction.
  4. Encourage a healthy diet rich in vitamin D and calcium:
    a. Supplement diet with calcium to achieve: 1,200 mg/d for individuals 51 years and older. Calcium citrate is superior to calcium carbonate, as it is more absorbable and usable by the body.
    b. Vitamin D supplements to achieve:
      i. 600 IU/d until the age of 70.
      ii. 800 IU/d for 71 years of age and older.
      iii. Consider vitamin D serum screening and, if deficient, treat accordingly.
  5. Encourage vaginal lubricants as needed for vaginal dryness, including water-soluble options and coconut oil. See section "Genitourinary Syndrome of Menopause."
  6. Avoid warm environments, caffeine, alcohol, spicy food, and emotional upset; these may trigger hot flashes. ▶

TABLE 17.4 HORMONE THERAPY

Name	Active Ingredient	Dosage
Estrogen Sequential or Continuous Combined		
Premarin	Conjugated estrogen	0.625 mg/d
Estratab	Esterified estrogen	0.625 mg/d
Menest	Esterified estrogen	0.625 mg/d
Estrace	Micronized estradiol	1.0 mg/d
Ortho-Est	Estropipate	0.625 mg/d
Progestin Only for Sequential Regimen		
Amen	Medroxyprogesterone	5–10 mg added to estrogen the first 10–14 days of month
Cycrin	Medroxyprogesterone	5–10 mg added to estrogen the first 10–14 days of month
Provera	Medroxyprogesterone	5–10 mg added to estrogen the first 10–14 days of month
Prometrium	Micronized progesterone	100–200 mg added to estrogen the first 10–14 days of month
Progestin Only for Continuous Combined Regimen		
Cycrin	Medroxyprogesterone	2.5 mg/d
Provera	Medroxyprogesterone	2.5 mg/d
Combination Packet for Continuous Combined Regimen		
Prempro	0.625 mg conjugated equine estrogen and 2.5 mg medroxyprogesterone	One tablet orally each day
Activella	1 mg 17-beta estradiol and 0.5 mg norethindrone	One tablet orally each day
FemHrt	5 mcg ethinylestradiol and 1 mg norethindrone	One tablet orally each day

Note: See complete prescribing reference or package insert for dosing, titration, contraindications, and side effects.

TABLE 17.5 TRANSDERMAL REPLACEMENT THERAPY

Delivery	Name	Active Ingredient	Dosage
Transdermal patch	Climara	Estradiol	0.25 mg/d, 0.0375 mg/d, 0.05 mg/d, 0.06 mg/d, 0.075 mg/d; apply one patch weekly (lower abdomen or upper buttocks)
Transdermal patch	CombiPatch	Estradiol 0.05 mg and norethindrone acetate 0.14 mg/d or estradiol 0.05 mg and norethindrone acetate 0.0.25 mg/d	Continuous combined regimen one patch twice weekly for 28-day cycle (lower abdomen)
Transdermal patch	Vivelle		One patch twice weekly to trunk
Gel pump	Elestrin	Estradiol	0.06% gel, one pump daily to clean dry skin of upper arm
Gel pump	EstroGel	Estradiol	0.75 mg/1.25 g gel, one pump daily to clean dry skin of upper arm
Spray	Evamist	Estradiol	1.53 mg/spray, one spray daily to inside of arm
	Vivelle	Estradiol	0.05/d, 0.1 mg/d

Note: See complete prescribing reference or package insert for dosing, titration, contraindications, and side effects.

7. Encourage sleep hygiene and adequate rest.
8. Discuss the risks and benefits of exogenous hormone therapy (HT).
9. Assess and manage clients at increased risk for osteoporosis according to current osteoporosis guidelines.
10. Assess and treat cardiac risk factors, including hypertension and dyslipidemia as indicated.
11. Give the client the relevant teaching guide. *See Client Teaching Guide for this chapter, "Menopause."*

B. Pharmaceutical therapy:
1. HT:
a. Estrogen therapy (ET) and estrogen plus progestogen therapy (i.e., HT) may be a strategy for management of menopausal symptoms. All individuals should be counseled regarding the indications, risk, and benefits of HT. HT is indicated for treatment of vasomotor symptoms: hot flashes and night sweats, and vulvovaginal atrophy and painful intercourse. Use of exogenous ET prevents bone loss when used for symptom management but is not indicated for treatment of osteoporosis. It may be used as a strategy for clients with osteopenia if other pharmacologic methods are insufficient or inadequate (see "Osteoporosis/Kyphosis/Fracture" section of Chapter 22, "Rheumatologic Guidelines").
b. Risks of HT include an increased risk of venous thromboembolism (VTE), and breast cancer. Long-term unopposed ET increases the risk of endometrial cancer, which is why all clients using ET must be prescribed a progestogen concomitantly. Potential areas of concern with the use of HT include gallbladder disease and cardiovascular events. ET should not be prescribed for clients with a history of significant cardiovascular disease warranting procedures such as coronary artery bypass graft (CABG) surgery or percutaneous transluminal coronary angioplasty (PTCA). The provider should carefully screen and educate the client prior to initiating HT (Table 17.4).
c. Postmenopausal clients without an intact uterus generally are not prescribed progesterone and are treated with estrogen alone.
d. Oral HT may be given either sequentially or continuously. The sequential regimen is given daily, with progesterone given 12 to 14 days of the month. It is common to have withdrawal bleeding with this regimen. An alternative to this is the continuous regimen, in which both estrogen and progesterone are taken daily.
2. Transdermal estrogen: patches, creams, sprays, lotions. Generally considered safer than oral medications with respect to VTE risk, as the first-pass metabolism effect is avoided and lower doses can achieve a therapeutic effect (Table 17.5).
3. Transvaginal estrogen (Table 17.6).
4. Absolute contraindications to use of ET also apply to use of oral and topical estrogen (breast cancer, active liver disease, and/or history of recent thromboembolic event). Vaginal estrogen creams are minimally systemically absorbed. Screening for endometrial hyperplasia and/or use of a progestogen episodically should be considered on an individual basis.
5. Absolute contraindications to ET:
a. Acute liver disease.
b. Cerebral vascular or coronary artery disease, myocardial infarction (MI), or stroke.
c. History of or active thrombophlebitis or thromboembolic disorders.
d. History of uterine or ovarian cancer.
e. Known or suspected cancer of the breast.
f. Known or suspected estrogen-dependent neoplasm.
g. Pregnancy.
h. Undiagnosed, abnormal vaginal bleeding.
6. Relative contraindications to ET:
a. Active gallbladder disease.
b. Familial hyperlipidemia.
7. Absolute contraindications to progesterone therapy:
a. Active thrombophlebitis or thromboembolic disorders.
b. Acute liver disease.
c. Known or suspected cancer of the breast.
d. Pregnancy.
e. Undiagnosed, abnormal vaginal bleeding. **Educate the client to notify care provider if unusual vaginal bleeding, calf pain, chest pain, shortness of breath,**

TABLE 17.6 TRANSVAGINAL REPLACEMENT THERAPY

Delivery	Name	Active Ingredient	Dosage
Cream	Estrace	Micronized 17-beta estradiol	0.1 mg/g, one-half (2 g) to one (4 g) applicator intravaginally at bedtime every night for 1–2 weeks. When vaginal mucosa is restored, maintenance dose is one-quarter applicator (1 g) one to three times weekly in a cyclic regimen.
	Premarin	Conjugated equine estrogen	0.625 mg/g, use 0.5- to 1.0-g applicator inserted intravaginally at bedtime every night for 1–2 weeks, then every other night for 1–2 weeks, then as needed
Ring	Estring	Micronized 17-beta estradiol	7.5 mg/24 hr insert new ring every 90 days
	Femring	Estradiol acetate	0.05–0.1 mg/d insert new ring every 90 days
Vaginal tablet	Vagifem	Estradiol acetate	25 mcg once daily for 2 weeks then twice weekly
IUD	Mirena	Levonorgestrel	20 mcg daily

Note: See complete prescribing reference or package insert for dosing, titration, contraindications, and side effects.
IUD, intrauterine device.

hemoptysis, severe headaches, visual disturbances, breast pain, abdominal pain, or jaundice occur while being prescribed HT.

C. Nonhormonal pharmacologic therapy for vasomotor symptoms:

1. Antidepressants:
   a. Fluoxetine (Prozac) 20 mg/d.
   b. Venlafaxine (Effexor) 37.5 to 75 mg/d.
   c. Paroxetine (Paxil) 12.5 to 25 mg/d.
   d. Paroxetine (Brisdelle) 7.5 mg/d at bedtime.

2. Anticonvulsant: gabapentin (Neurontin) 300 mg/d and titrate to three or four a day.

3. Antihypertensive: clonidine 0.05 to 0.1 mg/BID. For nonhormonal pharmacologic therapies, review prescribing literature for side effects, titrations, and discontinuation regimens.

D. Nonprescription remedies/herbals:

1. Many nonprescription remedies have been used for treatment of menopausal symptoms. These remedies include isoflavones (soy and red clover), black cohosh, dong quai, evening primrose oil (EPO), ginseng, licorice, and vitamins E and C. Data are lacking regarding the safety and efficacy of any of these alternatives.

2. The provider should review with the client the lack of standardization and evidence regarding the safety and efficacy of these products. Currently, results of research have been insufficient to support or refute the use of these remedies for treatment of menopausal symptoms.

FOLLOW-UP

A. Follow up in 3 to 6 months to assess response to treatment, and then yearly for annual exam (as indicated) and mammogram.

B. Prior recommendations pushed for discontinuation of HRT in 5 years based on client response and risks and benefits. Current recommendations note that the decision to discontinue HRT should not be based strictly on time, but with full consideration of the client's history, risk profile, and preferences.

CONSULTATION/REFERRAL

A. Consult a gynecologist if the client experiences symptoms resistant to treatment or vaginal bleeding from an unknown source.

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PELVIC INFLAMMATORY DISEASE

DEFINITION

A. Pelvic inflammatory disease (PID) is an inflammation caused by an infection of the upper genital tract. This inflammation can involve the uterine endometrium (endometritis), fallopian tubes (salpingitis), ovaries (oophoritis), broad ligament or uterine serosa (parametritis), and the pelvic vascular system or pelvic connective tissue. PID is often caused by sexually transmitted organisms (*Neisseria gonorrhoeae, Chlamydia trachomatis* [CT]), but can also be caused by microorganisms that compromise the genital flora as well as other microbes.

INCIDENCE

A. Annual incidence is difficult to obtain due to difficulty in definitive diagnosis and reporting.

B. PID is the leading cause of infertility in the world.

PATHOGENESIS

A. PID is caused by organisms that ascend from the vagina and cervix into the uterus. Menses facilitates gonococcal invasion of the upper genital tract as the luteal phase stimulates gonococcal growth and the cervical mucus barrier is removed. Infection and inflammation spread throughout the endometrium to the fallopian tubes. From there, it extends to the ovaries and peritoneal cavity.

B. The most common organisms cultured from clients with PID are CT, *N. gonorrhoeae, Mycoplasma hominis, Ureaplasma urealyticum, Bacteroides, Peptostreptococcus, Escherichia coli*, and some endogenous aerobes and anaerobes.

C. The incubation period varies with the infective organism.

PREDISPOSING FACTORS

A. Age: Rates of PID are higher in females at younger ages. It is highest in the younger than 30-year-old age group (70% incidence younger than age 25). Teens are particularly susceptible because they have an immature immune system and larger zones of cervical ectopy with thinner cervical mucus.

B. Sexual activity: Females with multiple sexual partners are three times more likely to develop PID when compared with females with only one partner.

C. Intrauterine contraceptive device (IUC): IUCs can lead to an iatrogenic development of PID and can promote the spread of vaginal or cervical organisms into the uterus by means of the IUC string.

D. History of PID.

E. Menstruation: supports the development and spread of PID. Individuals who are *not* currently menstruating have a decreased risk.

F. History of invasive procedures: These procedures may result in iatrogenic PID. PID is usually seen within 4 weeks of the procedure (dilatation and curettage [D&C], IUC insertion, hysterosalpingogram [HSG], and vacuum curettage abortion).

G. There is an increased incidence of PID in African Americans, non-White females, and females in lower socioeconomic groups.

H. Cigarette smoking.

I. Frequent vaginal douching.

COMMON COMPLAINTS

A. Lower abdominal pain.

B. Fever or chills.

C. Increased vaginal discharge.

D. Nausea and vomiting.

E. Low back pain.

OTHER SIGNS AND SYMPTOMS

A. Asymptomatic; vague and nonspecific symptoms.
B. Minimal to severe pelvic pain.
C. Right upper quadrant pain (25%).
D. Abnormal vaginal bleeding.

SUBJECTIVE DATA

A. Determine onset, duration, and course of presenting symptoms.
B. Review the characteristics of vaginal discharge (if any); history of recent dysmenorrhea and/or dyspareunia; any intestinal or bladder symptoms.
C. Question the client regarding sexual history: current number of sexual partners; current or most recent sexual activity; and contraceptive used (condoms, pills, diaphragm, IUC), and frequency of method used.
D. Question the client as to whether their current sexual partner has experienced any symptoms.
E. What is the client's current menstrual pattern? Does the client think they are pregnant? When did the pain begin in relation to their cycle?
F. Review prior pelvic or abdominal surgeries and procedures (HSG, abortion) and when they were done.
G. Review the history and quality of pain, how long, bilateral or unilateral, what makes it better, and what makes it worse (intercourse, Valsalva maneuver with bowel movement, activity).

PHYSICAL EXAMINATION

Lower abdominal or pelvic pain is the most common symptom of PID and typically is moderate to severe. However, many clients may have subtle or mild symptoms that are not readily recognizable as PID, including abnormal bleeding, dyspareunia, or vaginal discharge.

A. Check temperature, pulse, respirations, blood pressure (BP), and height and weight to calculate body mass index (BMI).
B. Inspect:
  1. Observe general overall appearance for discomfort before, during, and after exam. Watch client ambulate. Clients with PID often shuffle their feet to avoid pain with movement.
C. Auscultate:
  1. Abdomen for bowel sounds in all quadrants. Auscultation of the abdomen should precede any palpation or percussion due to the changes in intensity and frequency of sounds after manipulation.
D. Percuss:
  1. Abdomen for organomegaly.
E. Palpate:
  1. Palpate the groin for lymphadenopathy.
  2. Palpate the abdomen for masses.
  3. Palpate the levator ani muscle left and right, the urethra, and trigone of the bladder.
  4. Perform rebound, involuntary guarding, and jar tests. **The jar test is performed by intentionally hitting or jarring the examination table and watching for a pain response. Pelvic discomfort is exacerbated by the Valsalva maneuver, intercourse, or movement. Abdominal or pelvic pain with PID is usually bilateral. About 25% of clients complain of right upper quadrant pain; the pain usually occurs within 7 to 10 days of menses, remains continuously, and is most severe in the lower quadrants.**
F. Pelvic examination:
  1. Examine the vulva for Bartholin gland enlargement, fissures, condyloma, herpes, and pelvic relaxation.
  2. Palpate: "Milk" the urethra for discharge.
  3. Speculum examination: Inspect for cervicitis and friability. Evaluate vaginal discharge and bleeding for color, amount, and odor.
  4. Bimanual examination:
    a. Check cervical motion tenderness (CMT); evaluate the size, contour, mobility, and tenderness of the uterus.
    b. Palpate the adnexa for tenderness and masses. The pelvic area may feel hot. **Classic PID presentation is lower abdominal and adnexal tenderness and CMT (chandelier sign).**
  5. Rectovaginal examination: Assess for adnexal thickening and masses.

DIAGNOSTIC TESTS

A. Complete blood count (CBC) with differential; white blood cells (WBCs) greater than 10,500 cell/mm^3.
B. Sedimentation rate or C-reactive protein.
C. Quantitative beta human chorionic gonadotropin (HCG).
D. Rapid plasma reagin (RPR) hepatitis B surface antigen, and HIV.
E. Cultures for gonorrhea (GC) and CT.
F. Transvaginal ultrasonography.
G. Laparoscopy, by referral.

Always test the client for quantitative beta HCG even if the client claims their menses are regular and they are using reliable contraception due to the extreme risk of ectopic pregnancy. Cultures must always be done; lab work (such as HIV and RPR) may be done as indicated, depending on the client's history and presentation. Eliciting data in the health history about hysterectomy, previous appendectomy, abortions, and procedures such as HSG may provide exclusionary diagnoses.

H. Diagnostic criteria for clinical diagnosis of PID:
  1. Maintain a high index of suspicion and err toward the side of overtreatment in favor of prevention of long-term sequelae associated with missed diagnoses. Empiric treatment should be started in sexually active females with pelvic/lower abdominal pain and no other discernable cause and any ONE of the following:
    a. Uterine tenderness.
    b. Adnexal tenderness.
    c. CMT.
  2. Additional criteria to support diagnosis:
    a. Oral temperature greater than 101°F.
    b. Abnormal cervical or vaginal discharge.
    c. Elevated erythrocyte sedimentation rate (>15 mm/hr).
    d. Elevated C-reactive protein.
    e. Laboratory documentation of cervical infection with *N. gonorrhoeae* or *C. trachomatis.*
    f. Abundant WBCs on saline wet prep.
  3. Highly specific criteria for diagnosing PID:
    a. Ultrasound, Doppler, or MRI findings suggesting infection.
    b. Laparoscopic abnormalities consistent with PID.
    c. Endometrial biopsy showing endometriosis.

DIFFERENTIAL DIAGNOSES

A. Gynecologic factors:
  1. PID.
  2. Ectopic pregnancy.

▶

3. Pelvic endometriosis.
4. Dysmenorrhea.
5. Adenomyosis.
6. Functional ovarian cysts.
7. Endometrial polyps or fibroid.
8. Pelvic relaxation.
9. Anatomic abnormalities.

B. Gastrointestinal (GI) factors:
1. Acute appendicitis.
2. Irritable bowel syndrome (IBS).
3. Ulcerative colitis, Crohn's disease (CD).
4. Diverticulitis.
5. Hernia.

C. Genitourinary factors:
1. Cystitis, urethritis interstitial cystitis (IC).
2. Ureteral obstruction.
3. Carcinoma of bladder.

D. Musculoskeletal factors:
1. Myofascial pain.
2. Pelvic floor myalgia.
3. Spinal injuries or degenerative disease.

E. Neurologic factor:
1. Nerve entrapment syndrome.

PLAN

A. General interventions:
1. A low threshold is needed for diagnosis of PID due to the risk of damage to reproductive health. Early treatment with the use of antibiotics of an upper genital tract infection is imperative. Other causes of lower abdominal pain, such as IBS and endometriosis, are not likely to be impaired by empiric antibiotic therapy. The risk of ectopic pregnancy is 6 to 10 times greater in clients with PID compared with uninfected individuals.
2. Antibiotic therapy should be instituted promptly, based on clinical diagnosis without awaiting culture results, to minimize the risk of progression of the infection and risk of transmission of the organisms to other sexual partners.
3. Evidence is insufficient to recommend intrauterine device (IUD) removal in clients with PID. If it is left in place, follow-up is essential.
4. Ambulatory clients should be monitored closely and reevaluated within 3 days of initiating antibiotic therapy. A decrease in pelvic tenderness should be observed within 3 to 5 days of initiation of therapy; if not, additional evaluation is warranted.

B. Client teaching: *See Client Teaching Guide for this chapter, "Pelvic Inflammatory Disease."*
1. All partners of clients with PID must be examined, cultured when possible, and treated empirically for presumptive gonorrheal and chlamydial infection.
2. Females who do not use any contraception are at the greatest risk. Transmission of sexually transmitted infections (STIs) can be minimized with effective use of condoms.

C. Pharmaceutical therapy:
1. The current (2021) Centers for Disease Control and Prevention (CDC) guidelines are listed in Table 17.7.

D. Alternative intramuscular (IM)/oral regimens:
1. See the most current CDC STI treatment guidelines for alternative IM or oral regimens.
2. Diagnostic tests for gonorrhea must be obtained before initiating therapy, and persons should be managed as follows:
   a. If the culture for gonorrhea is positive, treatment should be based on results of antimicrobial susceptibility testing.
   b. If the isolate is determined to be quinolone resistant Neisseria gonorrhoeae or if antimicrobial susceptibility cannot be assessed (e.g., if only nucleic acid amplification testing [NAAT] is available), consultation with an infectious disease specialist is recommended.

FOLLOW-UP

A. Clients should demonstrate clinical improvement within 3 days of starting therapy. If no improvement is noted in less than 72 hours of outpatient IM or oral therapy, the client should be hospitalized for additional assessment, diagnostics, and management.

TABLE 17.7 2021 CDC RECOMMENDATIONS FOR TREATING PID

Recommended Parenteral Treatment Options	Recommended Intramuscular or Oral Treatment Options
Cefotetan 1 g IV Q24H Plus Doxycycline 100 mg IV or orally Q12H Plus Metronidazole 500 mg orally or IV Q12H	Ceftriaxone 500 mg IM in a single dose* Plus Doxycycline 100 mg orally BID for 14 days With metronidazole 500 mg orally BID for 14 days
or	or
Cefotetan 2 g IV Q12H Plus Doxycycline 100 mg IV or orally Q12H	Cefoxitin 2 g IM in a single dose with Probenicid 1 g orally administered concurrently in a single dose plus Doxycycline 100 mg orally BIDtwice a day for 14 days Wwith Metronidazole 500 mg orally BID2 times a day for 14 days
or	or
Cefoxitin 2 g IV Q6H plus Doxycycline 100 mg IV or orally Q12H	Other third-generation cephalosporin Doxycycline 100 mg orally BID for 14 days with metronidazole 500 mg orally BID for 14 days

[a] For clients weighing more than 150 kg with documented gonococcal infection, 1 g of ceftriaxone should be used.

CDC, Centers for Disease Control and Prevention; IM, intramuscular; IV, intravenous; PID, pelvic inflammatory disease.

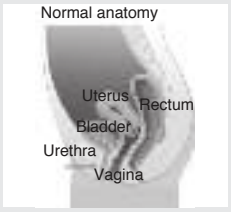
B. Due to the high risk of reinfection, the CDC recommends that all clients who have received a diagnosis of chlamydial or gonococcal PID should be retested 3 months after treatment, regardless of whether their sex partners were treated. If retesting at 3 months is not possible, these clients should be retested whenever they next present for medical care in the 12 months following treatment.

CONSULTATION/REFERRAL

A. Consult a gynecologist if the client's diagnosis is a typical, evidence for a presumptive diagnosis is present, or hospitalization is required. The following are the general criteria for hospitalization:

1. Diagnosis is uncertain.
2. Pelvic or tubo-ovarian abscess is suspected.
3. IUC in situ.
4. The client is pregnant.
5. The client is an adolescent or is believed to be incapable of adhering to outpatient regimen.
6. Outpatient therapy fails; the client is not better in 48 to 72 hours.
7. The client cannot be reevaluated in 48 to 72 hours.
8. The client is HIV-positive.
9. There is generalized peritonitis, nausea and vomiting, high fever, or other signs of severe illness.
10. The client cannot tolerate oral medication therapies.
11. Surgical emergencies cannot be ruled out.

TABLE 17.8 ANATOMIC DESCRIPTION OF POP

Previous Descriptors for POP	Current Anatomic Definitions
	Normal pelvic floor support with bladder located anteriorly; uterus located superior to the bladder
	Anterior vaginal wall prolapse
	Posterior vaginal wall prolapse
	Vaginal vault prolapse

POP, pelvic organ prolapse.
Source: From Goldwasser, S. (n.d.). Pelvic organ/vaginal prolapse. https://bladderdoc.com/condition/pelvic_organ. Used with permission from Steven Goldwasser, MD, Urogynecology Clinic, Jacksonville, FL.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Fluoroquinolones are generally contraindicated in pregnant and nursing mothers.
B. Partners:
1. Sexual partners should be evaluated and treated for STIs.

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PELVIC ORGAN PROLAPSE

DEFINITION

A. Many terms have been used to describe pelvic organ prolapse (POP), the protrusion or downward movement of the pelvic organs through the vaginal introitus. Frequently used descriptors include cystocele, urethrocele, rectocele, enterocele, vaginal prolapse, and uterine prolapse. Each term describes a specific organ that has been displaced and, while frequently used by clinicians, these descriptors do not accurately and completely describe the extent of what is actually occurring. Thus, it is recommended that when describing what is seen on exam, one may define prolapse by using anatomic definitions, such as anterior vaginal wall prolapse, apical prolapse, posterior vaginal wall prolapse, vault eversion, cervical prolapse, or rectal prolapse (Table 17.8).
B. POP is also defined by stages of descent:
1. Stage 0: no evidence of descent.
2. Stage 1: very minimal and usually asymptomatic descent.
3. Stage 2: protruding to the vaginal hymen and slightly beyond.
4. Stage 3: protruding well beyond the hymen.
5. Stage 4: complete or almost complete descensus.

INCIDENCE

A. POP is a common problem affecting females of all ages, races, and ethnicities.
B. Most authorities describe the incidence as somewhere between 10% and 30% of the female population, with increased incidence observed with advancing age. The Women's Health Initiative (WHI) found a 41% incidence of POP among clients aged 50 to 79, with the most commonly observed compartment being the anterior wall. Interestingly, researchers from the Pelvic Organ Support Study (POSST) saw that on routine gynecologic exams of asymptomatic females, 35% experienced stage 2 POP and 2% had descent that measured to stage 3.
C. It is believed that as the U.S. population continues to age and to expand, the incidence and prevalence of prolapse will continue to grow.

PATHOGENESIS

A. Although not fully understood, some logical pathologic factors may create the occurrence of POP.
B. Some evidence suggests that weakness of the pelvic floor muscles (PFMs) creates a more vertical angle for the vagina, thus no longer supporting the vagina and the pelvic organs in a slightly horizontal fashion:
1. This loss of a "backboard" increases the chance for the pelvic organs to be affected by gravity and descend downward through the canal.

2. Weakness of the muscles may also create an inability of the vaginal introitus to contract or close during periods of increased intra-abdominal pressure, such as during a cough.
3. Over time this can also allow the pelvic organs to descend through the widened opening.

C. Much research is ongoing into the role of connective tissue injuries that may occur to increase the risk of POP:
1. Direct injury to the muscles and connective tissue of the pelvic floor, such as during childbirth, may combine to encourage a herniation of the pelvic organs through the tear or tears in the fascia.
2. A more novel and very interesting theory involves the observation of possible familial or hereditary causes of prolapse.
3. Studies have indicated both "structural and biochemical" changes observed in the collagen of microscopically analyzed prolapsed tissue.
4. Other research has shown a weakening of collagen with increasing age. Over time the collagen becomes weaker and faster degradation is observed, thus increasing the prevalence of POP. This raises the question regarding whether this is a "normal" process of aging versus an actual hereditary cause for hastening of the collagen deprivation and resulting prolapse.
5. Research into all these theories of pathology continues to expand as we try to understand the root causes of prolapse and possible means for prevention.

PREDISPOSING FACTORS

A. Many external and some potentially preventable risk factors can combine to create the risk for POP. Yet it is not fully understood which of these factors creates the greatest risk:
1. Pregnancy and vaginal childbirth: Evidence supports the lack of consensus regarding pregnancy versus delivery as the predisposing risk factor. Other obstetric factors considered include the following:
a. Macrosomia.
b. Length of second stage of labor.
c. Midline episiotomy.
d. Forceps-assisted vaginal deliver.
e. Anal sphincter laceration.
f. Oxytocin use for labor initiation.
g. Epidural analgesia.
h. Higher gravidity.
2. Age and menopausal status:
a. The risk of POP doubles with each decade of life.
b. It is unclear how the loss of estrogen and the effects of age can be differentiated.
c. Questions remain regarding age, menopause, and collagen loss with aging.
3. Race:
a. African American and Asian females have the lowest risk for POP.
b. Hispanic females appear to have the highest risk, with white females directly following.
4. Hysterectomy:
a. Route of hysterectomy does not appear to influence risk, although some evidence exists regarding the benefit of keeping the cervix for prevention of POP after hysterectomy.
b. POP after a hysterectomy performed for the indication of prolapse appears to be a greater risk than hysterectomy for other causes.
5. Connective tissue disease:
a. Ehlers–Danlos syndrome.
b. Marfan syndrom.
6. Increases in intraabdominal pressure:
a. Obesity.
b. Chronic constipation.
c. Chronic lifting (conflicting data).

COMMON COMPLAINTS

A. Clients may experience a plethora of symptoms that may or may not be connected to the POP. Furthermore, research has shown a direct correlation between increased stage of prolapse and symptom occurrence. Many of the complaints are more noticeable to the client as the prolapse advances beyond the hymen. It is also important to note that while many of these complaints may be identified by the client as being caused by the POP, they may not be directly related to the problem at all. This is noted in the inconsistencies of prolapse symptoms in the literature.
1. Feeling of a bulge or ball in the vagina.
2. Vaginal or pelvic pressure.
3. Rubbing of bulge against undergarments and/or vaginal bleeding.
4. Concurrent urinary incontinence (UI).
5. Urinary urgency or frequency.
6. Urinary hesitancy.
7. Constipation and/or anal incontinence (AI).
8. Incomplete rectal evacuation/stool trapping.
9. Low back pain.
10. Sexual dysfunction/dyspareunia.
11. Pelvic pain.

B. When determining the severity of symptoms, the clinician may find it helpful to use validated prolapse symptom questionnaires, including the Pelvic Floor Distress Inventory (PFDI), found at centerforadvancedgyn.com/wp-content/uploads/2016/06/pelvic-floor-disability-index.pdf; Urinary Distress Inventory 6 (UDI-6), found at www.ohsu.edu/sites/default/files/2019-06/Female-Urology-Questionnaire-6.pdf; the Pelvic Organ Prolapse Distress Inventory 6 (POPDI-6), found at everybodyphysicaltherapy.com/wp-content/uploads/2014/05/PFDI.pdf; or the the Pelvic Floor Impact Questionnaire, found at www.nppt.com/files/forms/PFIQShortform.pdf.

POTENTIAL COMPLICATIONS

A. Most prolapse complications center around the impact on the client's quality of life. However, some researchers and clinicians believe some direct, serious complications may occur as a result of increasing stages of POP, including the following:
1. Urinary retention, with resulting increased risk for hydronephrosis and hydroureter.
2. Vaginal ulcers/sores/infection and vaginal bleeding.
3. Recurrent urinary tract infection (UTI).

SUBJECTIVE DATA

A. Ask the client if they feel a bulge or a feeling of pressure in the vagina.
B. Does the client feel as if they are sitting on something, much like a ball?
C. Ask the client if they feel something rubbing against their undergarments. ▶

D. Have the client describe whether symptoms worsen when standing.

E. Ask client to describe if they need to manually assist themselves to empty their bowel or bladder. Do they need to maneuver on the toilet?

F. Determine if the client has had any prior treatments for the prolapse, surgical, or conservative.

G. Ask about progression of the bulge; has it become more pronounced over time?

H. Is it difficult to have sex, as if something is blocking the opening of the vagina?

I. Review the client's full obstetric history specifically for factors as noted earlier.

J. Review the client's medical history with specific queries about chronic obstructive pulmonary disease (COPD) and smoking.

K. Review all medications, including over-the-counter (OTC) and herbal products. Evaluate problems such as chronic cough secondary to angiotensin-converting enzyme (ACE) inhibitors.

PHYSICAL EXAMINATION

A. As clients with POP may have other conditions caused by pelvic floor dysfunction, it is important for the clinician to perform a thorough examination to evaluate the full spectrum of problems and the resulting impact on function and quality of life.

B. General inspection of external genitalia performed with client in lithotomy position:

1. Evaluate the perineal area for skin rash, such as with changes consistent with atrophy, as well as bowel or bladder incontinence.

2. Evaluate for other skin changes indicative of vulvar dermatoses or yeast infections.

3. Evaluate for allodynia: a positive pain response to light touch.

4. Evaluate the urethra for discharge, masses, or atrophy.

5. Perform a cough stress test (CST) to evaluate for stress urinary incontinence (SUI):

a. This test is positive if leakage is noted during the cough.

b. A positive result may indicate a significant weakness of the urethral sphincter or a poorly supported urethra.

c. While performing the test, look to see if the urethra is hypermobile as this may indicate SUI, even if the CST is negative for leakage.

C. Neurologic exam of sacral reflexes:

1. Evaluate S2 to S4 dermatomes:

a. Evaluate for symmetry of sensation.

b. Evaluate soft sensation with cotton-tipped end of Q-tip.

c. Evaluate sharp sensation with wooden end of Q-tip.

2. Bulbocavernosus reflex:

a. Elicit the reflex response by using a cotton swab; gently stroke the bilateral labia majora looking for a positive reflex response by observing subtle contraction or movement of the labial tissue (Figure 17.1).

b. No movement of the tissue is a negative response, which may indicate nerve damage to the pelvic floor.

c. Up to 20% of normal clients may have a negative response.

d. Neurologic testing for the bulbocavernosus reflex is also useful in evaluating a cauda equina lesion.

3. Anal reflex, "wink":

a. Using the cotton-tipped end of Q-tip, strike bilateral sides of the anal meatus, observing for contraction (see Figure 17.1).

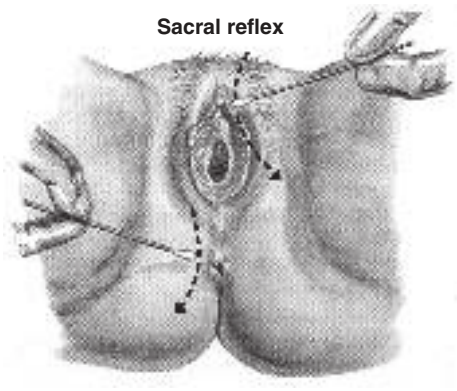


FIGURE 17.1 Pelvic floor neurologic testing: bulbocavernosus and anal "wink" reflexes.

b. Up to 25% of normal clients may have absent response.

D. Pelvic organ prolapse quantification (POPq) exam:

1. System of measurement of prolapse defined by the International Continence Society (ICS).

2. The prolapse is measured in specific segments or sections, with all measured points in relation to the hymen.

3. Exam is performed with the client in either lithotomy or standing positions.

4. A split speculum and measuring stick are used to visualize and measure the extent of the prolapse (Figure 17.2).

5. All points of measurement are performed during Valsalva with exception of one: total vaginal length (TVL), which is a resting measurement.

6. Measured points are plotted on a "Tic-Tac-Toe" grid (Figure 17.3).

a. Point Aa: anterior wall; this point can be 3 cm from the hymen to its fullest extent, +3 cm beyond the hymen.

b. Point Ba: This is the more proximal or deeper portion of measurement of the anterior wall located from the vaginal cuff/cervix or anterior vaginal fornix to point Aa. In the absence of prolapse, the value would be −3 cm from point Aa and with prolapse it can fall anywhere from −2 cm from the hymen to a positive value that can be equal to the cuff's length. For example, point Ba can be −3 to +10, depending on the length of the anterior wall to the cuff.



FIGURE 17.2 Split speculum and measuring stick.

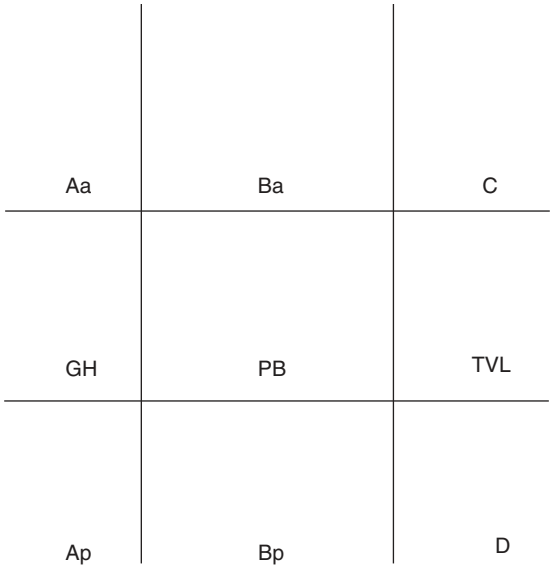


FIGURE 17.3 Tic-Tac-Toe grid for plotting prolapse.

c. Point C: This point represents the most distal edge of the cervix or vaginal cuff if no uterus is present.

d. Point D: This measurement is not included in the POPq measurement if the client has had a hysterectomy; this point differentiates a well-supported uterus from true uterine prolapse, and measurement is taken from the posterior fornix in the client with a cervix. This point represents the level of the uterosacral ligament. Point D can be −10 to as much as +10.

e. Point Ap: This point is a measurement of the posterior wall, and like the anterior wall this point can be from the hymen to +3 cm beyond the hymen.

f. Point Bp: This is the more proximal or deeper portion of measurement of the posterior wall located from the vaginal cuff or posterior fornix to point Ap. In the absence of prolapse, the value would be −3 cm from point Ap and with prolapse it can fall anywhere from −2 cm from the hymen to a positive value that can be equal to the cuff's length. For example, point Bp can be −3 to +10, depending on the length of the posterior wall to the cuff/fornix.

E. Stages of prolapse:

1. Stage 0: no prolapse.

2. Stage 1: leading edge of POP comes to within 2 cm of hymen.

3. Stage 2: leading edge of POP moves from −1 cm above to +1 cm beyond hymen.

4. Stage 3: leading edge of POP is +2 cm beyond the hymen but is still 2 cm less than the TVL.

5. Stage 4: complete prolapse (Figure 17.4).

DIAGNOSTIC TESTS

A. Obtain a clean-catch urinalysis to screen the urine for clarity, hematuria, and infection:

1. Send the urine for culture and sensitivity to evaluate for presence of infection.

2. Urine for cytology is recommended for evaluation of hematuria.

B. Immediately after void, obtain a postvoid residual (PVR) by inserting a catheter into the bladder or use a bladder scanner.



FIGURE 17.4 Complete prolapse.

Source: Used with permission of Barry Jernigan, MD, Center for Pelvic Health. http://www.centerforpelvichealth.org.

C. Blood urea nitrogen (BUN), creatinine, for evaluation of kidney function.

D. Pelvic ultrasound for evaluation of uterus and ovaries if there is vaginal bleeding. Consider an endometrial biopsy if ultrasound shows an abnormal endometrium.

E. Pap smear if indicated by guidelines.

F. Urodynamic testing for evaluation of bladder function and UI.

G. Cystoscopy for evaluation of bladder structure and hematuria. CT scan to evaluate for hydronephrosis and hydroureter (important to assess if client is experiencing increased PVRs).

DIFFERENTIAL DIAGNOSES

A. Prolapsed uterine fibroid.

B. Anterior or posterior vaginal wall cyst.

C. Ureterocele.

D. Rectal prolapse.

PLAN

A. Client teaching:

1. Educate client about proper body mechanics and avoidance of repetitive, chronic strain.

2. Smoking cessation.

3. Discuss perineal hygiene to avoid irritation to the vulva and prolapsing vagina.

B. Treatment options:

1. Observation.

2. Pelvic floor muscle exercises (PFME):

a. Little research exists to indicate that Kegel exercises can reduce prolapse stage.

b. Primary goals for pelvic floor physical therapy (PFPT) serve to improve symptoms of prolapse, enhance proper body mechanics, and serve as a potential prevention for increasing prolapse stage.

3. Pessary (Figures 17.5–17.8):

a. Offered as an option for management of the prolapse; not curative.

b. Pessary is used for the client who desires nonsurgical treatment or is unable to achieve surgical clearance.

c. Can be for temporary or long-term use.

d. The client must undergo a pessary-fitting session. See Section II: Procedures, "Pessary Insertion and Management." ▶



FIGURE 17.5 Ring with support pessary.

Source: Used with permission from CooperSurgical. coopersurgical.com.

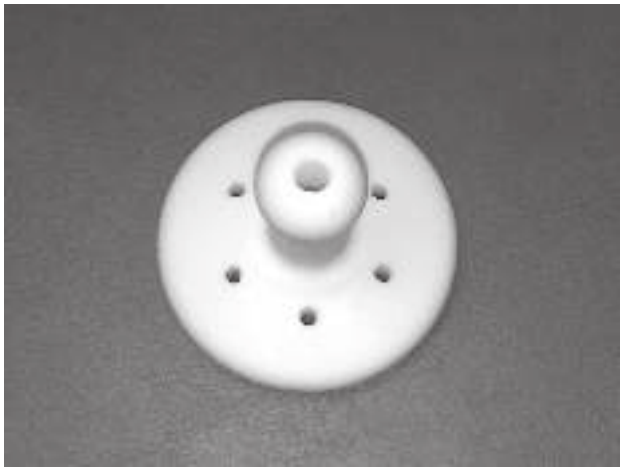


FIGURE 17.6 Gellhorn pessary.

Source: Used with permission from CooperSurgical. coopersurgical.com.



FIGURE 17.7 Donut pessary.

Source: Used with permission from CooperSurgical. coopersurgical.com.



FIGURE 17.8 Cube pessary.

Source: Used with permission from Cooper Surgical. (2018). Milex cube pessary. Retrieved from https://www.coopersurgical.com/Medical-Devices/Detail/milex-cube-pessary.

e. Follow-up schedule is dependent on client's willingness to manage the pessary.
f. Clinician must be aware of potential complications with pessary use:
   i. Pain may occur if pessary is too large.
   ii. Pessary may fall out if it is too small.
   iii. Vaginal bleeding.
   iv. Erosion or ulcer.
g. Reasons clients discontinue use of pessaries:
   i. Unsuccessful, especially in clients with posterior prolapse.
   ii. Difficulty removing.
   iii. Inability to void.
   iv. Client desires surgery.

4. Surgery: Surgical correction techniques will not be discussed in this chapter and require the evaluation of a subspecialist physician.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy and reproductive-age individuals:

1. Pregnancy is not a contraindication to pessary use.

2. Practice of PFME is acceptable during pregnancy.

3. In the absence of large prolapse, no treatment is necessary during pregnancy if the client and baby are stable.

4. If the client has not completed childbearing, surgical correction is not recommended. Offer a pessary or PFME to this client until they are past this stage.

B. Geriatrics:

1. Physical examination in the elderly:

a. The pelvic examination may be more difficult related to the lack of hip mobility, arthritis, or spinal deformities.
b. The examination may need to be performed with the client turned to their left side.
c. Allow extra time to explain each step of the examination and for positioning. An assistant can help the client onto the exam table and may help gently support the client's legs if necessary.

2. Must consider the geriatric client's ability to manage their pessary:

▶

a. Frailty and decreased mobility may prevent the client's personal management of a pessary. Scheduled follow-up care with provider can remove, clean, and replace the pessary.
b. Dementia may inhibit the geriatric client's ability to perform a regimen of PFME.
c. Close family involvement is important to ensure proper management and follow-up for the geriatric client with dementia.
d. If surgical correction is desired in this client, preoperative clearance must be obtained. In cases of dementia, family members must provide consent.

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PREMENSTRUAL SYNDROME AND PREMENSTRUAL DYSPHORIC DISORDER

DEFINITION

A. Premenstrual syndrome (PMS) is a psychoneuroendocrine disorder with a constellation of symptoms that occur in the luteal phase and interfere with an individual's life; this is followed by a symptom-free period. According to the American Congress of Obstetricians and Gynecologists (ACOG, PMS is diagnosed if the individual has symptoms that are present in the 5 days before menses for at least three menstrual cycles in a row, end within 4 days after the menses starts, and interfere with some normal activities of life. Moderate to severe forms of premenstrual distress are now classified in the American Psychiatric Association's (APA) *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; 2013) as premenstrual dysphoric disorder (PMDD).

INCIDENCE

A. Virtually every menstruating individual experiences some symptoms sometimes. There is about a 25% prevalence rate when the ACOG diagnostic standards are applied; however, this rate may be lower than actual rates due to nonreporting of mild to moderate symptoms.

PATHOGENESIS

A. The basis of PMS and PMDD is presumably hormonal. During the luteal phase, progesterone levels increase and estrogen levels decrease, causing a shift in the ratio of these hormones, which contributes to the symptoms experienced during PMS. These hormones are also known to interact with neurotransmitters in the brain, such as serotonin, and these interactions are thought to cause some of the symptoms experienced, such as changes in mood and pain thresholds, during PMS and PMDD.

PREDISPOSING FACTORS

A. Female of reproductive age.
B. First-degree female relatives affected.
C. History of stressful life events.
D. History of sexual and/or physical abuse.

SUBJECTIVE DATA

A. Persistent and marked anger or irritability.
B. Marked anxiety or tension.
C. Markedly depressed mood and feelings of hopelessness.
D. Decreased interest in usual activities.
E. Easily fatigued or a marked lack of energy.
F. Subjective sense of difficulty in concentrating.
G. Hypersomnia or insomnia.
H. A subjective sense of being overwhelmed or out of control.
I. Marked change in appetite, overeating, or specific food cravings.
J. Physical symptoms such as breast tenderness, headaches, edema, abdominal bloating, joint or muscle pain, and weight gain.

OTHER SIGNS AND SYMPTOMS

A. The symptoms interfere with work, usual activities, or relationships.
B. The symptoms are not an exacerbation of another psychiatric disorder.
C. "I've got PMS; I'm so miserable."
D. Feelings of irritability and emotional lability.

SUBJECTIVE DATA

A. Obtain a complete menstrual history:
1. Menarche; frequency, duration, and regularity of periods.
2. Ask about premenstrual symptoms that are physical: weight gain, edema, acne, nausea, vomiting, constipation, backache, headache, migraine, syncope, breast tenderness, breast swelling, hot flashes, paresthesia of hands or feet, aggravation of convulsive disorder, increased appetite, food cravings (sweets, salt, or food in general), and fatigue.
3. Ask about premenstrual symptoms that are emotional: irritability, emotional lability, anxiety, depression, crying, palpitations, fatigue, aggression, lethargy, and sleep disturbances.
4. Ask particularly about the timing of the symptoms: When do the symptoms begin and end in relationship to the menstrual period? Has the client kept a calendar of symptoms?
B. Ask about symptoms of dysmenorrhea. Some females confuse menstrual cramps and PMS.
C. Note type of contraception the client uses.
D. Review the client's obstetric history, if applicable.
E. Elicit the types of treatment the client has tried and efficacy of treatment.
F. Ask the client about the amount and type of exercise they get. Individuals with PMS often get little exercise.

PHYSICAL EXAMINATION

A. Check height, weight, and blood pressure (BP). Calculate body mass index (BMI).
1. Note overall appearance; inspect thyroid.
B. Palpate:
1. Palpate the neck, noting thyroid enlargement or nodules. Palpate the abdomen, noting enlargement, masses, or tenderness.
C. Auscultate:
1. Auscultate the heart, lungs, and abdomen.
D. Pelvic examination:
1. Inspect the external genitalia for pubic hair pattern, lesions, or discharge.
2. Speculum examination: Check for discharge and lesions.
3. Bimanual examination: Check for size, mobility, shape, and tenderness of the uterus and adnexal area.
4. No physical abnormality or changes are consistent with PMS.

DIAGNOSTIC TESTS

A. PMS is diagnosed primarily based on the pattern of symptoms; however, the following diagnostic tests should be considered if indicated:

1. Pap and human papillomavirus (HPV) testing, as indicated by guidelines.

2. Screen for sexually transmitted infections (STIs), as indicated.

3. Thyroid-stimulating hormone (TSH).

4. Screen for psychiatric disorders, as indicated.

DIFFERENTIAL DIAGNOSES

A. PMS.
B. PMDD.
C. Major depression.
D. Dysmenorrhea.
E. Substance abuse.
F. Perimenopausal symptoms.
G. Sexual dysfunction.
H. Fibromyalgia.
I. There are rarely major medical problems, but hypothyroidism, hyperthyroidism, anemia, and autoimmune disorders (such as systemic lupus erythematosus [SLE]) must be kept in mind.

PLAN

A. General interventions:

1. Have the client keep a menstrual calendar or diary for at least 3 months to document occurrence of symptoms in the luteal phase.

2. Symptomatic treatments: Treatment must be individualized.

B. Client teaching: *See Client Teaching Guide for this chapter, "Premenstrual Syndrome and Premenstrual Dysphoric Disorder."*

1. Diet: Have the client eat six small meals a day to even out glucose load. Have them avoid caffeine to decrease irritability and facilitate sleep. Encourage them to avoid simple sugars and eat complex carbohydrates to provide a slow, steady source of energy. They should avoid salt to decrease edema.

2. Activity: Instruct the client to increase exercise, preferably aerobic exercise. Suggest exercising every day (walking, swimming, and stretching). Encourage stress reduction activities such as imagery or yoga, as well as support or counseling groups. Encourage smoking cessation as well as adequate sleep and rest.

C. Pharmaceutical therapy:

1. Nonsteroidal anti-inflammatory drugs (NSAIDs) for relief of muscular aches, headaches, and menstrual

TABLE 17.9 MEDICATIONS USED WITH PMS AND PMDD

Drug	Dose	Purpose
Antidepressants		Decreases depression and anxiety and improves mood
Fluoxetine (Prozac)	10–40 mg every day or during the luteal phase (individual dose may vary); alternatively, may be taken daily	
Paroxetine (Paxil)	10–30 mg every day or during the luteal phase (individual dose may vary); alternatively, may be taken daily	
Sertraline hydrochloride (Zoloft)	25–30 mg every day or during the luteal phase (individual dose may vary); alternatively, may be taken daily	
Combined hormonal contraceptive modes (pill, patch, ring)	Take on a daily basis	Evens the hormonal milieu, blocks ovulation
Oral contraceptive pills (Yaz) *Only combined oral contraceptive pills that contain progesterone drospirenone in addition to EE are FDA approved to treat PMD	Take on a daily basis	Evens the hormonal milieu, blocks ovulation
Antianxiety Drugs		Decreases anxiety
Alprazolam (Xanax) Diazepam (Valium) Lorazepam (Ativan)	**Not considered first line treatment. Many anxiolytics cause addiction, tolerance, and sedation. Short term only. Use with caution.**	
Buspirone (BuSpar)	7.5–15 mg BID	
Diuretics		Decreases edema peripherally and, perhaps, centrally
Spironolactone (Aldactone)	25 mg BID as needed	

EE, ethinylestradiol; FDA, Food and Drug Administration; PMDD, premenstrual dysphoric disorder; PMS, premenstrual syndrome.

cramps. Follow directions for the particular NSAIDs, whether over-the-counter (OTC) or prescription.

2. Minerals:

a. Calcium 500 mg/d may offer some benefit.

3. Vitamins are used to decrease anxiety and irritability, food cravings, breast pain, depression, fatigue, and lethargy:

a. Vitamin B_6 (pyridoxine) 50 to 150 mg daily (limited benefit).

b. Vitamin E 400 to 600 mg daily (limited benefit).

4. Herbs and botanicals:

a. Curcumin 100 mg BID beginning 7 days before cycle until 3 days after cycle starts (some evidence of effectiveness).

b. Vitex Agnus-Castus (chaste tree berry) 30 to 40 mg daily (little evidence on effectiveness).

c. Evening primrose oil (EPO) 500 to 1,000 mg each day (mixed results on effectiveness).

5. See Table 17.9 for other therapies.

FOLLOW-UP

A. Follow up every 3 to 4 months to assess or alter treatment and/or therapy.

CONSULTATION/REFERRAL

A. Consult a physician if symptoms are severe or not relieved by first- or second-line therapies.

INDIVIDUAL CONSIDERATIONS

A. Partners:

1. Encourage the client to have their partner come to a visit. Partner education and support are helpful.

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PREVENTIVE CARE FOR ADULT SURVIVORS OF SEXUAL VIOLENCE

DEFINITION

A. Preventive care: care provided to prevent pregnancy and/or sexually transmitted infections (STIs) after sexual assault or rape.

B. Adult survivors: persons 18 years of age or older who have survived sexual violence.

C. Sexual violence includes sexual assault and rape. The legal definitions of each vary from state to state. The terms include the following:

1. Sexual assault: any sexual act performed on a person without consent.

2. Rape: penetration of a body orifice without consent.

INCIDENCE

A. According to the Rape, Abuse and Incest National Network (RAINN), it is estimated that every 68 seconds a person will experience sexual violence. One in six female-identifying and 1 in 33 male-identifying individuals will be the victims of sexual violence in their lifetimes.

B. The incidence of sexual violence is highest, at 54%, in ages 18 to 34; declines to 28% in ages 35 to 64; and is lowest, at 3%, in ages 65 and up. The exact incidence of sexual assault and rape is hard to determine as many go unreported.

PREDISPOSING FACTORS

A. Predisposing factors generally include lifestyle factors and characteristics that may make an individual vulnerable to a certain condition:

1. Individuals who are sexually assaulted or raped are sometimes targeted by the perpetrator.

2. Others may be victims of the perpetrator's convenience who are selected at random.

B. Although alcohol and prescription and recreational drugs are substances that may affect judgment and lead to unsafe choices, the voluntary ingestion of these substances does not imply that the survivor is responsible for sexual violence.

COMMON COMPLAINTS

A. Sexual violence survivors may present with explicit memory, much confusion, or no memory of the assault.

OTHER SIGNS AND SYMPTOMS

A. Survivors may report injuries associated with physical force during the assault.

EVALUATION

A. Every effort should be made to have the initial acute evaluation performed by care providers who are trained and have expertise in the care of people who have been sexually assaulted. Sexual Assault Nurse Evaluation (SANE) nurses and Sexual Assault Forensic evaluators (SAFE clinicians) are examples of such providers. Providers trained in the care of sexual assault have expertise in conducting this sensitive history and physical, as well as collecting and protecting evidnce that can aid in the prosecution of the perpertrator. These care providers also have knowledge of community resources such as counsleing and referrals as needed.

SUBJECTIVE DATA

A. A detailed history of the body parts and actions involved in the assault is essential in guiding the physical examination and determining the management plan.

B. Regardless of how much time has elapsed, recounting details of the assault can be emotionally difficult for the survivor. To alleviate some of the discomfort, providers should explain how they will use information they are requesting. For example, if oral penetration occurred but there was no genital contact, the physical exam would be limited to the oral cavity.

C. Review a full medication history, asking specifically about the use of hormonal contraception and CYP3A4 inducers.

D. Review all allergies, including medications, latex, and copper (if desires intrauterine device [IUD] insertion).

E. Review medical history for any contraindications to contraception.

F. Review immunization history:

1. Human papillomavirus (HPV).

2. Hepatitis B virus (HBV).

3. Tetanus, if no history of immunization in the past 10 years of a booster is indicated.

G. Ask about current pregnancy or previous exposure for pregnancy. Previous exposure refers to consensual, unprotected sex within the past 72 to 96 hours.

H. Is the client breastfeeding?

I. When was the client's last alcohol ingestion?

PHYSICAL EXAMINATION

A. The physical examination is limited to inspection of body parts involved in the assault.

DIAGNOSTIC TESTS

A. Pregnancy testing:

1. Pregnancy testing should be offered if the survivor has had consensual, unprotected intercourse since their last menstrual period (LMP):

a. Urine pregnancy test is advisable to do point-of-care testing whenever possible due to the low rate of follow-up. The urine test provides results at the time of the examination.

b. Serum beta human chorionic gonadotropin (HCG) may be offered if earlier detection is essential.

2. Diagnostic testing for sexually transmitted infections (STIs), alcohol, and recreational drugs is controversial. If done in days after the acute assault, STI testing will only confirm the presence of prior infection, not infection resulting from the assault. All test results become a part of the medical record. Should the survivor choose to pursue legal prosecution of the assailant, it is possible that positive results prior to the assault may be used to discredit the survivor. Current infection status is not required to prescribe preventive treatment. Survivors should be provided with this information to make a fully informed decision regarding testing.

PLAN

A. Emergency contraception (EC) options:

1. Levonorgestrel (LNG) 1.5 mg:

a. Available over-the-counter (OTC) in a single-tablet regimen to anyone of any age.

b. Available behind-the-counter in a two-tablet regimen to persons ages 17 years and older.

c. 50% efficacy if taken within 72 hours of exposure.

d. Efficacy may be decreased if body weight is greater than 165 lbs.

2. Ulipristal acetate (UPA): 30 mg:

a. Prescription only.

b. Limited product distribution to pharmacies. Online ordering may be necessary.

c. 66% efficacy if taken within 120 hours of exposure.

d. Contraindicated with breastfeeding.

e. Concomitant use with CYP3A4 inducers or hormonal contraception may decrease effectiveness.

f. Efficacy may be decreased if body weight is greater than 195 lbs.

3. Copper IUD:

a. Must be inserted by trained professional.

b. Hormone-free.

c. Contraindicated with copper allergy.

d. No drug interactions.

e. 99% efficacy if inserted within 120 hours of exposure.

B. EC client teaching:

1. The primary mechanism of action for LNG and UPA is to inhibit or delay ovulation.

2. The primary mechanism of action for the copper IUD is inhibition of fertilization through release of copper ions toxic to sperm.

3. LNG is a progestin. UPA is an antiprogestin. The two agents cannot be used together as they will negate effectiveness of the other.

4. Survivors using hormonal contraception regularly do not need EC. Survivors using hormonal contraception irregularly should be advised to use LNG not UPA for EC.

5. Survivors wanting to begin or resume hormonal contraception after UPA use should be advised to wait at least 120 hours.

6. The copper IUD may be left in place as contraception for up to 12 years after insertion.

7. Survivors with limited financial access to EC can find the nearest low-cost family planning clinics by typing in their zip codes at this Health and Human Services sponsored site: opa-fpclinicdb.hhs.gov.

8. If EC is declined, advise pregnancy testing 2 weeks after assault.

C. STI prophylaxis:

1. *Chlamydia trachomatis* (CT): doxycycline 100 mg orally BID for 7 days.

2. Gonorrhea (GC): ceftriaxone 500 mg intramuscularly x 1 for a person weighing <150 kg or 1 g for persons weighing >150 kg.

3. Trichomoniasis: metronidazole 500 mg orally BID for 7 days for females and metronidazole 2 g orally in a single dose for males.

4. HBV:

a. Exposure source hepatitis B surface antigen (HBsAg)-positive:

i. Unvacinated or person who did not respond to vaccine should receive both hepatitis B immune globulin (HBIG) and hepatitis vaccine as soon as possible.

ii. Person exposed and had not yet completed hepetitis B vaccine schedule should receive HBIG and complete vaccine series.

iii. Person with documentation of vaccine series and did nto receive postvaccination testing should receive single vaccine booster dose.

iv. Exposed person with know postiive response to vaccine series needs no vaccine or HBIG.

b. Exposure source with unknown HBsAg unknown:

i. For previously unvaccinated survivors, administer HBV immunization as soon as possible after exposure and complete age-appriorate series..

ii. Exposed person with uncompleted vaccine series should complete the series.

iii. Exposed person with written documentation of completed serivices needs no furthr treatment.

5. HPV:

a. For previously unvaccinated or vaccine series incomplete survivors, the Centers for Disease Control and Prevention (CDC) recommends initial dose at time of exam with follow-up doses at 1 to 2 and 6 months after the first dose for males and females aged 9 to 26. A two-dose schedule is recommended for persons starting the series under age 15 years.

6. HIV:

a. CDC recommends evaluation of risk status to determine need for postexposure prophylaxis (PEP).

b. Due to the complexity of clinical management, collaboration with specialists is advised.

c. Call the National Clinician's PEPline (1-888-448-4911) for assistance with PEP-related decisions.

7. Syphilis: CDC does not recommend prophylaxis.

8. Herpes simplex virus (HSV): CDC does not recommend prophylaxis. ▶

D. STI prophylaxis considerations: Survivors have a low return rate for follow-up visits. Whenever possible, provide single-dose regimens of prophylactic treatments during time of exam.

E. STI client teaching:

1. Advise sexual abstinence x 7 days after medication completion.

2. If STI prophylaxis is declined, advise STI testing (CT, GC, trichomoniasis) 2 weeks after assault.

FOLLOW-UP

A. If lab testing was done during initial exam, return in 2 weeks for results.

B. Return for evaluation anytime if symptoms appear.

C. Additional lab testing as indicated.

D. Immunization doses as indicated.

CONSULTATION/REFERRAL/INDIVIDUAL CONSIDERATIONS

A. If an evidentiary or forensic examination is requested by survivor, call the National Sexual Assault Hotline at 1-800-656-4673 (HOPE) for locations of the nearest sexual assault providers.

B. Mandatory reporting requirements vary from state to state. A listing of individual state requirements can be found at www.rainn.org/public-policy-action.

C. All states require reporting if the sexual violence involved children (ages 17 and younger) or elderly (ages 60+).

D. To report sexual violence, call 911 or local enforcement. Information on individual state statutes on reporting can be found at www.rainn.org/public-policy-action.

E. It is essential survivors know support with trained professionals is available. Provide resources to all survivors during initial encounter.

RESOURCES

1-800-656-4673 (HOPE) National Sexual Assault Hotline.
1-800-879-1999 Crisis and Support Line.
Rape, Abuse, and Incest National Network: https://www.rainn.org
Darkness to Light: https://www.d2l.org
1 in 6: https://1in6.org

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VULVOVAGINAL CANDIDIASIS

DEFINITION

A. Candidiasis (also known as moniliasis) is a common, yeast-like fungal infection of the vulva and vagina. In 90% of the cases, the cause is *Candida albicans* infection. Vulvovaginal Candidiasis (VVC) is classified as uncomplicated or complicated.

1. Uncomplicated VVC: if it is sporadic or infrequent, and mild to moderate in severity, likely to be *C. albicans*, and is in a non-immunocompromised client.

2. Complicated Vulvovaginal Candidiasis (VVC): recurrent or severe, or non-albicans candidiasis, or presenting in a client with diabetes, immunocompromising conditions, has underlying immunodeficiency or is on immunosuppressive therapy.

INCIDENCE

A. Approximately 75% of all females have at least one episode of candidiasis. It is estimated that 50% of these individuals have recurrences. Yeast has been identified with circumcised males, but symptomatic complaints are more common with uncircumcised males.

PATHOGENESIS

A. Multiple fungal species cause candidiasis, including *C. albicans* (90%), *C. tropicalis, Torulopsis glabrata* (10%), *C. parapsilosis*, and *C. krusei.*

B. *C. albicans, C. tropicalis*, and *T. glabrata* are part of the normal flora of the mouth, gastrointestinal (GI) tract, and vagina. They may become pathogenic with changes in the vaginal pH that encourage the overgrowth of the fungus.

C. The incubation period is 96 hours.

PREDISPOSING FACTORS

A. Diabetes.
B. Systemic antibiotic use.
C. Pregnancy.
D. Oral contraceptive pill use.
E. Obesity.
F. Warm climate.
G. Immunocompromised.
H. HIV.
I. Wearing tight, restrictive clothing.
J. Corticosteroid use.
K. Tub bathing.
L. Frequent use of hot tubs or whirlpools.

COMMON COMPLAINTS

A. Thick white "cheesy" vaginal discharge.
B. Itching, mild to intense vulvar pruritus.
C. Vaginal or vulvar irritation, red and swollen.
D. Discomfort during and after sexual intercourse.

OTHER SIGNS AND SYMPTOMS

A. Vulvar excoriation.
B. Vaginal swelling or inflammation.
C. Burning with urination.
D. Burning with or during intercourse.
E. Increased symptoms near menses.

SUBJECTIVE DATA

A. Determine onset, course, and duration of symptoms; note if infection is first occurrence, recurrent, persistent, or chronic.

B. Obtain medication history; include antibiotics, steroids, and birth control pills.

C. Review the client's medical history, and review systems for evidence of diabetes, HIV, or any immunocompromise.

D. Review hobbies that include the use of hot tubs, whirlpools, frequent swimming, or tight exercise clothing.

E. Review the client's history of wearing polyester underwear, wearing underwear to bed, or wearing tight jeans.

F. Review previous treatment, self-treatment measures, and compliance with previous treatments.

G. Determine if the client is pregnant; note first day of last menstrual period (LMP). ▶

H. Review sexual activity and partners. Do the partner(s) have any of the same symptoms, "jock itch," or oral candidiasis?
I. Review the use of vaginal deodorants or spray, scented toilet paper, tampons, pads, and douching.
J. Has there been any change in soaps, laundry detergent, or fabric softeners?
K. Review diet for high sugar content.

PHYSICAL EXAMINATION

A. Check temperature, pulse, and blood pressure (BP).
B. Inspect:
1. Inspect the vulva for inflammation, fissures, lesions, excoriation, rashes, and condyloma.
2. Examine the hair (Hart's) line and skin folds for inflammation, irritation, or skin breakdown.
3. Note skin changes that suggest secondary bacterial infection (erythema, drainage). **Inflammation that spares the skin folds is consistent with contact irritation. Inflammation that is within the skin folds suggests *Candida*.**
C. Palpate:
1. Perform external exam for enlarged or tender inguinal lymph nodes, vulvar masses, and lesions.
D. Pelvic examination:
1. Inspect: Observe side walls of vagina. Note amount, smell, and color of the discharge. **Typical discharge with *Candida* is adherent to vaginal side walls and characteristically thick, white, and curd-like (resembles cottage cheese). Side walls may exhibit erythema. The discharge has a musty odor.**
2. Speculum examination: Inspect the cervix for discharge and friability.
3. Bimanual examination: Check for cervical motion tenderness (CMT). Palpate for the size of the uterus and for adnexal masses or tenderness.

DIAGNOSTIC TESTS

A. Wet prep with 10% potassium hydroxide and normal saline prep. **Yeast hyphae and/or spores are determined by microscopic examination of vaginal discharge prepared with 10% potassium hydroxide or normal saline. A positive whiff test indicates bacterial vaginosis (BV).**
B. Test discharge with nitrazine paper. **The pH with candidiasis remains in the normal range of less than 4.5.**
C. Consider 2-hour glucose testing.
D. Consider testing for gonorrhea (GC) and *Chlamydia trachomatis* (CT).
E. Herpes culture, if lesions present.
F. Urinalysis and culture, if indicated.

DIFFERENTIAL DIAGNOSES

A. VVC.
B. Vulvar dystrophy.
C. Allergic vulvitis.
D. BV.
E. Urinary tract infection (UTI).
F. CT.
G. GC.
H. *Trichomonas.*
I. Herpes simplex virus (HSV) type 2.
J. Chemical vaginitis.
K. Normal physiologic discharge.

PLAN

A. General interventions:
1. Although vaginal candidiasis is treated using over-the-counter (OTC) products, encourage clients with initial presenting symptoms to have an evaluation to rule out other vaginal infections prior to self-treatment.
2. Consider treating partners. **While candidiasis is not considered an sexually transmitted infection (STI), it can be sexually transmitted. The partner should be treated in cases of recurrent infections, even if the partner is asymptomatic.**
3. For recurrent infections, consider fasting and 2-hour postprandial glucose tests for chronic yeast infections.
4. Consider testing for HIV for chronic yeast infections.
5. Vaginal culture or polymerase chain reaction should be obtained for clients with complicated VVC to confirm diagnosis and identify non-*albicans Candida*.
B. Client teaching: *See Client Teaching Guide for this chapter, "Vaginal Yeast Infection."*
1. Clients should be encouraged to present for evaluation if, after appropriate therapy has been instituted, they continue to have symptoms.
2. Treatment should continue even during menstruation.
3. Recommend avoiding wearing tight clothing that will restrict airflow to the vulvar area.
4. Recommend cotton-crotched underwear only, and preferably white.
5. Advise changing out of damp exercise clothing and swimwear as soon as possible after activities are finished.
6. Recommend not wearing underwear during sleep.
7. Use only mild soap (like Dove, Ivory) for washing the vulvar area.
C. Pharmaceutical therapy for uncomplicated VVC:
1. OTC vaginal antifungal creams: Mild cases may respond to 1 to 3 days of therapy; severe cases may require 10 to 14 days.
a. Clotrimazole 1% cream 5 g intravaginally daily for 7 to 14 days.
b. Clotrimazole 2% cream 5 g intravaginally daily for 3 days.
c. Miconazole 2% cream 5 g intravaginally daily for 7 days.
d. Miconazole 4% cream 5 g intravaginally daily for 3 days.
e. Miconazole 100 mg vaginal suppository one suppository intravaginally daily for 7 days.
f. Miconazole 200 mg vaginal suppository one suppository intravaginally daily for 3 days.
g. Miconazole 1,200 mg vaginal suppository one suppository intravaginally daily for 1 day.
h. Tioconazole 6.5% ointment 5 g intravaginally in a single application 100 mg.
2. Prescription vaginal antifungal creams:
a. Butoconazole 2% cream 5 g intravaginally in a single application.
b. Terconazole 0.4% cream 5 g intravaginally daily for 7 days.
c. Terconazole 0.8% cream 5 g intravaginally daily for 3 days.
d. Terconazole 80 mg vaginal suppository daily for 3 days.
3. Oral antifungal agents:
a. Fluconazole (Diflucan) **150 mg** orally once. If treatment is not successful, the prescription may be refilled one time; if it is still unsuccessful, consider culture

of the vagina, as a resistant, or uncommon, strain of *Candida* may be present (e.g., a non-*albicans* strain). Conventional pharmacologic therapies are not as effective against non-*albicans* species.

FOLLOW-UP

A. The client who presents with recurrent candidiasis (defined as three or more symptomatic episodes within 1 year) should be evaluated for HIV and/or other immunocompromised etiologies as well as diabetes mellitus.

B. If fasting and 2-hour blood glucose testing is normal, other options for recurrent candidiasis include treatment with clotrimazole, one applicator every other week, for 2 months. If the client remains symptom-free, reduce treatment to once each month, in the week before their menstrual period.

C. In case of recurrent vulvovaginal candidiasis, some recommend longer initial therapy (7–14 days of topical therapy or a 100-, 150-, or 200-mg oral dose of fluconazole every third day for a total of 3 doses [days 1, 4, and 7]) to initiate an infection-free period before initiating a maintenance regimen. Oral fluconazole (in either 100-, 150-, or 200-mg dosages) once weekly for 6 months is the first-line maintenance regimen. Topical treatments, used intermittently, are an alternative to the weekly fluconazole regimen. While suppression therapies show efficacy in reducing the number of recurrences, a significant percentage of females will have a recurrence after maintenance therapy ceases. Symptomatic females who continue to have positive cultures, despite being on maintenance therapy, should be managed in consultation with a specialist.

CONSULTATION/REFERRAL

A. Consult or refer the client to a physician if there is no response to the previous treatments and/or in presence of concurrent systemic disease.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Candidiasis may lead to an increase in VVC due to the increased glycogen content of the vagina and to the stimulatory effects of estrogen and progesterone on candidal growth. OTC antifungal creams are appropriate for use in this population if there is no rupture of membranes. Candidiasis may be transmitted from the infected mother to the newborn at delivery.

2. Uncomplicated VVC is not usually acquired through intercourse and does not require treatment. If the male partner exhibits symptoms of balanitis, it is appropriate to treat with topical antifungals.

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CLIENT TEACHING GUIDE

AMENORRHEA

PROBLEM For some reason that we do not fully understand, you have stopped ovulating, or putting out an egg each month, and you have stopped having periods. This is a very common problem.

CAUSE Although the cause is usually unknown, amenorrhea is often associated with low thyroid activity; excessive exercise, such as that of an athlete or dancer; or excessive weight loss; or it may be a sign of early menopause.

PREVENTION There is no specific prevention.

TREATMENT PLAN **Activity:** If you have fewer or stop having periods, you may decrease the intensity of exercise. Take at least 2 days off each week, and decrease the amount of time during each exercise session.

Sexual activity: Although you may not be having periods, you may have a return of fertility without warning. If pregnancy is undesired, be sure to use an effective birth control prevention method to prevent unintended pregnancy.

Diet: If you have not had a period and have lost a lot of weight, increasing calories will help to start your periods again.

Medications: You and your healthcare provider should discuss use of progesterone to replace what your ovaries are not making.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:

A. You have any new symptoms.
B. You have problems taking your medication.
C. You think you might be pregnant.
D. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

BACTERIAL VAGINOSIS

PROBLEM You have been diagnosed with a vaginal infection, also known as bacterial vaginosis (BV). This is a very common problem that has a "fishy vaginal discharge." The odor increases after sexual intercourse, but it is not considered a sexually transmitted infection (STI). Recurrence is common, and your partner may also need to be treated.

YOU CAN BE TREATED IN PREGNANCY During pregnancy, BV has been associated with premature rupture of the membranes and preterm labor.

CAUSE BV is caused by an alteration in the normal flora of the vagina. There are many contributing pathogens and factors, including the routine use of douches, antibiotic use, menses, and pregnancy. Females do not get BV from having sex with males. However, when a female client has been diagnosed with BV, they are more likely to catch other types of infections from their partner. Females who have sex with other females are likely to pass along a vaginal infection to a sexual partner.

PREVENTION

A. Wear cotton panties or panties with a cotton crotch.
B. Do not wear tight, restrictive clothes, such as tight jeans.
C. Leave your underwear off during sleep.
D. Limit tub bathing and the use of hot tubs or whirlpools.
E. Avoid the use of bubble bath, feminine deodorant sprays, and perfumed sanitary products (sanitary pads, tampons, and toilet paper).
F. Practice good hygiene:
  1. Wipe with toilet tissue from front to back after urinating and bowel movements.
  2. Wipe from front to back using clean towels with each bath or shower.
  3. Change your tampons and pads often during your period. Do not use a tampon while using vaginal creams because it will absorb the medication.
G. Routine douching destroys the normal vaginal flora. Avoid douching unless you are prescribed a medicated douche.
H. Practice safe sex and use condoms to protect against STIs.

TREATMENT PLAN

A. You may be given a prescription for pills or vaginal creams.
B. Clindamycin is an oil-based, medicated cream used to treat BV and will weaken latex condoms for at least 72 hours after stopping the therapy.
C. All treatments (medications and douches) may be used during your period.
D. Metronidazole (Flagyl) oral tablets may be prescribed. The side effects include a sharp, unpleasant metallic taste in the mouth, furry tongue, and some urinary tract symptoms. Please remind your provider if you have a history of seizures or if you are on any blood-thinning drug. Alcohol should not be ingested while taking the antibiotic metronidazole because it could cause serious side effects.

OTHER METHODS OF TREATMENT

A. Vinegar and water douches: one tablespoon of white vinegar in one pint of water. Douche one to two times a week.
B. *Lactobacillus acidophilus* culture four to six tablets daily.
C. Garlic suppositories: Place one peeled clove of garlic wrapped in a cloth dipped in olive oil into your vagina overnight, and change daily for five nights. You will not smell like garlic.
D. Male partners do not need to be treated with antibiotics for BV.
E. Female partners should be tested/treated for BV.

(*continued*)

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CLIENT TEACHING GUIDE

BACTERIAL VAGINOSIS (*CONTINUED*)

Activity: As tolerated.

Diet: When taking the antibiotic metronidazole (Flagyl), you must **avoid alcohol during the entire week you are taking the medicine and 24 hours after your last dose.** The combination of the medicine and alcohol may cause nausea, vomiting, stomach upset, and a headache.

Medication: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:

A. You vomited your medication (Flagyl).
B. Your vaginal odor and discharge are not relieved after the medications.
C. You continue to have repeated infections after following the instructions.
D. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

CERVICITIS

PROBLEM You are being treated for cervicitis. The cervix is the lower section of the uterus that opens into the vagina. Cervicitis is a condition or inflammation of the cervix. You may not be aware or have any symptoms of having this problem.

CAUSE Certain germs, such as *Chlamydia trachomatis* or *Neisseria gonorrhoeae*, may cause cervicitis; however, in many cases, no specific germ may be identified. In these cases, inflammation may be caused by douching, chemical irritants, or altered vaginal flora. In many cases, no cause may be found.

Your healthcare provider will perform a physical evaluation, including pelvic examination, and obtain certain tests to diagnose the cause of cervicitis. If a sexually transmitted organism is found, you and your partner will need to be treated.

PREVENTION In cases where cervicitis is caused by sexually transmitted organisms, use of condoms may prevent infection. Do not douche or use any other chemically irritating products.

TREATMENT PLAN

A. You may be prescribed an antibiotic by your healthcare provider.
B. Depending on the cause of cervicitis, your sexual partner(s) may also need medical evaluation and treatment.

Activity: Avoid sexual activity until treatment is completed.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You need to finish all of your antibiotics even though you may feel better.

You Need to Notify the Office If:
A. You are not able to take your medicine.
B. You have other new symptoms.
C. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

CONTRACEPTION: HOW TO TAKE BIRTH CONTROL PILLS (FOR A 28-DAY CYCLE)

You have been prescribed an oral contraceptive, also known as a birth control pill. Most birth control pills contain a combination of synthetic estrogen and progestin.

A. Birth control pills suppress ovulation.

B. They make the lining of the uterus unreceptive for an egg to implant and grow. Birth control pills also alter the cervical mucus, making it thicker and harder for sperm to penetrate.

C. A birth control pill does not prevent any sexually transmitted infection (STI) or HIV. A condom must still be used to protect yourself from the HIV virus or other infections.

D. You will be asked to return to the office in 3 months after starting birth control pills to check your blood pressure(BP) and for other side effects of the pill, such as your potassium level and nausea.

E. If your BP is normal and you are not having any other problems taking the pills, your prescription for birth control pills may be written for 1 year.

F. At the end of that time, you will need another physical examination and possibly a Pap smear. Then your prescription can be refilled for another year.

You Have Been Prescribed: _____

A. This is a combination pill of estrogen and progestin:

1. Your packet contains 28 pills. Notice that your pills are of different colors. **You must take them in the order that they come in the packet**. There are 21 "active" pills, and the last seven are inactive or "sugar" pills to keep you in the habit of taking a pill every day.

2. You must take a pill **every day** at approximately the same time. Develop the habit of taking the pill with brushing your teeth, for example. **You cannot share your birth control pills with anyone else.**

3. Start your packet on the Sunday of your period. Take the pill marked "1," "start here," or "Sunday."

4. You take a pill every day for 21 days; when you start the last seven pills, you will have a period or "withdrawal bleed."

5. Your period may not start for 1 to 2 days into the last week of pills. This is normal. You generally have a shorter, lighter period on birth control pills.

6. When you start your period, it is time to refill your prescription for your next month of pills.

7. If this is your first packet of birth control pills, you are not considered protected and may get pregnant. Use a backup method of birth control for the first packet of pills.

8. Missed pills instructions:

a. If you miss one pill, take it as soon as you remember, then get back on your regular schedule (you take two pills in 1 day).

b. If you miss two pills, take two pills as soon as you remember, then get back on schedule (you take three pills in 1 day). You must use a **backup method of birth control**, such as a condom, until you finish that packet of pills. You may have spotting if you miss two pills. This is normal.

c. If you miss three pills, you may have a period. Discard that packet of pills and start a new packet on Sunday. You must use a **backup method of birth control** such as a condom for the first 7 days of the new packet.

d. If one or more birth control pills are missed, no backup method of contraception is used, and if you miss your period you should do a pregnancy test.

9. If you are prescribed antibiotics while taking birth control pills, you must use a **backup method of birth control** such as a condom. You can get pregnant. Antibiotics and other medications such as those used to prevent seizures make birth control pills less effective, making it possible to get pregnant.

You Need to Notify the Office If You:

A. Vomit your birth control pills.

B. Have a severe or migraine-like headache.

C. Are depressed (cannot make yourself happy).

D. Have pain in your legs, especially if your calf hurts when walking or flexing your foot.

E. Break your leg and require a cast.

(*continued*)

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CLIENT TEACHING GUIDE

CONTRACEPTION: HOW TO TAKE BIRTH CONTROL PILLS (FOR A 28-DAY CYCLE) (*CONTINUED*)

F. Think you are pregnant (skipped pills or are taking antibiotics).
G. Have blurred vision, loss of vision, or spots before your eyes.
H. Feel chest pain or shortness of breath.
I. Feel severe abdominal pain.
J. Have lots of swelling of the fingers, hands, ankles, or face.
K. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

DYSMENORRHEA (PAINFUL MENSTRUAL CRAMPS OR PERIODS)

PROBLEM Painful menstrual cramps, or dysmenorrhea, can cause occasional diarrhea, nausea or vomiting, and headache with periods.

CAUSE A substance called prostaglandin causes most painful menstrual cramps. This substance is made in the uterus and causes the uterus to contract. Most menstrual cramps are normal and are not a sign of anything wrong. However, menstrual cramps may cause you to feel badly enough that you are unable to go to school or work. If that is the case, your healthcare provider can suggest medication to decrease the painful periods.

TREATMENT PLAN/CARE

A. Your healthcare provider may suggest a medicine such as a prostaglandin inhibitor. This medication helps decrease or eliminate the most likely substance that is causing the cramping of your uterus. The medications most often suggested are ibuprofen (Motrin, Advil) and naproxen (Aleve). Generic options for either of these are acceptable:

1. These are available at your local drug store, grocery store, or convenience store.
2. Take any of these with a snack or meal to protect your stomach lining and prevent nausea.
3. If you usually have very painful menstrual cramps, begin your medication as soon as your period begins or even the day before your period.

B. If your cramps are not better using over-the-counter (OTC) medications, your healthcare provider may write a prescription for a stronger medicine.

C. Many females take birth control pills to relieve menstrual cramps. A prescription is necessary.

D. Some famales find that exercise such as walking helps ease the cramps.

E. Another idea is a warm bath, shower, or a warm heating pad on your abdomen.

F. General health practices, such as regular exercise, yoga, routine sleep habits, and regular sexual activity, are beneficial.

Activity: Try to continue your usual activity. Try taking a walk, swimming, or doing yoga. Try a warm bath or shower.

Diet: Eat your normal diet. If you are nauseated, drink a clear carbonated soda (7UP or Sprite).

Medication: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Any questions concerning your condition.

B. Problems taking the medicine.

C. No relief when taking the medicine, and other measures do not help.

D. Any signs of infection, such as fever, chills, bad-smelling vaginal discharge, or burning sensation when you urinate.

Phone: _____

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DYSPAREUNIA (PAIN WITH INTERCOURSE)

PROBLEM As many as 60% of females complain of pain with sexual intercourse, also known as dyspareunia. Pain may occur with insertion of and/or with deep penetration of the penis into the vagina.

CAUSES There are physical causes, such as episiotomy scars; a short vagina, infections, and endometriosis; musculoskeletal causes, such as disc problems; vaginal dryness caused by hormonal causes such as the lack of estrogen in menopause; and/or not being aroused before sex, lack of foreplay, and poor communication with partners. Fear may also be a cause of pain; having a prior painful experience of having sex may interfere with being able to relax during intercourse.

PREVENTION AND TREATMENT PLAN

A. Inadequate lubrication:
  1. More prolonged foreplay increases natural vaginal lubrication.
  2. Use a vaginal lubricant such as Replens, K-Y Jelly, Silk-E, or Astroglide prior to intercourse. Crisco oil is also a hydrogenated vegetable oil that is solid at room temperature and can be used effectively by applying this to the posterior vaginal area prior to intercourse to help prevent pain. Place a small amount of Crisco oil in a decorative jar next to the bedside for use.
  3. Use a moisturizer gel with lactic acid on a regular basis to decrease vaginal dryness and pain with intercourse. Luvena is a vaginal moisturizer and lubricant that can help to improve symptoms.
  4. Do not use Vaseline as a lubricant.
  5. Do not use contraceptive creams for lubrication; they often cause dryness (dehydration) and may worsen soreness.

B. Pain on insertion of penis:
  1. Try different positions that give you more control.
  2. Guide the penis for insertion.
  3. If menopausal, you may be prescribed estrogen cream to use on an intermittent basis.

C. Pain with deep penetration:
  1. Use a side-lying position during intercourse; this may be more comfortable so that deep penetration is limited.
  2. You may need to be referred to a gynecologist for further treatment and/or surgery if you have any masses or scar tissue noted on a physical examination.

D. If you have or suspect an infection:
  1. Inform your provider.
  2. A culture will be done.
  3. Antibiotics will be prescribed for you and possibly your partner(s).
  4. Refrain from sexual intercourse until all medications are gone (unless otherwise instructed).

E. If you have a very narrow vaginal opening, you may need to be evaluated for use of vaginal dilators.

F. Spasm of the muscles upon touching the vaginal area may be treated with medication, relaxation techniques, and Kegel exercises.

G. You and your partner may be referred to a sex counselor.

Medication: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. No relief of your symptoms after your prescribed treatment.

B. Other: _____

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EMERGENCY CONTRACEPTION PILLS

The morning-after pill can be used as emergency contraception (EC) after unprotected sex or your birth control failed. There are two medications used for EC treatment: ella (ulipristal acetate) OR using levonorgestrel birth control pills a different way.

ELLA Ella (ulipristal acetate) is one pill to use for EC. You must have a prescription. There are several places that ella is available, including family planning clinics, community health centers, and campus health clinics. In some states including Alaska, California, Hawaii, Maine, Massachusetts, New Hampshire, New Mexico, Vermont, and Washington, your local pharmacist can give you a prescription for ella.

Ella is also available by ordering it over the Internet. The website is PRJKT RUBY™ (https://www.prjktruby.com); you must complete a free online medical consultation and the medicine will be shipped to you.

Ella is not meant to replace regular methods of birth control.

EC does not protect you against HIV or other sexually transmitted infections.

A condom must still be used to protect yourself from the HIV virus or other infections.

EC in the form of hormonal pills will not ***interrupt an already established pregnancy.***

TREATMENT PLAN FOR ELLA

A. Ella has to be prescribed by a healthcare provider, including pharmacists in some states, or ordered online after a medical consultation.

B. Your provider will tell you full details about how to take ella and what to expect:

1. Ella should be taken as soon as possible after unprotected sex.
2. But ella can be taken up to 5 days (up to 120 hours) after unprotected sex.
3. Ella works just as well on the fifth day as it does if you take it on the first day.

C. The most common side effects include headache, stomach pain, nausea, painful period, tiredness, and dizziness.

D. If you have lower abdominal pain after taking ella, you need to go to the clinic to see if you are pregnant outside of your uterus; this is called a tubal or ectopic pregnancy.

LEVONORGESTREL-BASED MORNING-AFTER PILLS AVAILABLE OVER THE COUNTER Levonogestrel morning-after pills are available over the counter (OTC) without a prescription in most drugstores and pharmacies and include: Plan B One Step, Next Choice One Dose, Take Action, My Way, and AfterPill. Anybody can buy them OTC at drugstores, no matter your age or gender.

Levonorgestrel morning-after pills are not meant to replace regular methods of birth control.

Levonorgestrel morning-after pills do not protect you against HIV or other sexually transmitted infections.

A condom must still be used to protect yourself from the HIV virus or other infections.

EC in the form of hormonal pills will not ***interrupt an already established pregnancy.***

Read and follow all the instructions.

TREATMENT PLAN FOR OTC LEVONORGESTREL PILLS

A. Take these pills as soon as possible after unprotected sex.

B. These morning-after pills work best if taken within 3 days (72 hours) after unprotected sex.

C. They can be taken up to 5 days after unprotected sex (the sooner the better).

D. The most common side effects include breast tenderness, headache, or dizziness. These side effects go away in a day or two.

E. Do not take more than one dose of either type of morning-after pill; it will not give you any extra protection from pregnancy and can make you sick.

F. Do not take a morning-after pill like Plan B with ella because they may counteract with each other and not work at all.

G. If you have lower abdominal pain after taking OTC EC, you need to go to the clinic to see if you are pregnant outside of your uterus; this is called a tubal or ectopic pregnancy.

Ella is not meant to replace regular methods of

(*continued*)

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EMERGENCY CONTRACEPTION PILLS (*CONTINUED*)

LEVONORGESTREL-BASED EC USING BIRTH CONTROL PILLS ***Levonogestrel pills for EC are not meant to replace regular methods of birth control.***

Levonogestrel pills do not protect you against HIV or other sexually transmitted infections.

A condom must still be used to protect yourself from the HIV virus or other infections.

EC in the form of hormonal pills will not interrupt an already established pregnancy.

A. You will be given a prescription or a supply of birth control pills.
B. You must start levonorgestrel-based EC within 72 hours of unprotected sex.
C. It is best to start within the first 12 to 24 hours.
D. The sooner you begin EC, the more effective it will be.
E. The number of pills depends on the brand of the pill.
F. Your healthcare provider will give you clear instructions.
G. The birth control pills prevent pregnancy because the hormones cause the mucus in the cervix to thicken and the lining of the uterus and tubes to change.
H. You may not ovulate, but if you do ovulate the egg will not be ready to be fertilized by a sperm.
I. Because you are taking more female hormones than you are used to, you may become sick to your stomach.
J. Your healthcare provider will tell you what medication to buy or give you a prescription for medicine to keep you from being sick to your stomach.
K. Take this medicine at least 1 hour before you take the hormone pills.
L. Other common side effects include breast tenderness, headache, or dizziness. These side effects go away in a day or two.
M. You should have a period a week or so after you take the pills.
N. If you have not had a period by 3 weeks, call the office.
O. It is unlikely that you would get pregnant, but if you do and choose to have a baby, the EC is *not* associated with any increased chance of birth defects.

Medication: _____

You Have Been Prescribed the Following EC Medication: _____

You Have Been Prescribed the Following Nausea Medicine: _____

You need to call us if you have any questions or problems.

You Need to Notify the Office If You Have:
A. Serious side effects of medicine.
B. Severe chest pain.
C. Severe abdominal pain.
D. Headache.
E. Vision changes.
F. Shortness of breath.

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FIBROCYSTIC BREAST CHANGES AND BREAST PAIN

PROBLEM You are being treated for breast pain or breast lumpiness that is from changes in the breast tissue that are not cancerous, but do cause pain. You likely have had an extensive physical examination by your healthcare provider and may have had diagnostic imaging, perhaps with a mammogram, ultrasound, and/or a breast biopsy.

CAUSE The cause is unknown, but probably related to hormonal fluctuations that occur with menstrual periods.

PREVENTION None known.

TREATMENT PLAN Being fitted for a well-fitting bra is important to eliminate breast movement/stress as a source of pain. Try ice packs on your breasts for 20 minutes every few hours. Some females find that heat on the breast can also relieve discomfort.

Activity: There are no activity restrictions. When exercising, wear a well-fitted, supportive bra.

Diet: Eliminate or decrease salt in your diet to decrease water retention if you have swelling of your breasts near your period. Some females have reported decreased breast pain with reduced caffeine and nicotine intake.

Medications: Several medications have been found to relieve breast pain from fibrocystic breast changes. These include medications that decrease and/or stabilize estrogen, such as hormonal contraceptive options. Ask your healthcare provider which is best for you.

Complementary: Recent research has shown that flaxseed may reduce cyclic pain (flaxseed 25 mg/d). Additionally, iodine (1.5–6 mg daily) may be effective in reducing cyclic mastalgia and tenderness associated with fibrocystic changes.

You Have Been Prescribed: _____

You Need to Take: _____

Phone: _____

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GENITOURINARY SYNDROME (ATROPHIC VAGINITIS)

PROBLEM You have been diagnosed with genitourinary syndrome, sometimes called atrophic vaginitis. This means that the lining of your vagina is thinner, less pliable, and less lubricated. This condition leaves the lining more prone to tears and abrasions. This is a natural part of aging, and it is also very common with teens as they begin menstruating and with breastfeeding female.

CAUSE Genitourinary syndrome is caused by an alteration in estrogen either from premature ovarian failure, delayed puberty, breastfeeding, or naturally occurring menopause.

PREVENTION/CARE

A. This is a physical problem, *not* an emotional problem.
  1. If you are a preadolescent girl, the symptoms will decrease as puberty approaches.
  2. If you are breastfeeding, your symptoms will resolve after weaning the baby.
  3. If you are menopausal, or have premature ovarian failure, your symptoms will improve after starting a hormone replacement pill or using a hormonal vaginal cream.

B. You also can help your symptoms by doing the following:
  1. Avoid perfumed hygiene sprays, talc, and deodorant soaps in the perineal area.
  2. Wear cotton-crotched underwear, preferably white (free of dye).
  3. Sleep without wearing underwear.
  4. Use a water-soluble, nonglycerin-containing vaginal lubricant with sexual intercourse, such as Good Clean Love or Slippery Stuff. **Do not use Vaseline.** Vaseline can contribute to infections.
  5. Regular sexual activity/masturbation facilitates the natural production of lubricating secretions of your body.
  6. Kegel exercise (using the muscles that start and stop the flow of the urine stream) improves the muscle tone and elasticity of the vagina.
  7. The female-superior "on top" or side-lying position for sexual intercourse gives you the ability to control the depth of thrusting with the penis, and this may make sex more comfortable.
  8. Plain yogurt douches or acidophilus tablets by mouth, or inserted into your vagina, can help maintain the vaginal pH to prevent vaginitis,

TREATMENT PLAN

A. Vitamin E oil may be used for vaginal dryness. Do not use vitamin E with latex condoms as this can cause disintegration of the condom.

B. Use water-soluble, nonglycerin-containing vaginal lubricants for intercourse.

C. You may be prescribed an estrogen cream or hormone replacements for menopause.

D. If you still have your uterus, hormones need to be balanced with estrogen and progesterone to prevent the lining of the uterus from overgrowing. Follow your hormone therapy instructions.

E. You still need regular cervical cancer screening (Pap and human papillomavirus test if indicated by age) even if you do not have a period.

Activity: Increase foreplay for increased lubrication. Try the previous suggestions on sexual positions for greater comfort and control.

Diet: As tolerated.

Medication: _____

You Have Been Prescribed: _____

You Need to Take It/Use It: _____

(*continued*)

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GENITOURINARY SYNDROME (ATROPHIC VAGINITIS) (*CONTINUED*)

You Need to Notify the Office If You Have:

A. No relief of symptoms after following the instructions above.
B. No relief of symptoms after beginning the hormonal replacements.
C. Vaginal bleeding after intercourse.
D. A change in your symptoms.
E. Other: _____

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MENOPAUSE

PROBLEM Menopause is the cessation of menses (stopping of menstrual periods) for 12 consecutive months and is generally experienced in females between 45 and 55 years of age; however, some females may be earlier or later:

A. Menopause before the age of 40 is considered premature.

B. Induced menopause is the abrupt cessation of menses related to chemical or surgical interventions.

Perimenopause is the time preceding menopause and may last several years. The average age of onset in females is usually in their 40s, but may occur earlier. Symptoms may occur during this time period due to fluctuations in hormone levels. **Pregnancy may still occur during this time and reliable contraception should be used.**

CAUSE Menopause is a normal function of aging and can occur naturally or induced by surgical or chemical intervention. Symptoms vary from being absent, very mild or severe. Some symptoms include hot flashes, night sweats, insomnia, vaginal dryness and /or pain with interccourse.

TREATMENT

A. Plan of care: Your care provider will work with you to develop a plan of treatment that is based on your individual symptom pattern. Inform your care provider if you have:

1. Acute liver disease.
2. Cerebral vascular or coronary artery disease, myocardial infarction (MI), or stroke.
3. History of or active thrombophlebitis or thromboembolic disorders.
4. History of uterine or ovarian cancer.
5. Known or suspected cancer of the breast.
6. Known or suspected estrogen-dependent neoplasm.
7. Pregnancy.
8. Undiagnosed, abnormal vaginal bleeding.

B. Activity:

1. Regular physical exercise can be beneficial for weight reduction and symptom control.
2. Dress in layers to accommodate hot flashes and avoid warm areas.
3. Avoid hot showers and baths.
4. Regular sexual intercourse is encouraged, and you may use vaginal lubricants (K-Y Jelly, Astroglide, Replens), which are helpful for vaginal dryness.

C. Over-the-counter (OTC) products for vaginal dryness/pain with intercourse:

1. More prolonged foreplay increases natural vaginal lubrication.
2. Use a vaginal lubricant such as Replens, K-Y Jelly, Silk-E, or Astroglide prior to intercourse. Crisco oil is also a hydrogenated vegetable oil that is solid at room temperature and can be used effectively by applying to the posterior vaginal area prior to intercourse to help prevent pain. Place a small amount of Crisco oil in a decorative jar next to the bedside for use.
3. Use a moisturizer gel with lactic acid on a regular basis to decrease vaginal dryness and pain with intercourse. Luvena is a vaginal moisturizer and lubricant that can help to improve symptoms.
4. Do not use Vaseline as a lubricant.
5. Do not use contraceptive creams for lubrication; they often cause dryness (dehydration) and may worsen soreness.

D. Be sure to use a reliable method of **birth control** to prevent undesired pregnancy if you have had a period within 1 year.

Diet: You need to eat a well-balanced diet (three meals a day). Supplement your diet to achieve calcium 1,200 mg/d and vitamin D 800 IU/d. Avoid alcohol, caffeine, and spicy food as they may trigger hot flashes.

Medication: _____

You Have Been Prescribed: _____

You Need to Take: _____

(*continued*)

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MENOPAUSE (*CONTINUED*)

You Need to Notify the Office If:

A. You experience unexpected vaginal bleeding.

B. Your symptoms worsen.

C. You are on hormone replacement therapy and you experience calf pain, chest pain, or shortness of breath; cough up blood; or have severe headaches, visual disturbances, breast pain, abdominal pain, or yellowing of the skin.

D. Other: _____

Phone: _____

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PELVIC INFLAMMATORY DISEASE

PROBLEM You have been diagnosed as having pelvic inflammatory disease (PID). This inflammation can involve the uterus, tubes, ovaries, and all areas in your pelvis.

CAUSE Organisms that go up from the vagina and cervix into the uterus cause PID. The two most common with PID are *Chlamydia trachomatis and Neisseria gonorrhoeae.*

PREVENTION

A. Condoms and a spermicidal foam or cream with nonoxynol-9 are protective against PID.
B. Condoms must be used with *every* sexual intercourse.
C. Vaginal douching may lead to an increased risk for PID; routine douching is *not recommended*.
D. The more sex partners you have, the greater the chances are of contracting sexually transmitted infections.

TREATMENT

A. Your partner(s) need(s) to be evaluated and treated with antibiotic therapy, too.
B. Sexual abstinence:
1. You should not have sex until all of your symptoms are gone and your partner(s) has (have) completed antibiotic therapy.
2. If you do have sex, you should use condoms consistently.

Activity: Limit yourself to bedrest for about 3 to 4 days, then you can be active as tolerated.

Diet:
A. You need to drink at least 10 to 12 glasses of liquid every day.
B. You need to eat a well-balanced diet (three meals).
C. If you have been prescribed Flagyl (metronidazole), you must avoid all alcohol ingestion for at least 2 days after the last dose or you will have severe nausea and vomiting.

Medications:
A. You will be prescribed *two or more* antibiotics; it is extremely important to **take all of the antibiotics**.
B. You may take acetaminophen (Tylenol) for fever.
C. You may be prescribed some pain medication.

You Have Been Prescribed: _____

You Need to Take: _____

Take all of your antibiotics, even if you feel better.

Your Second Prescribed Antibiotic Is: _____

You Need to Take: _____

You need to take all of both antibiotics, even if you feel better.

You Need to Notify the Office If:
A. Your fever does not get better after taking Tylenol.
B. Your symptoms worsen, even while taking both antibiotics.
C. You vomit or cannot tolerate your antibiotics.
D. You must return to the office 2 or 3 days after the antibiotics have been started for a repeat examination, or if you are unable to return for your follow-up office visit.
E. Other: _____

Phone: _____

Next Appointment: Date _____ **Time** _____

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PREMENSTRUAL SYNDROME AND PREMENSTRUAL DYSPHORIC DISORDER

PROBLEM Premenstrual syndrome (PMS) and premenstrual dysphoric disorder (PMDD) are common problems experienced by females in their reproductive years. You may have some or all of the following symptoms:

A. Cravings for food, particularly chocolate and salty foods.
B. Irritability.
C. Feelings of depression; crying spells.
D. Bloated stomach.
E. Weight gain and water retention.
F. Difficulty concentrating.
G. Tiredness.
H. Feelings of faintness.
I. Occasional clumsiness.
J. Sore breasts.

CAUSE Although the cause is really not known, PMS is a response of your body to the changes in female hormones during the last half of your menstrual cycle.

PREVENTION All of your symptoms probably cannot be prevented, but some of them may be made less severe.

TREATMENT PLAN

A. Keep track of your symptoms for at least 3 months so that your healthcare provider can determine if the symptoms always happen in the last half of your cycle.
B. Eat six small meals each day. Eat breakfast, have a morning snack like fruit or a glass of milk, eat lunch, have an afternoon snack, eat supper, and then have another evening snack. This helps keep your blood sugar even, to avoid low blood sugar.
C. Avoid candy, desserts, and other sugars. They may be associated with episodes of low blood sugar. Complex carbohydrates such as pasta, potatoes, fresh fruit, rice, and bread break down more slowly than sweets and keep your blood sugar steadier.
D. Stay away from salty foods such as chips, fast food, and pickles.
E. Avoid caffeine in soda, coffee, and chocolate. Caffeine makes you irritable and nervous.
F. Exercise daily. It is a good idea to do aerobic exercises or even walk. Exercise increases chemicals in your brain that help with your mood.
G. Join a PMS group so that you can get support from other females who have similar symptoms. You may get ideas of how other females handle PMS, and you can share your ideas, too.
H. If you smoke, try to cut down or quit.
I. Get a good night's rest and take naps during the day if possible.
J. Try stress reduction classes or yoga. Local community organizations usually have classes available.

Medications: There are a number of medications available that your healthcare provider may suggest for you.

You Have Been Prescribed: _____

You Need to Take: _____

You Have Also Been Prescribed: _____

You Need to Take: _____

You Need to Call the Office If:

A. You have questions or concerns.
B. You feel that things are not improving.
C. Other: _____

Phone: _____

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VAGINAL YEAST INFECTION

PROBLEM You have been diagnosed with a vaginal yeast infection. This is an infection or inflammation of the vagina that is caused by a fungus known as yeast *(Monilia* or *Candida albicans)*. Symptoms include vaginal itching with burning, soreness, and irritation. This may also cause related symptoms such as pain and burning with urination. Other symptoms may also include pain with intercourse.

CAUSE Yeast cells *(Monilia)* are normally present on the skin in healthy people. An overgrowth of these cells results in a yeast infection. These cells may be found in the vagina or rectal area. However, due to a disturbance in the body's hormones and pH, an overproduction of these cells occurs and causes an infection. Several factors can cause this disturbance, including menstrual periods, pregnancy, diabetes, antibiotics or other medications, increased dietary intake of sugars and alcohol, and an increase in moisture and warmth in the vaginal or rectal area by wearing tight, restrictive clothing.

PREVENTION

A. Keep the vaginal and rectal areas clean and dry.
B. Shower daily and avoid tub baths.
C. Avoid tight, restrictive clothing such as tight jeans and underwear.
D. Wear cotton panties that allow air to circulate. At bedtime, do not wear underwear with your pajamas.
E. Obesity can contribute to this problem, too. If you have gained an excessive amount of weight, try to lose these extra pounds.
F. Avoid douching because it changes the normal flora and pH of the vagina, which can contribute to causing yeast infections.

TREATMENT PLAN A pelvic exam may have been necessary to identify the source of your infection. Practice preventive tips to speed your recovery.

Activity: Avoid excessive exercise and activities that produce excessive sweating; also avoid sexual intercourse until your infection is gone. Your partner may also need to be treated for this same infection.

Diet: Drink plenty of water and other liquids. Avoid alcohol and excessive sugars. Increase the intake of yogurt and buttermilk in your diet.

Medications: Antifungal medications may be prescribed for you:
A. Over-the-counter (OTC) medications may include Monistat vaginal suppositories and cream. This is also known as miconazole nitrate, which you may find in the drugstore at a much lower price and which can be just as effective.
B. You must use the full dosage of the OTC medication. If you stop too early, the yeast can regrow.

If you have also been diagnosed with a bacterial infection of the vagina, other medications may also be prescribed. If your provider has prescribed Flagyl (metronidazole), **please do not drink any alcohol while taking this medication and for the next 3 days following this medication. The combination of this medication and alcohol can make you very sick**.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. OTC medications do not help your symptoms.
B. You develop other symptoms.
C. Other: _____

Phone: _____

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CHAPTER 18

SEXUALLY TRANSMITTED INFECTIONS GUIDELINES

Dana N. Hughes

CHLAMYDIA

DEFINITION

A. Chlamydia is a sexually transmitted infection (STI) caused by the *Chlamydia trachomatis* bacterium.

INCIDENCE

A. *Chlamydia trachomatis* is the most common sexually transmitted organism in the United States. In 2019, the Centers for Disease Control and Prevention (CDC) reported 1,808,703 cases of chlamydia, which is approximately 553 cases per 100,000 population, an increase of 19% over 2015. Health department notification is required.

B. Testing and screening recommendations:

1. Males:

a. The CDC notes that although chlamydia screening programs have demonstrated a reduction rate in pelvic inflammatory disease (PID), there is insufficient evidence to recommend routine screening in sexually active males due to feasibility, efficacy, and cost-effectiveness.

b. Consider screening in clinical settings such as correctional facilities, or in populations of high burden of infection, such as men having sex with men (MSM).

2. Females:

a. The primary focus of chlamydia screening should be to detect chlamydia, to prevent complications such as PID, and to test and treat partners:

i. Annual screening of all sexually active females aged less than 25 years is recommended.

ii. Screen older females (>25 years) at increased risk for infection.

1) New sex partner.
2) More than one sex partner.
3) Sex partner with concurrent partners.
4) Sex partner who has an STI.

3. Routine oropharyngeal screening for chlamydia is not recommended.

PATHOGENESIS

A. *C. trachomatis* is an intracellular bacterium with parasitic properties. It is transmitted by sexual contact or perinatally when a vaginal delivery occurs through an infected birth canal.

PREDISPOSING FACTORS

A. History of STIs.
B. Multiple sexual partners.
C. Early age at first coitus.
D. Unprotected intercourse.
E. Inconsistent condom use.
F. Gay.
G. Bisexual.
H. MSM.
I. Factors contributing to increase of STIs in older adults:

1. Undereducated: Older adults are less likely to perceive themselves at risk. Safe sex and STI prevention education came in the 1980s dealing with the HIV crisis and during the time older adults were middle-aged and married. Seniors may feel sex education is only directed to youth and prevention of pregnancy.

2. Medications for erectile dysfunction have contributed to more males being able to engage in sexual activity throughout their older years.

3. Online dating lowers the chance that partners know the background and sexual history of people they date.

4. Those who are postmenopausal and do not worry about getting pregnant with new partners

COMMON COMPLAINTS

A. Up to 80% of those infected are asymptomatic.
B. Mucopurulent cervical, vaginal, or urethral discharge.
C. Dysuria.
D. Urinary frequency and urgency.
E. Pelvic pain (dull or severe).

OTHER SIGNS AND SYMPTOMS

A. Cervical friability.
B. Cervical motion tenderness.
C. Uterine and/or adnexal tenderness.
D. Rectal discharge.
E. Diarrhea.

SUBJECTIVE DATA

A. Elicit history of onset of symptoms, location, frequency, duration, aggravating and alleviating factors, and associated symptomatology.

B. Question the client about sexual partners, sexual habits, and history of other STIs.

The contributions of Cheryl A. Glass and Leslie B. Norman to this chapter in prior editions are acknowledged here.

PHYSICAL EXAMINATION

A. Inspect:
- **1.** Males: Observe for anal and/or urethral discharge.
- **2.** Female pelvic exam: Observe for anal and/or vaginal discharge.
- **3.** Female speculum exam: Inspect vaginal wall and cervix for discharge and irritation.

B. Palpate:
- **1.** Males:
  - a. Palpate inguinal lymph nodes.
  - b. Milk the penis for discharge.
  - c. Palpate the groin.
- **2.** Females:
  - a. Palpate the inguinal lymph nodes.
  - b. Bimanual exam:
    - i. Milk urethra.
    - ii. Palpate periurethral and Bartholin glands for exudate.
    - iii. Assess for cervical motion tenderness.
    - iv. Assess for uterine and adnexal tenderness.

DIAGNOSTIC TESTS

A. It is important to obtain a thorough sexual history from the client and to screen all orifices used during sexual contact (e.g., oropharynx, rectum).

B. Nucleic acid amplification tests (NAATs) are preferred to culture for diagnosis of *C. trachomatis*. The Food and Drug Administration (FDA) has approved NAATs for both male and female urogenital specimens collected via clean-catch. urine. It is important to obtain a thorough sexual history from the client and to screen all orifices used during sexual contact (vagina, oropharynx, rectum).
- **1.** Rectal and oropharyngeal testing for persons engaging in receptive anal or oral intercourse can be diagnosed by testing at the anatomic site of exposure:
  - a. Brush or swab the oropharynx.
  - b. Insert the brush or swab 1 to 2 cm into the endocervix, urethra (males only), or rectum.
  - c. Rotate for 30 seconds, withdraw, and place in appropriate culture media for transport.

C. NAAT is the recommended and most sensitive test.
- **1.** NAATs are FDA-cleared for use with vaginal swab specimens collected by a provider or self-collected in a clinical setting. Self-collected vaginal swab specimens are equivalent in sensitivity and specificity to collection by providers.
- **2.** Optimal urogenital specimen types for chlamydia screening using NAAT include:
  - a. Males: first catch urine.
  - b. Females: vaginal swabs.
- **3.** NAATs are not FDA-cleared for use with rectal or oropharyngeal swab specimens.
- **4.** Some NAATs have been FDA-cleared for use on liquid-based cytology specimens.

DIFFERENTIAL DIAGNOSES

A. Chlamydia.
B. Gonorrhea.
C. Urethritis.
D. *Mycoplasma genitalium.*

PLAN

A. General interventions:
- **1.** Culture samples immediately for timely treatment. Consider testing for other STIs such as gonorrhea, syphilis, HIV, and trichomoniasis.

B. Client teaching: *See Client Teaching Guide for this chapter, "Chlamydia."*
- **1.** Inform the client of the need for partner notification and treatment. Notification is recommended for any partner with whom the client has had sexual contact as soon as possible but within 30 days of the onset of symptoms or 60 days if asymptomatic.
- **2.** Stress the importance of completing treatment regimen.
- **3.** Advise the client to avoid sexual intercourse after single-dose therapy or until treatment completion and for 7 more days following the last day of antibiotic treatment and resolution of symptoms, if present.
- **4.** Repeat infections place females at an elevated risk for PID and other complications.
- **5.** Persons who have chlamydia and HIV infection should receive the same treatment regimen as those who do not have HIV infection.

C. Pharmaceutical therapy:
- **1.** The (2021) CDC recommends the following regimens:
  - a. Doxycycline 100 mg PO BID, for 7 days is the preferred treatment. If adherence to medication is of great concern, then azithromycin 1 g PO in a single dose.
- **2.** Alternative regimen:
  - a. Levofloxacin 500 mg orally once daily for 7 days.

FOLLOW-UP

A. The CDC recommends all individuals treated for chlamydia be retested in 3 months.

CONSULTATION/REFERRAL

A. Consult or refer the client to a specalist when treatment with the recommended dosage fails if client noncompliance and reexposure have been ruled out.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
- **1.** Azithromycin 1 g is recommended in pregnancy as doxycycline and levofloxacin are contraindicated.
- **2.** All pregnant individuals diagnosed with chlamydial infection should be retested in 4 weeks following treatment.
- **3.** For individuals younger than 25 years of age, those with new partners, and/or those at a high risk for infection, repeat chlamydial testing during the third trimester.
- **4.** All pregnant individuals diagnosed with chlamydial infection during the first trimester should be retested at 4 weeks following treatment and again in 3 months after treatment.
- **5.** The CDC recommends the following regimens for pregnant individuals:
  - a. Azithromycin 1 g orally in a single dose.

B. Adults:
- **1.** Untreated or long-standing chlamydial infection in females may lead to infertility.

C. Geriatrics:
- **1.** Medicare offers free STI screening and treatment for seniors.
- **2.** Clinicians need to address the STI issue by including questions about sexual activity in their senior client assessment.

3. Seniors are at increased risk for STIs due to a weaker immune system and hormone changes.
4. Seniors with cognitive impairment and lower functioning may be vulnerable and at risk for STI.

D. Partners:
1. The CDC and state laws recommend good faith effort from providers in notifying partners regarding positive results for STIs and providing partners with a prescription for treatment of STIs without being seen. Review state public health laws.

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GONORRHEA

DEFINITION

A. Gonorrhea is a sexually transmitted infection (STI) caused by the *Neisseria gonorrhoeae* organism.

INCIDENCE

A. Gonorrhea is a Centers for Disease Control and Prevention (CDC)-reportable infection. In 2017, there were 616,392 cases reported to the CDC in the United States, which is 188 cases per 100,000 population, an increase of 56% since 2016. The rate of increase in infections was higher among males (60.6%) than among females (43.6%). This reflects either increased transmission or increased case ascertainment among men have sex with men (MSM).

B. Testing and screening recommendations:
1. Screening for gonorrhea in males and older females who are at low risk for infection is not recommended.
2. Annual screening is recommended in older clients who have a new sex partner, more than one sex partner, or a partner who has other partners or an STI.
3. Males:
   a. Screen at least annually for sexually active men having sex with men (MSM regardless of condom use.
   b. Every 3 to 6 months is indicated for MSM at increased risk:
      i. Concurrent HIV infection if risk behaviors persist.
      ii. If they or their sexual partners have multiple partners.
4. Females:
   a. The primary focus of a screening should be to detect gonorrhea, to prevent complications such as pelvic inflammatory disease (PID), and to test and treat partners:
      i. Annual screening of all sexually active females aged less than 25 years is recommended.
      ii. Screen older females (>25 years) at increased risk for infection:
         1) New sex partner.
         2) More than one sex partner.
         3) Sex partner with concurrent partners.
         4) Sex partner who has an STI.

PATHOGENESIS

A. *N. gonorrhoeae*, a gram-negative diplococcus, is the causative organism. The infection begins with adherence of *N. gonorrhoeae* to the mucosal cells in the genitourinary tract or endocervix. The incubation period is typically 2 to 5 days for urethritis and 5 to 10 days for cervical infection.

B. Gonorrhea treatment is complicated by the ability of *N. gonorrhoeae* to develop resistance to antimicrobials.

PREDISPOSING FACTORS

A. History of STIs.
B. New partner or one who is not monogamous.
C. Trading sex for drugs or money.
D. Multiple sexual partners.
E. Early age at first coitus:
1. Unprotected intercourse.
2. Inconsistent condom use.
3. Gay.
4. Bisexual.
5. MSM (high risk for gonorrhea).
6. Factors contributing to increase of STIs in older adults:
   a. Undereducated: Older adults are less likely to perceive themselves at risk. Safe sex and STI prevention education came in the 1980s dealing with the HIV crisis during the time older adults were middle-aged and married. Seniors may feel sex education is directed only to youth and prevention of pregnancy.
   b. Medications for erectile dysfunction have contributed to more males being able to engage in sexual activity throughout their older years.
   c. Online dating lowers the chance that partners know the background and sexual history of people they date.
   d. Those who are postmenopausal and do not worry about getting pregnant with new partners.

COMMON COMPLAINTS

A. Dysuria.
B. Yellow, white, or mucoid urethral discharge in males.
C. Greenish, irritating vaginal discharge in females.
D. Menstrual irregularities.
E. Pelvic pain.
F. Fever.
G. Rectal discharge.

OTHER SIGNS AND SYMPTOMS

A. Asymptomatic, including rectal and pharyngeal.
B. Uterine or adnexal tenderness.
C. Mucopurulent discharge from endocervix.
D. Polyarthralgias.
E. Necrotic skin lesions.

SUBJECTIVE DATA

A. Elicit history of onset, duration, and location of symptoms. Note aggravating and alleviating factors and associated symptomatology.

B. Question the client about sexual partners (male, female, or both), history of other STIs, and sexual habits including anal and oral sexual exposure.

C. The CDC recommends a recent travel history with sexual contacts outside of the United States should be part of any gonorrhea evaluation.

PHYSICAL EXAMINATION

A. Check temperature (if indicated). ▶

B. Inspect:
- **1.** Inspect the skin for necrotic skin lesions.
- **2.** Oral examination.
- **3.** Males: Inspect for anal and/or urethral discharge; elicit latter by milking penis.
- **4.** Females:
  - a. Inspect anus and introitus for discharge; milk the urethra.
  - b. Speculum exam: Inspect the vaginal walls and cervix for discharge and irritation.

C. Palpate:
- **1.** Examine joints for effusion and swelling.
- **2.** Males: Palpate inguinal lymph nodes.
- **3.** Females:
  - a. Palpate inguinal lymph nodes.
  - b. Palpate periurethral and Bartholin glands for exudate.
  - c. Bimanual exam:
    - i. Assess for cervical motion, tenderness, and friability.
    - ii. Assess for uterine and adnexal tenderness.

DIAGNOSTIC TESTS

A. It is important to obtain a thorough sexual history from the client and to screen all orifices used during sexual contact (e.g., oropharynx, rectum).

B. NAAT is the FDA)-approved culture for diagnosis of *N. gonorrhoeae* via urine sample or samples of body fluid where infection is suspected. The FDA has approved NAAT tests for both male and female urogenital specimens collected via clean-catch urine.

C. Nucleic acid amplification test (NAAT) allows for the widest variety of FDA-cleared specimen types, including endocervical swabs, vaginal swabs, urethral swabs (males), and urine (both males and females):
- **1.** NAAT is not FDA-cleared for rectal, oropharyngeal, and conjunctival gonococcal infection.
- **2.** NAATs are FDA-cleared for use with vaginal swab specimens collected by a provider or self-collected in a clinical setting. Self-collected vaginal swab specimens are equivalent in sensitivity and specificity to collection by providers.
  - a. Opti mal urogenital specim en types for gonorrhea screening using NAAT include:
    - i. Males: first catch urine.
    - ii. Females: vaginal swabs:
      - 1) NAATs are not FDA-cleared for use with rectal or oropharyngeal swab specimens.
      - 2) Some NAATs have been FDA-cleared for use on liquid-based cytology specimens.

D. For persons diagnosed with gonorrhea, testing should also be performed for chlamydia, syphilis, and HIV.

E. In cases of suspected or documented treatment failure, both cultures and antimicrobial susceptibility testing are needed because nonculture tests cannot provide antimicrobial susceptibility results.

F. During septic joint stage, gonococci can be recovered from the joint by aspiration for culture.

DIFFERENTIAL DIAGNOSES

A. Gonorrhea.
B. Chlamydia.
C. Arthritis (rheumatoid or septic).
D. Nongonococcal urethritis (NGU).
E. *Mycoplasma genitalium.*

PLAN

A. General interventions:
- **1. Gonorrhea treatment is complicated by the ability of *N. gonorrhoeae* to develop resistance to antimicrobials:**
  - a. **In 2007, emergence of fluoroquinolone-resistant *N. gonorrhoeae* in the United States prompted the CDC to cease recommending fluoroquinolones for treatment of gonorrhea, leaving cephalosporins as the only remaining class of antimicrobials available for treatment of gonorrhea in the United States.**
  - b. **The CDC no longer recommends the routine use of cefixime as a first-line regimen for treatment of gonorrhea in the United States.**
  - c. **In addition, U.S. gonococcal stains with elevated minimal inhibitory concentration (MIC) to cefixime are also likely to be resistant to tetracyclines but susceptible to azithromycin.**
- **2.** All persons who receive a diagnosis of gonorrhea should be tested for other STIs, including chlamydia, syphilis, and HIV.
- **3.** Report positive test results to the health department.

B. Client teaching: *See Client Teaching Guide for this chapter, "Gonorrhea."*
- **1.** Stress the importance of completing the medication regimen.
- **2.** Inform the client of the need for partner notification and treatment. Notification is recommended for any partner with whom the client has had sexual contact within 60 days of the onset of symptoms.
- **3.** Advise the client to avoid sexual intercourse after single-dose therapy or until treatment completion and for 7 more days following the last day of antibiotic treatment and resolution of symptoms, if present.
- **4.** Repeat infections place females at an elevated risk for PID and other complications.
- **5.** Persons who have gonorrhea and HIV infection should receive the same treatment regimen as those who are HIV-negative.

C. Pharmaceutical therapy:
- **1. Uncomplicated gonococcal infections of the cervix, urethra, and rectum:**
  - a. Ceftriaxone 500 mg by intramuscular (IM) injection if <150 kg or 1 g if >150 kg. If chlamydia has not been ruled out, doxycycline 100 mg BID for 7 days should be added to the treatment. For pregnancy, zithomax 1 g single dose is recomemended.
  - b. It is preferable that the dual therapy of ceftriaxone and doxycycline should be administered/started on the same day.
  - c. If ceftriaxone is not available, the CDC recommends gentamicin 240 mg IM single dose plus azithromycin 2 g dose orally. OR
  - d. Cefixime 800 mg orally in a single dose.

FOLLOW-UP

A. The CDC no longer recommends a test of cure except for oropharyngeal infection; however, clients who continue to exhibit symptoms should have culture performed.

B. All individuals should be closely monitored for treatment failure/reinfection caused by failure of sex partners to receive treatment or initiation of sexual activity with a new infected partner.

C. Individuals who have been treated for gonorrhea should be retested 3 months after treatment regardless of whether they believe their sex partners were treated. If retesting at ▶

3 months is not possible, clinicians should retest whenever they present for medical care within 12 months following initial treatment.

D. Providers are advised to report all treatment failures to the local or state public health department within 24 hours.

CONSULTATION/REFERRAL

A. Consult with a specialist or refer the client if treatment with the recommended dosage fails and client noncompliance and reexposure have been ruled out.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Transmission during vaginal birth is possible and may result in conjunctivitis and blindness in neonates.

2. Treatment during pregnancy should be with ceftriaxone 500 mg in a single IM dose and azithromycin 1 gm orally in a single dose.

3. Pregnant clients who test positive for gonorrhea in the first trimester and have been treated should undergo screening again in 3 months.

B. Adults:

1. Untreated or long-standing gonorrheal infection in females can lead to infertility.

2. For clients with allergies to cephalosporins, using gentamicin plus azithromycin treatment is recommended.

C. Geriatrics:

1. Medicare offers free STI screening and treatment for seniors.

2. Clinicians need to address the STI issue by including questions about sexual activity in their senior client assessment.

3. Seniors are at increased risk for STIs due to a weaker immune system and hormone changes.

D. Partners:

1. The CDC and state laws recommend good faith effort from providers in notifying partners regarding positive results for STIs and providing partners with a prescription for treatment of STIs without being seen. Review state pubic health laws.

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HERPES SIMPLEX VIRUS: GENITAL HERPES

DEFINITION

A. Herpes simplex is a chronic lifelong viral infection that is transmitted by direct contact with the secretions or mucosa of an infected individual who is shedding the virus. The virus is usually characterized by painful vesicular lesions that form an ulcer, crust over, then dry and resolve (without scarring) within 2 to 3 weeks. There are two types of herpes virus. Herpes simplex virus type 1 (HSV-1) is oral herpes and type 2 (HSV-2) is genital herpes. HSV-1 can result in cold sores or fever blisters around the mouth. Oral herpes can spread from the mouth to the genitals through oral sex and cause genital outbreaks. Genital ulcerative disease is also transmitted through vaginal sex and anal sex with a partner that has the disease. Many infected individuals are not aware of their status and can transmit the virus when asymptomatic.

B. Herpes infection can cause sores or breaks in the skin or lining of the mouth, vagina, and rectum, providing a way for exposure to the HIV virus. A person infected with both HIV and genital herpes increases the chance that HIV will spread to an HIV-uninfected sexual partner during contact with their mouth, vagina, or rectum.

C. The Centers for Disease Control and Prevention (CDC) does not recommend screening for HSV-1 or HSV-2 in the general population.

D. Herpes is not a notifiable disease to the state or local health departments. Public health surveillance for herpes infections is mainly done through population-based, national surveys, such as the National Health and Nutrition Examination Survey (NHANES).

E. Both laboratory-based assays and point-of-care testing that provide results for HSV-2 antibodies from capillary blood or serum during a clinic visit are available.

INCIDENCE

A. It is estimated that 85% of adults have had oral sex. More than one of every six people aged 14 to 49 years in the United States have genital herpes.

B. The risk for neonatal transmission from an infected mother is 30% to 50% among those who acquire genital herpes near the time of delivery and low (<1%) among those with prenatal histories of recurrent herpes or who acquire genital HSV during the first half of pregnancy.

PATHOGENESIS

A. HSV is the causative organism. HSV-1 typically produces oral lesions, and HSV-2 most commonly causes genital lesions. However, HSV-1 can cause genital outbreaks as well as oral. HSV-2 is passed in saliva with oral herpes infection; genital secretions, vaginal delivery, and autoinoculation are all possible routes of transmission.

B. The virus remains dormant, and outbreaks can be stimulated by several factors, including stress, illness, sunlight exposure, and menstruation. A prior infection with HSV-1 can increase the incidence of an asymptomatic HSV-2 infection by threefold.

C. Failure to detect HSV by culture or polymerase chain reaction (PCR), especially in the absence of active lesions, does not indicate an absence of HSV infection because viral shedding is intermittent.

PREDISPOSING FACTORS

A. Early age at first coitus.
B. Multiple sexual partners.
C. History of sexually transmitted infections (STIs).
D. Men having sex with men (MSM).

COMMON COMPLAINTS

A. Dysuria.
B. Pruritus.
C. Burning.
D. Swelling sensation.

OTHER SIGNS AND SYMPTOMS

A. Primary episode:

1. Painful, vesicular, ulcerated, or crusted oral or genital lesion, singly or in clusters.

2. Flulike syndrome: fever, chills, headache, malaise, and myalgia. ▶

B. Recurrent episode:
1. Less painful lesions and little or no systemic symptoms.
2. Prodromal symptoms prior to an outbreak described a localized genital pain and tingling or shooting pains in the legs, hips, or buttocks that occur hours to days before the eruption of the herpetic lesions.

SUBJECTIVE DATA

A. Elicit history of onset of symptoms; their location, frequency, and duration; aggravating and alleviating factors; and associated symptomatology.
B. Question the client about the history of other STIs and sexual habits (oral, vaginal, and anal).
C. Has the client ever had cold sores?
D. Does their sexual partner have any symptoms or sores?

PHYSICAL EXAMINATION

A. Check temperature (if indicated).
B. Inspect:
1. Assess all mucus membranes including eyes, nose, mouth, throat, perineum, vagina, penis, and anogenital areas for blister-like, vesicular lesions.
C. Palpate:
1. Check the neck lymphadenopathy.
2. Assess for inguinal lymphadenopathy.

DIAGNOSTIC TESTS

A. Viral cell culture and PCR are the preferred tests, although the sensitivity of viral culture is low and declines as lesions heal. The sensitivity of viral culture is low for recurrent lesions.

Clinical diagnosis is often reliable, but confirmation with viral culture should be attempted. Vesicles should be unroofed or crust removed for the most reliable sample.

1. Viral isolation: Obtain vesicular fluid by swabbing lesion with cotton- or Dacron-tipped applicator.
2. Place applicator in appropriate transport viral medium before drying.
3. Refrigerate until ready for transport.
B. Serology: Nucleic acid amplification methods, including PCR assays for HSV DNA, are more sensitive. HSV-specific glycoprotein G2 (HSV-2) and glycoprotein G1 (HSV-1) tests vary in sensitivity and false-negative results may occur in early stages of infection so repeat testing may be necessary.

PCR is the test of choice for diagnosing HSV ocular infections and infections affecting the central nervous system and systemic infections (e.g., meningitis, encephalitis, and neonatal herpes). Many infected individuals are not aware of their status and can transmit the virus when asymptomatic. PCR can be used to detect asymptomatic viral shedding.

C. Pap smear is not a sensitive test for HSV-2.
D. Persons with genital herpes should also be tested for HIV.
E. A punch biopsy provides more reliable material for histologic examination, particularly when lesions are infected with bacteria and fungi.

DIFFERENTIAL DIAGNOSES

A. HSV.
B. Primary syphilis.
C. Chancroid.
D. Lymphogranuloma venereum.
E. Folliculitis.
F. Candidal fissure.
G. Vestibular vulvitis.
H. Mucocutaneous manifestations of Crohn disease.
I. Behcet syndrome.
J. Hand-foot-and-mouth disease (HFMD).

PLAN

A. General interventions.
1. For episodic treatment, the client should be provided with a supply of drug or a prescription for medication with instructions to initiate treatment immediately when symptoms begin.
B. Client teaching: *See Client Teaching Guide for this chapter, "Herpes Simplex Virus."*
1. Counseling of infected persons and their sex partners is critical to the management of genital herpes. The goals of counseling include helping clients cope with the infections and preventing sexual and perinatal transmission.
a. Advise clients to abstain from sexual activity during prodrome or while lesions are present.
b. Condom use should be encouraged when sexually active.
C. Pharmaceutical therapy. Systemic antiviral drugs can partially control the signs and symptoms of genital herpes when used to treat the first clinical and recurrent episodes or when used as daily suppressive therapy. Newly acquired genital herpes can cause prolonged clinical illness with severe genital ulcerations and neurologic involvement. Even persons with first-episode herpes who have mild clinical manifestations initially can develop severe or prolonged symptoms. Therefore, the CDC recommends all clients with first episodes of genital herpes receive antiviral therapy.
1. Primary first clinical episode:
a. Acyclovir 400 mg PO TID for 7 to 10 days. OR
b. Famciclovir 250 mg PO TID for 7 to 10 days. OR
c. Valacyclovir 1 g PO BID for 7 to 10 days.
2. Episodic therapy for recurrent genital herpes: Effective episodic treatment of recurrent herpes requires initiation of therapy within 1 day of lesion onset or during the prodrome that precedes some outbreaks:
a. Acyclovir 800 mg PO BID for 5 days.
b. Acyclovir 800 mg PO TID for 2 days.
c. Famciclovir 125 mg PO BID for 5 days.
d. Famciclovir 1,000 mg PO BID for 1 day.
e. Famciclovir 500 mg once, followed by 250 mg BID for 2 days.
f. Valacyclovir 500 mg PO BID for 3 days.
g. Valacyclovir 1 g PO once daily for 5 days.
3. Suppressive therapy for recurrent genital herpes:
a. Acyclovir 400 mg PO BID.
b. Famciclovir 250 mg PO BID.
c. Valacyclovir 500 mg PO once daily.
d. Valacyclovir 1 g PO once daily.
e. Periodically during suppressive treatment (e.g., once a year), providers should discuss the need to continue therapy. Neither treatment discontinuation nor laboratory monitoring in a healthy person is necessary.

EMERGENCY ISSUES/INSTRUCTIONS

A. Intravenous (IV) acyclovir therapy should be provided for clients who have severe HSV disease or complications that necessitate hospitalization (e.g., disseminated infection, ▶

pneumonitis, or hepatitis) or central nervous system (CNS) complications (e.g., meningoencephalitis).

FOLLOW-UP

A. Follow-up is not recommended unless it is warranted by clinical presentation.

CONSULTATION/REFERRAL

A. Consultation with a dermatologist may be beneficial in cases of atypical lesions.

B. Consult or refer the client to a specialist when there is prolonged ulceration unresponsive to therapy.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Acyclovir and valacyclovir are considered safe to use during pregnancy. These medications can be used for treatment as well as for suppression during pregnancy. The American College of Obstetricians and Gynecologists recommends that suppressive therapy begin at approximately 36 weeks' gestation to prevent an outbreak of lesions and to increase the chance of having a vaginal delivery (Table 18.1).

2. Culture lesions if outbreak occurs. Lesions must be crusted for 7 days for vaginal delivery to be an option; otherwise, a Cesarean delivery is recommended to avoid transmission of virus to newborn.

3. Pregnant individuals without genital herpes should be advised to avoid intercourse during the third trimester with partners known or suspected of having genital herpes.

4. Acyclovir allergy: No effective alternatives to acyclovir have been identified.

B. Partners:

1. Symptomatic partners should be evaluated and treated in the same manner as any client with genital lesions.

C. Geriatrics:

1. Recurrent infection of the buttocks region is not uncommon, especially in females.

2. Medicare offers free STI screening and treatment for seniors.

3. Clinicians need to address the STI issue by including questions about sexual activity in their senior client assessment.

4. Seniors are at increased risk for STIs due to a weaker immune system and hormone changes.

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HUMAN PAPILLOMAVIRUS

DEFINITION

A. The human papillomavirus (HPV) is a sexually transmitted infection (STI). Condylomata acuminata, genital warts, and venereal warts are other names for HPV.

INCIDENCE

A. HPV is the most common STI in the United States.

B. In 2013 to 2014, among adults ages 15 to 59 years, about 45% of males and 40% of females had genital HPV infections. About 25% of males and 20% of females had high-risk genital HPV infection. The prevalence of any type of oral HPV infection among adults ages 18 to 69 in 2011 to 2014 was about 7% and the prevalence of high-risk oral HPV infections was 4%.

C. Most sexually active persons become infected with HPV at least once in their lifetime. Condoms provide some protection against HPV transmission, but contact with genital lesions not covered by the condom can result in infection. Clients with anogenital warts may be at risk for other STIs.

D. Prevention:

1. Several HPV vaccines are available for use in the United States. Gardasil protects against two to nine strains of HPV. The vaccine protecting against nine strains addresses types 6, 11, 16, 18, 31, 33, 45, 52, and 58, which are most likely to be associated with cancer, warts, and other diseases.

E. For pediatric clients, vaccination may start at as young as 9 years of age. The vaccine is recommended at 11 to 12 years of age. Vaccination is recommended for everyone through age 26 years if not adequately vaccinated when younger. The vaccine has been approved by the Food and Drug Administration for use up to age 45, and individuals aged 27 to 45 should make a decision with their clinician based on individual risk.

F. Administration for children 9 to 14 years of age should be two doses of intramuscular (IM) vaccine, with the second dose given 6 to 12 months later. Administration for children 15 years of age or older consists of a three-dose series of IM injections. Dose 1 is given at month 0, with the second dose given 1 to 2 months after the first, and the third given 6 months after the first.

G. HPV vaccine is available for those younger than 19 years of age via the Vaccines for Children (VFC) program (call CDC-INFO [1-800-232-4636] for more information).

H. Uninsured clients eligible for the vaccine may be able to access client assistance programs via vaccine manufacturers.

TABLE 18.1 PHARMACOLOGICAL TREATMENT OF HERPES IN PREGNANCY

Antiviral	Acyclovir Dosage	Valacyclovir Dosage
Primary or first lesion outbreak	400 mg TID for 7–10 days	1 g BID for 7–10 days
Symptomatic recurrent lesions	400 mg TID for 5 days or 800 mg BID for 5 days	500 mg BID for 3 days or 1 g daily for 5 days
Daily suppression from 36-week gestation until delivery	400 mg TID	500 mg BID

BID, twice a day; TID, three times a day.

PATHOGENESIS

A. The HPV, a slow-growing DNA virus of the Papovavirus family, is the causative organism. The usual incubation period for clinical warts is 3 weeks to 8 months. Over 100 strains of the virus have been identified. At least 40 of the HPV types can infect the genital area.

B. HPV types 6 and 11 are most associated with genital warts and recurrent respiratory papillomatosis.

C. Types 16, 18, 31, 33, and 35 are high risk and are associated with cervical, penile, vulvar, vaginal, anal, and oropharyngeal neoplasia and precancers.

D. Ninety percent of anogenital warts are caused by nononcogenic HPV type 6 or 11. HPV type 16, 18, 31, and 35 are occasionally found in anogenital warts usually as coinfections with HPV 6 or 11 and can be associated with foci of high-grade squamous intraepithelial lesions (HSIL), particularly in persons who have HIV. Anogenital warts and HPV types 6 and 11 have been associated with conjunctival, nasal, oral, and laryngeal warts.

E. HPV might also be transmitted during genital-to-genital contact without penetration and oral sex.

F. Warts may appear as early as 1 to 2 months after exposure, but most infections remain subclinical. The first recognition of a lesion may represent a relapse rather than a first episode.

PREDISPOSING FACTORS

A. Early first coitus.
B. Multiple sexual partners.
C. Individual and partner's history of STIs.
D. Gay, bisexual, and other men having sex with men (MSM).
E. Factors contributing to increase of STIs in older adults:

1. Undereducated: Older adults are less likely to perceive themselves at risk. Safe sex and STI prevention education came in the 1980s dealing with the HIV crisis during the time older adults were married and middle-aged. Seniors may feel sex education is directed to youth and pregnancy prevention.

2. Medications for erectile dysfunction have contributed to more individuals being able to engage in sexual activity throughout their older years.

3. Online dating lowers the chance that partners know the background and sexual history of people they date.

4. Thsoe who are postmenopausal and do not worry about getting pregnant with new partners.

COMMON COMPLAINTS

A. Painless genital "bumps" or warts.
B. Pruritus.
C. Bleeding during or after coitus.
D. Malodorous vaginal discharge.
E. Dysuria.

OTHER SIGNS AND SYMPTOMS

A. Wart-like growths on the genital area, which are elevated and rough or flat and smooth.

B. Lesions occurring singly or in clusters, from less than 1 mm to cauliflower-like aggregates.

C. Anogenital warts are flat, papular, or pedunculated growths on the genital mucosa.

D. Papillomas that are pale pink in color.

SUBJECTIVE DATA

A. Elicit history of onset of symptoms, location, frequency, duration, and associated symptomatology.

B. Question the client about the history of other STIs, sexual behaviors, recent change in sexual partner, and partner's history of STIs.

C. Males with external condylomata acuminata may have associated involvement of the urethra or anal canal.

D. Review any previous treatments and their outcome.

PHYSICAL EXAMINATION

A. Inspect:

1. Inspect vaginal introitus. Warts can also occur at multiple sites in the anogenital epithelium or within the anogenital tract (cervix, vagina, urethra, perineum, perianal skin, anus, and scrotum).

2. Female speculum exam: Inspect vaginal walls and cervix for lesions.

3. Males: Inspect under the foreskin of the uncircumcised penis and on the shaft of the circumcised penis.

4. Oral examination.

DIAGNOSTIC TESTS

HPV tests are available to detect oncogenic types of HPV and are used in the context of cervical cancer screening and management or follow-up of abnormal cervical cytology or histology. HPV testing should not be used for male partners of females with HPV or females less than 25 years of age for diagnosis of genital warts. HPV testing is not a general STI test. HPV testing is not recommended for anogenital wart diagnosis because the test results are not confirmatory and do not guide genital wart management.

A. Visual identification is adequate in most cases.

B. The routine use of applying 3% to 5% acetic acid to detect genital mucosa changes attributed to HPV infection is not recommended because the results do not influence clinical management.

C. Cytology: Pap smears are useful for screening. Pap results of koilocytosis, dyskeratosis, keratinizing atypia, atypical inflammation, and parakeratosis are all suggestive of HPV.

D. Histology: Colposcopy with directed biopsy is diagnostic for subclinical lesions, dysplasia, and malignancy.

E. Anoscopy: Refer client to a gastroenterologist or a colorectal surgeon to examine the anal canal.

F. Indication for biopsy:

1. Client is immunocompromised, including those infected with HIV.

2. Lesions do not respond to standard therapy.

3. Worsens during therapy.

G. DNA typing: Determination of specific strains is useful in diagnosing subclinical infections (the test is costly and false-negatives occur).

DIFFERENTIAL DIAGNOSES

A. HPV.
B. Condylomata latum of syphilis.
C. Molluscum contagiosum.
D. Seborrheic keratosis.
E. Pearly penile papules.
F. Fordyce spots.
G. Lichen planus.
H. Papulosquamous lichen planus of the penis.
I. Carcinoma.

PLAN

A. General interventions:

1. Make diagnosis promptly to discuss management. If left untreated, warts may resolve spontaneously within a year, remain unchanged, or increase in size or number.

B. Client teaching: *See Client Teaching Guide for this chapter, "Human Papillomavirus."*

1. Educate about HPV vaccines. Although HPV vaccines have been found to be safe when given to people who are already infected with HPV, the vaccines do not treat infection. They provide maximum benefit if a person receives them before they are sexually active.

2. Most HPV infections are self-limited and are asymptomatic or unrecognized. Counsel clients that anogenital HPV infection is common but can infect other areas including the mouth and throat.

3. Explain to the client that therapy eliminates visible warts but does not eradicate the virus. No therapy has been shown to be effective in eradication of HPV. Ablation of warts may decrease viral load and transmissibility.

4. Advise the client to abstain from genital contact while lesions are present.

C. Pharmaceutical therapy:

1. Treatment is directed to genital warts or pathologic precancerous lesions caused by HPV and to alleviate symptoms (pruritus, bleeding, burning, tenderness, obstruction of the vagina/anus). All therapies cause localized discomfort including itching, burning, erosions, and pain because the epithelium is disrupted.

2. Therapy is not recommended for subclinical infections (absence of exophytic warts). Subclinical genital HPV typically clears spontaneously:

a. Therapy is not recommended for subclinical infections (absence of exophytic warts).

3. Client-applied:

a. Imiquimod 3.75% or 5% cream. OR
b. Podofilox 0.5% solution or gel. OR
c. Sinecatechins 15% ointment.

4. Provider-administered:

a. Cryotherapy with liquid nitrogen or cryoprobe. OR
b. Trichloroacetic acid (TCA) or bichloroacetic acid (BCA) 80% to 90% solution.

D. Medical/surgical management

1. Surgical removal by tangential scissor excision, tangential shave excision, curettage, laser, or electrosurgery.

FOLLOW-UP

A. Short-term follow-up is not recommended if the client is asymptomatic after treatment.

B. The Centers for Disease Control and Prevention (CDC) does not recommend more frequent Pap smears for females with external warts.

C. Long-term follow-up should include annualpelvic exams. The American College of Obstetricians and Gynecologists (AGOG) guidelines recommend pap smear screening based on previous pap smear results. Encourage the client to self-examine genitalia.

CONSULTATION/REFERRAL

A. Consult or refer the client to a physician when lesions persist after six consecutive treatments or when cervical or rectal warts are diagnosed.

B. Bladder obstruction is a rare complication. Clients with warts at the urethral orifice should be referred to a urologist for further evaluation.

C. Anoscopy: Refer client to a gastroenterologist or a colorectal surgeon to examine the anal canal.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Worsening of anogenital wart occurs during pregnancy.

2. There is a potential that those who are pregnant can transmit HPV to an infant during delivery.

3. TCA has no systemic absorption and no known fetal effects; therefore, it is the preferred treatment for those who are pregnant.

4. Podophyllum and podofilox are contraindicated during pregnancy.

5. There is insufficient information regarding the safety of imiquimod in pregnancy.

6. There is minimal information on the risk of use of sinecatechens during pregnancy.

B. Partners:

1. Treatment is recommended if visible lesions are present.

C. Geriatrics:

1. Verrucous carcinoma and vulvar intraepithelial neoplasia (VIN) can be indistinguishable from condyloma. Older females are more likely to have VIN or carcinoma.

2. Diagnosis made by biopsy; colposcopy is strongly advised.

3. Immunocompetence should be investigated in new or recurrent condyloma.

4. Medicare offers free STI screening and treatment for seniors.

5. Clinicians need to address the STI issue by including questions about sexual activity in their senior client assessment.

6. Seniors are at increased risk for STIs due to a weaker immune system and hormone changes.

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SYPHILIS

DEFINITION

A. Syphilis is a systemic disease (sexually transmitted infection [STI]). The disease is divided into stages based on clinical finding, helping to guide treatment and follow-up. Syphilis is characterized by distinct primary, secondary, and tertiary stages that occur over several years or decades.

B. Because latent syphilis is not transmitted sexually, the objective of treating persons in this stage of disease is to prevent complications and transmission from those who are pregnant to their fetus:

1. Latent or inactive periods occur between the stages:

a. Early latent is less than 1 year after infection.
b. Late latent is more than 1 year after infection.

C. Health department notification of infection is required by law in all states.

INCIDENCE

A. In 2019, there were 129,813 new diagnoses of syphilis in the United States, with 38,992 being primary and secondary syphilis. In 2019, men having sex with men (MSM) accounted for 41.6% of clients diagnosed with primary or secondary syphilis. There were 1,870 reported cases of children born with congenital syphilis in 2019.

PATHOGENESIS

A. *Treponema pallidum*, a spirochete bacterium, is the causative organism that infects the mucous membrane through microscopic abrasions during intercourse. The growth characteristics and metabolism of *T. palladium* are due to the inability to grow the organism in a culture.

PREDISPOSING FACTORS

A. History of STIs.
B. Multiple sexual partners.
C. Illicit drug use.
D. Trading sex for drugs or money.
E. Factors contributing to increase of STIs in older adults:
1. Undereducated: Older adults are less likely to perceive themselves at risk. Safe sex and STI prevention education came in the 1980s dealing with the HIV crisis during the time older adults were middle-aged and married. Seniors may feel sex education is only directed to youth and prevention of pregnancy.
2. Medications for erectile dysfunction have contributed to more males being able to engage in sexual activity throughout their older years.
3. Online dating lowers the chance that partners know the background and sexual history of people they date.
4. Those who are postmenopausal and do not worry about getting pregnant with new partners.

COMMON COMPLAINTS

A. Genital lesion, generalized rash involving the palms and soles, mucous patches, and condyloma latum are common.

OTHER SIGNS AND SYMPTOMS

A. Primary syphilis:
1. Chancre: painless or minimally painful.
2. Round, indurated lesion with little or no purulent exudate.
3. Regional bilateral lymphadenopathy.
4. Skin rash.
B. Secondary syphilis:
1. Generalized maculopapular rash that is nonpruritic and copper-colored, on the palms or soles; may be erythematous or scaly.
2. Mucous patches: painless, white, mucous membrane lesions.
3. Generalized lymphadenopathy: flu-like syndrome including fever, headache, sore throat, and malaise.
4. Patchy alopecia.
C. Tertiary syphilis:
1. Cardiac gumma lesions: locally destructive granulomatous tumors involving various organs or systems; commonly seen on liver but can occur on other organs (heart, brain, skin, bone, testis).
2. Cardiovascular: aortic involvement, aneurysms, and valve insufficiency.
3. Neurologic: tabes dorsalis (loss of coordination) and general paresis.
D. Latent syphilis:
1. Asymptomatic, detected by serologic testing.

Latent-phase syphilis manifests itself after treatment failure or no history of treatment. Spirochete can lie dormant for years.

E. *T. pallidum* can infect the central nervous system (CNS) and result in neurosyphilis, which can occur at any stage of syphilis:
1. Early neurologic clinical manifestations:
a. Cranial nerve dysfunction.
b. Meningitis.
c. Stroke.
d. Acute altered mental status.
e. Auditory or ophthalmic abnormalities.
2. Late neurologic clinical manifestations:
a. Tabes dorsalis and general paresis occur 10 to 30 years after infection.
F. Congenital syphilis:
1. Symptoms range from asymptomatic to fatal.

SUBJECTIVE DATA

A. Elicit history of onset, location, frequency, and duration of symptoms; aggravating and alleviating factors; and associated symptomatology.
B. Question the client about history of other STIs, illicit drug use, and sexual habits.

PHYSICAL EXAMINATION

A. Check temperature (if indicated), pulse, respirations, and blood pressure.
B. Inspect:
1. Inspect the skin; note lesions and rashes.
2. Observe the head; note patchy alopecia.
3. Ophthalmic examination.
4. Examine the mouth and throat; note lesions.
5. Inspect the perianal area, perineum, and vagina in females and underneath the foreskin in uncircumcised males to evaluate for mucosal lesions.
C. Palpate:
1. Palpate the lymph nodes (neck, supraclavicular, axillary, epitrochlear, and inguinal regions).
D. Auscultate:
1. Auscultate heart and lungs.
E. Neurologic exam:
1. Assess sensory functioning.
2. Test cranial nerves, first through twelfth.

DIAGNOSTIC TESTS

A. A presumptive diagnosis of syphilis requires use of two tests: a nontreponemal test, Venereal Disease Research Laboratory (VDRL) or rapid plasma reagin (RPR), and a treponemal test, fluorescent treponemal antibody absorption (FTA-ABS). False-positive nontreponemal test results can be associated with various medical conditions and factors unrelated to syphilis, including other infections (e.g., HIV), autoimmune conditions, immunizations, injection drug use, and older age.

Evaluate for other STIs and HIV in clients presenting with syphilis.

B. Serology: nontreponemal tests:
1. VDRL test.
2. RPR tests. The Food and Drug Administration has issued an alert for false-positive reactvitiy that using the ▶

Bio-Rad Laboratories BioPlex 2200 Syphilis Total & RPR kit can result in reactive positive for some clients who recently received a COVID-19 vaccine.

3. HIV.

Results are reactive (positive) or nonreactive (negative). Titers correlate with active disease and should be quantitative. These tests are equally valid but cannot be compared due to titer differences (RPR is slightly higher than VDRL). All reactive results require confirmation with treponemal tests.

C. Serology: treponemal tests:

1. FTA-ABS test.

2. Microhemagglutination assay for antibody to *T. pallidum.*

Once FTA-AB for antibody to T. pallidum, *VDRL, and RPR are reactive, these tests usually remain reactive for life.*

D. Cerebrospinal fluid (CSF) tests (CSF cell count or protein and a reactive CSF-VDRL). No single test can be used to diagnose neurosyphilis in all instances. The diagnosis of neurosyphilis depends on a combination of a CSF test in the presence of reactive serologic test results and neurologic signs and symptoms.

E. Microscopy: *T. pallidum* cannot be seen with light microscopy; dark-field microscopy exam of the serous exudate from lesions is the definitive test for early syphilis. Properly equipped labs with specially trained personnel must be available for this test.

DIFFERENTIAL DIAGNOSES

A. Syphilis.

B. HSV).

PLAN

A. General interventions:

1. Staging of the disease may be difficult but guides management decisions.

B. Client teaching: *See Client Teaching Guide for this chapter, "Syphilis."*

1. Advise the client to abstain from sexual activity until treatment is completed.

2. Inform the client of Jarisch–Herxheimer reaction (acute fever, headache, myalgia) that may occur within the first 24 hours of treatment. Antipyretics may be prescribed to manage symptoms, but have not been proven to prevent this reaction. The Jarisch–Herxheimer reaction occurs most frequently among persons who have early syphilis, presumably because bacteria burdens are higher during these states

3. Discuss the importance of partner notification.

4. Stress the importance of adhering to the follow-up regimen.

C. Pharmaceutical therapy.

1. Penicillin G administered parenterally is the preferred drug for treating all persons in all stages of syphilis. There are no proven alternatives to penicillin available for treating neurosyphilis and congenital syphilis in those who are pregnant. Penicillin is also recommended, whenever possible, for persons with HIV infection.

2. Primary syphilis, secondary syphilis, and early latent syphilis:

a. Adults: benzathine penicillin G 2.4 million units injected intramuscular (IM) in a single dose.

3. Late latent syphilis, latent syphilis of unknown duration, and late syphilis (manage with expert consultation):

a. Adults: benzathine penicillin G 50,000 units/kg IM up to the adult dose of 2.4 million units, administered as three doses injected IM at 1-week intervals (total 150,000 units/kg to the adult dose of 7.2 million units).

4. Tertiary syphilis (manage with expert consultation). Tertiary syphilis refers to gummas and cardiovascular syphilis but not to neurosyphilis:

a. Persons who are not allergic to penicillin and have no evidence of neurosyphilis (i.e., normal CSF examination) should be treated with benzathine penicillin G 7.2 million units total, administered at three doses of 2.4 million units IM at 1-week intervals.

5. Neurosyphilis (manage with expert consultation):

a. Refer to the Centers for Disease Control and Prevention (CDC) guidelines for treatment.

6. Refer to the CDC guidelines for recommendation on management of persons who have a history of penicillin allergy.

7. Pregnancy: Parenteral penicillin G is the only therapy with documented efficacy for syphilis during pregnancy. For pregnant clients with penicillin allergy, penicillin treatment after desensitization is recommended.

FOLLOW-UP

A. Primary and secondary syphilis: Clinical and serologic exams should be conducted at 6 and 12 months. Absence of fourfold decrease within 12 months is indicative of treatment failure.

B. Latent syphilis: Clinical and serologic exams should be conducted at 6, 12, and 24 months. Absence of fourfold decrease within 12 to 24 months is indicative of treatment failure.

C. Tertiary syphilis: Minimal evidence regarding follow-up exists. Follow-up largely depends on nature of lesions.

D. Neurosyphilis: CSF examination should take place every 6 months until cell count is normal.

CONSULTATION/REFERRAL

A. Consult or refer the client to a specialist when the recommended treatment fails and client noncompliance and reexposure have been ruled out, or when neurosyphilis is diagnosed.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Draw blood samples for RPR/VDRL from all prenatal clients.

2. Administer appropriate regimen of penicillin for the client's stage of syphilis. Consider a second dose of penicillin 1 week after the initial treatment.

3. Clients who are allergic to penicillin should be desensitized and treated with penicillin during pregnancy.

4. Follow-up: Perform serologic tests monthly until adequacy of treatment has been ensured.

5. The Jarisch–Herxheimer reaction may predispose those who are pregnant to premature labor or fetal distress if treatment occurs in the second half of the pregnancy. Advise these clients to immediately seek medical attention if they experience uterine contractions or changes in fetal movement.

6. Risk for abortions and stillbirths are increased.

B. Partners:

1. Identify at-risk partners who have had sexual contact with the client within these time frames:

a. Primary syphilis: 3 months plus duration of symptoms.
b. Secondary syphilis: 6 months plus duration of symptoms.
c. Early latent syphilis: 1 year.

C. Geriatrics.

1. Dementia, tremors, and pupillary changes are the result of long-term untreated syphilis.

2. The CSF should be tested using the FTA-ABS test. The VDRL is usually not adequate.

3. Medicare offers free STI screening and treatment for seniors.

4. Clinicians need to address the STI issue by including questions about sexual activity in their senior client assessment.

5. Seniors are at increased risk for STIs due to a weaker immune system and hormone changes.

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TRICHOMONIASIS

DEFINITION

A. Trichomoniasis is a nonviral sexually transmitted infection (STI) caused by the *Trichomonas vaginalis* protozoan. Nonsexual transmission by means of fomites is possible but rare.

INCIDENCE

A. Trichomoniasis is the most common curable STI.

B. Trichomoniasis is not a Centers for Disease Control and Prevention (CDC)-reportable infection. Therefore, exact incidence is unavailable. The CDC estimates the prevalence as 2.6 million people ages 14 to 49 years old.

C. The infection is more common in females than in males. Older females are more likely than younger females to have been infected with trichomoniasis.

1. The most common infected body part in females is the lower genital tract (vulva, vagina, cervix, or urethra).

2. Among females infected with HIV, *T. vaginalis* is associated with increased risk of pelvic inflammatory disease (PID). Up to 53% of females with HIV are also infected with trichomoniasis. Treatment of trichomoniasis is associated with significant decreases in genital tract HIV viral load and viral shedding.

3. In males, the most common infected body part is in the urethra.

4. The prevalence of trichomoniasis in men having sex with men (MSM) is low.

D. Although untreated infections may last for months or even years, it is transmitted easily during penile-vaginal sex.

E. Data are insufficient to recommend routine screening, alternative treatment regimens of longer duration, or retesting in males.

PATHOGENESIS

A. *T. vaginalis*, a flagellated protozoan, is the causative organism.

PREDISPOSING FACTORS

A. History of STIs.

B. Multiple sexual partners.

COMMON COMPLAINTS

A. Copious, yellow-green, or watery gray vaginal discharge.

B. Vaginal odor, "fishy smell."

C. Dysuria.

D. Dyspareunia.

E. Postcoital spotting or bleeding.

F. Abdominal discomfort.

G. Pruritus.

H. Some infected males have symptoms of urethritis, epididymitis or prostatitis, and penile discharge.

OTHER SIGNS AND SYMPTOMS

A. Asymptomatic (only about 30% develop any symptoms of trichomoniasis).

B. Perineal irritation.

SUBJECTIVE DATA

A. Elicit history of onset, location, frequency, duration of symptoms, and aggravating and alleviating factors; note associated symptomatology.

B. Question the client about history of other STIs or vaginal infections, changes and characteristics of vaginal discharge, and recent change in sexual partner.

PHYSICAL EXAMINATION

A. Check temperature (if indicated).

B. Inspect:

1. Inspect introitus for discharge and irritation.

C. Speculum exam:

1. Inspect the vaginal walls for discharge and irritation.

2. Inspect the cervix for discharge, erythema, punctate hemorrhages (strawberry-patch cervix), and friability.

D. Bimanual exam:

1. Assess for cervical motion tenderness.

2. Palpate for uterine and adnexal tenderness.

DIAGNOSTIC TESTS

A. The testing algorithm is wet mount first followed by nucleic acid amplification test (NAAT) if negative.

B. Wet prep: presence of motile, flagellated trichomonads. Increased number of white blood cells (>0.10 per high-power field) may be present. This is the most common method due to convenience and is relatively low-cost, although sensitivity is low (51%–65%) and lower from males (urethral specimens, urine sediment, and semen). See Section II: Procedures, "Wet Mount/Cervical Cultures."

1. Vaginal pH is usually greater than 4.5.

2. The potassium hydroxide/wet prep "whiff test" may be positive.

C. NAAT methods are the current standard and are reportedly three to five times more accurate than wet-mount microscopy. New tests using vaginal, endocervical, and urine samples are becoming more widely available.

D. OSOM Trichomonas Rapid Test has also been cleared by the Food and Drug Administration to detect *Trichomonas* in vaginal secretions and can be performed at point of care with results available in 10 minutes.

E. Culture was considered the gold standard method for diagnosing *T. vaginalis* infection before molecular detection methods became available. Culture has a sensitivity of 75% to 90%. Vaginal secretions are the preferred specimen type for ▶

culture. In males, culture specimens require a urethral swab, urine sediment, and/or semen.

F. Pap smear: Report may include trichomonads, but sensitivity is low.

1. Pap smear is not a diagnostic test for trichomoniasis because false-negatives and false-positives may occur.

G. Testing for other STIs including HIV should be performed in persons infected with *T. vaginalis*.

DIFFERENTIAL DIAGNOSES

A. Trichomoniasis.
B. Bacterial vaginosis.
C. Vulvovaginal candidiasis.
D. Chlamydia.
E. Gonorrhea.
F. PID.
G. Foreign-body vaginitis.

PLAN

A. General interventions:

1. Prompt diagnosis helps to initiate treatment.

B. Client teaching: *See Client Teaching Guide for this chpater, "Trichomoniasis."*

1. Advise the client to abstain from sexual activity until each partner has been adequately treated and symptoms have resolved.

2. Current partners should be referred for presumptive therapy to avoid reinfection.

3. Advise the client to avoid alcohol consumption during and 24 hours after metronidazole treatment due to the possible disulfiram-like reaction from the medication. Advise the client to abstain from alcohol 72 hours after completion of tinidazole.

4. Inform the client that urine may darken in color during treatment.

5. Inform the client of a possible metallic taste in the mouth during treatment.

C. Pharmaceutical therapy:

1. Drug of choice:

a. Females: metronidazole 500 mg orally BID for 7 days.

b. Males: metronidazole 2 g orally in a single dose.

D. Alternative treatment for males and females: tinidazole 2 g orally in a single dose.

1. Females with HIV should be treated with metronidazole 500 mg BID for 7 days.

2. Treatment failure: The CDC recommends retreatment with metronidazole 500 mg PO BID for 7 days.

3. If repeated failure occurs, treat with metronidazole 2 g PO QID for 3 to 5 days.

4. Metronidazole gel is unlikely to achieve therapeutic levels in the urethra or perivaginal glands. It is a considerably less efficacious treatment than oral preparations and is not recommended for use.

FOLLOW-UP

A. Immediate follow-up is not recommended if the client is initially asymptomatic or symptoms are relieved by treatment. Due to concern of resistance, a 3-month follow-up for sexually active females is recommended regardless of whether they believe their sex partners were treated.

B. Test of cure by NAAT recommended in 3 weeks.

C. In females with HIV, retesting is recommended within 3 months following initial treatment; NAAT is the test of choice.

CONSULTATION/REFERRAL

A. Consult or refer the client to a specialist when recommended treatment fails and client noncompliance and reexposure have been ruled out.

B. The CDC guidelines recommend consultation and susceptibility testing for treatment failures.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Symptomatic pregnant persons, regardless of pregnancy stage, should be tested and treated. Metronidazole 500 mg BID for 7 days is preferred.

2. Lactation: Metronidazole 500 mg BID for 7 days is preferred.

B. Partners:

1. Recommended treatment is metronidazole 2 g orally one time.

2. The CDC and state laws recommend good faith effort from providers in notifying partners regarding positive results for STIs and providing partners with a prescription for treatment of STIs without being seen. Review state public health laws.

C. Geriatrics:

1. Uncommon pathogen in those who are postmenopausal.

2. Symptoms are vulvar irritation with discharge.

3. Diagnose with wet prep.

4. Treat with metronidazole 1 g in the morning and 1 g in the evening.

5. Medicare offers free STI screening and treatment for seniors.

6. Clinicians need to address the STI issue by including questions about sexual activity in their senior client assessment.

7. Seniors are at increased risk for STIs due to a weaker immune system and hormone changes.

D. Clients with HIV:

1. Screen all females at first visit and annually for trichomoniasis. Treatment for trichomoniasis is multiple dose therapy: metronidazole 500 mg BID for 7 days.

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CLIENT TEACHING GUIDE

CHLAMYDIA

PROBLEM Chlamydia is a sexually transmitted infection (STI). Often no problems are present, but you may notice a yellowish discharge from the penis or vagina, burning during urination, frequent and urgent urination, or pelvic pain.

Untreated chlamydia in females may lead to a condition called pelvic inflammatory disease (PID). PID is a leading cause of infertility, increased ectopic pregnancies, and chronic pelvic pain.

You can get chlamydia more than once.

CAUSE Chlamydia is caused by a bacterium called *Chlamydia trachomatis*. This bacterium is spread through sexual contact and may infect the eyes, throat, vagina, penis, and rectum.

PREVENTION

A. Use condoms with sexual activity.
B. Do not have sex if you or your sexual partner has abnormal discharge or burning with urination.
C. Limit sexual partners.
D. Have routine screening tests for chlamydia prior to beginning a new sexual relationship.

TREATMENT PLAN

A. Chlamydia can be cured when you and all of your sexual partners take antibiotics.
B. Take all of your antibiotics.
C. Do not share your antibiotics with your partner(s). Each of you need to have your own prescription for antibiotics.
D. Until you and your partner(s) have completed all of your prescribed antibiotics, do not have sex.

Your healthcare provider is required to report this disease to the public health department. The health department may contact you.

Medications: Chlamydia can be cured by the prescribed antibiotics.

You Have Been Prescribed: _____

You Need to Take: _____

It is very important that you keep your follow-up appointment with your provider in 3 months so that you can be retested to make sure the infection is gone.

Your Appointment Has Been Scheduled For: _____

You Need to Notify the Office If:

A. You are unable to take your antibiotics due to nausea, vomiting, or a reaction.
B. Other: _____

Phone: _____

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GONORRHEA

PROBLEM Gonorrhea is a sexually transmitted infection (STI). You may have the following symptoms: burning during urination, yellowish discharge from the penis or vagina, heavier menstrual periods, or pelvic pain.

Untreated gonorrhea in females can lead to a condition called pelvic inflammatory disease (PID). PID is a leading cause of infertility, increased ectopic pregnancies, and chronic pelvic pain in females.

You can get gonorrhea more than once.

CAUSE Gonorrhea is caused by an organism called *Neisseria gonorrhoeae*. This organism is spread through sexual contact and may infect the eyes, throat, vagina, penis, or rectum.

PREVENTION

A. Use condoms when having intercourse.
B. Do not have sex if you or your sexual partner has abnormal discharge or burning with urination.
C. Limit sexual partners.
D. Have routine screening tests for gonorrhea prior to beginning a new sexual relationship.

TREATMENT PLAN

A. Gonorrhea can be cured when you and all of your sexual partners take antibiotics.
B. Take all of your antibiotics.
C. Do not share your antibiotics with your partner(s). Each of you need to have your own prescription of antibiotics.
D. Until you and your partner(s) have completed all of your prescribed antibiotics, do not have sex.

Your healthcare provider is required to report this disease to the public health department. The health department may contact you.

Medications: Gonorrhea can be cured by the prescribed antibiotics.

You Have Been Prescribed: _____

You Need to Take: _____

It is very important that you keep your follow-up appointment in 3 months so that you can be retested to make sure the infection is gone.

Your Appointment Has Been Scheduled for: _____

You Need to Notify the Office If:

A. You have any new symptoms.
B. You are unable to take all of the antibiotics due to nausea, vomiting, or a reaction.
C. Other: _____

Phone: _____

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HERPES SIMPLEX VIRUS

PROBLEM You may experience oral or genital bumps or blister-like lesions (often painful), burning, itching, sensation of pressure, painful urination, painful lymph nodes (bumps along underwear line), flulike symptoms (fever, headache, muscle aches, tired feeling).

Herpes simplex virus (HSV) is very common. There are two types of herpes virus. Herpes simplex virus type 1 (HSV-1) is oral herpes and type 2 (HSV-2) is genital herpes. HSV-1 can result in cold sores or fever blisters around the mouth. Oral herpes can spread from the mouth to the genitals through oral sex and cause genital outbreaks. Genital ulcerative disease is also transmitted through vaginal sex and anal sex with a partner who has the disease.

CAUSE

A. The HSV is spread by direct contact with the secretions of someone who has the virus.
B. Viruses cannot be cured, but the problems or symptoms caused by them can often be managed with medication.
C. It is possible for someone to have HSV and have no symptoms. The first outbreak after contact with an infected individual usually occurs within 2 to 10 days, but it may take up to 3 weeks.
D. More severe symptoms are experienced with the first outbreak of HSV. The symptoms usually peak 4 to 5 days after the onset of infection and resolve after 2 to 3 weeks without medication.
E. Medication may reduce the severity and duration of the symptoms. Recurrent outbreaks usually last 5 to 7 days.
F. The virus may be spread even when symptoms are not present. This is known as viral shedding. Medication may also reduce the time of viral shedding.
G. Often, individuals with HSV feel localized genital pain or tingling or shooting pains in the legs, hips, or buttocks at the site hours to days (24–48 hours) prior to an outbreak. This is known as prodrome.
H. Sexual activity should be avoided during this time of the prodrome and until any outbreak resolves completely because the viral shedding is occurring, which means the infection may be spread.

TREATMENT PLAN

A. Although it is hard to do, it is important to talk with your current/future partners about having herpes.
B. Avoid sexual activity when lesions are present or when you feel the prodrome.
C. Use condoms with sexual activity.
D. Limit sexual partners.
E. Do not use any creams, lotions, or powders on lesions unless instructed by your healthcare provider.
F. If urination is painful, pour water over the genital area while urinating.
G. Wash your hands after going to the bathroom and making any contact with the lesion.
H. Pat dry the affected area thoroughly; use a different portion of a towel and do not dry other body parts with the same towel. Do not share towels.
I. If you are pregnant at any time, notify your provider of your diagnosis of herpes to allow the provider to treat you accordingly prior to delivery to prevent spreading the herpes infection to your baby.

Activity: Triggers for an outbreak include stress, illness, and even menstruation. Exercise may help with keeping your stress level down.

Diet: There is no special diet.

Medications:
A. Antiviral medications are used to suppress the virus. They do not cure it, but reduce the frequency and severity of viral outbreaks as well as reduce the likelihood of transmission to a partner who has not been exposed to the virus.
B. The medications need to be taken the first time you have an outbreak and may be given again where other outbreaks occur, or you may choose to take medications daily to keep the virus suppressed.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Call the Office If:
A. You are unable to empty your bladder when you have an outbreak.
B. Other: _____

Phone: _____

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HUMAN PAPILLOMAVIRUS

PROBLEM Human papillomavirus (HPV), also known as condyloma, or genital or venereal warts, is a sexually transmitted infection (STI). HPV is the most common STI.

A. You may experience "bumps" or lesions on the genitals or perianal area. They may be raised and rough-appearing, or flat and smooth. They are often wart-like in appearance.

B. Lesions may appear singly or in clusters and may be small or large. They are usually soft, painless, and pale pink to grayish in color, and they may itch.

C. Some people who have the infection never have symptoms (bumps or warts). It is possible to spread the infection even when no symptoms are present.

D. HPV can infect other areas, including the mouth and throat from oral sex.

E. It is very important to get regular Pap smears. Some of the HPV viruses can cause cervical cancer.

F. There are several treatment options that your health care provider will discuss.

CAUSE HPV is acquired by having genital contact, intercourse, or oral sex with someone who has the infection.

PREVENTION/TREATMENT PLAN

A. There is no cure for HPV, but the following may reduce the spread of HPV.

1. Prevention:
   a. Ask your provider about receiving the shingles vaccine for preventing the HPV virus. It is available for males and females ranging from 9 years to 25 to 26 years of age.
   b. The vaccine does not treat the HPV infection.
2. Treatment:
   a. Up to 40% of warts go away without any treatment within 1 year.
   b. Condoms provide some protection against HPV transmission, but contact with genital lesions not covered by the condom can result in infection.
   c. Limit sexual partners and openly discuss the need to use a condom.
   d. Examine new partners for bumps or warts.
   e. Your partners also need to be seen to discuss their treatment.
   f. There are several treatments for HPV. Some of the treatments must be done in the clinic and others can be done by you at home.
   g. If you choose using a cream/gel, you will be instructed on where to apply it with your finger.

Medications: _____

You Have Been Prescribed: _____

You Need to Use It By Following These Instructions:

A. Wash your hands.

B. Use your finger to apply the medicine and rub it in.

C. Wash your hands again.

D. Apply the medicine _____ times every day on the following schedule: _____

E. Your medicine may be used daily, twice a day, or only a few days a week.

F. Your medicine will need to be used for _____weeks.

You Need to Notify the Office If:

A. You have any new symptoms or the warts are getting worse.

B. Other: _____

You Need to Return to the Office on: _____

Phone: _____

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SYPHILIS

PROBLEM You may have round or oval painless lesions, most commonly in the genital region, but they may occur anywhere on the body where transmission occurred.

A. You may experience a rash covering your body, including the palms of your hands and soles of your feet.
B. Flulike symptoms include fever; headache; sore throat; swollen, tender lymph nodes; and decreased appetite.

CAUSE Syphilis is contracted by genital or oral contact with someone who has the infection. The infection is spread when lesions are present.

PREVENTION

A. Use condoms.
B. Limit sexual partners.
C. Screen new sexual partners by asking about any known infections.

TREATMENT PLAN Do not engage in sexual activity while lesions are present. Notify all partners of the need for treatment.

Syphilis is a reportable sexually transmitted infection (STI) to the local health department. It is important that all of your partners are contacted about the need to be treated too. If antibiotic treatment is not given, over time syphilis attacks the heart, brain, liver, skin, and bones.

Keep follow-up appointments to determine if treatment has been effective.

Diet: There is no special diet.

Medications: Penicillin is the drug of choice for treating syphilis. Other antibiotics can be used if you are allergic to penicillin. Within 24 hours of antibiotic treatment, you may experience fever or headache. Aspirin, acetaminophen (Tylenol), or ibuprofen may be taken if these symptoms occur.

You Have Been Prescribed: _____

You Need to Take: _____

You need to finish all of your antibiotics.

You Need to Notify the Office If You Have the Following:

A. Any new symptoms.
B. Any reaction to your antibiotics.
C. Any other concerns about syphilis.

Phone: _____

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TRICHOMONIASIS

PROBLEM You may experience increased vaginal discharge that is yellow-green or watery gray in color. It may have a foul, "fishy" odor. You may also have vaginal itching or irritation, burning during urination, discomfort during sexual intercourse, spotting or bleeding during or after sexual intercourse, or abdominal discomfort.

Trichomoniasis is considered a sexually transmitted infection (STI).

CAUSE ***Trichomonas vaginalis*** **is acquired by having sex with someone who has the infection.**

PREVENTION

A. Use condom when having intercourse.
B. Limit the number of sexual partners.
C. Screen new sexual partners.
D. Do not have sex if your sexual partner has abnormal discharge or burning with urination.

TREATMENT PLAN

A. Do not have sexual activity until you and your partner have both completed medications. There are no limitations in other physical activity.
B. Diet:
  1. Do not drink alcohol during the use of medication and for 3 days after taking the last dose of your medicine. Alcohol use while taking this medication may result in nausea, vomiting, and severe upset stomach.
  2. You may have a metallic taste from the medicine that may slightly alter the taste of food temporarily.
  3. No other limitations in diet are required.

Medications:

There are two medications that are used to treat the trichomoniasis infection: metronidazole (Flagyl) OR tinidazole (Tindamax).

You Have Been Prescribed: _____

You Need to Take: _____

Finish all of the medications.

You Need to Notify the Office If:
A. You are unable to tolerate the medicine.
B. Any new symptoms develop.
C. Other: _____

Phone: _____

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CHAPTER 19

INFECTIOUS DISEASE GUIDELINES

LaDawna R. Goering

CYTOMEGALOVIRUS

DEFINITION

A. Cytomegalovirus (CMV) is a double-stranded DNA virus in the herpesvirus family. It has the largest genome of the herpesviruses, grows only in human cells, and replicates best in human fibroblasts. Multiple genetically distinct strains exist; the differences in CMV genotypes are associated with differences in virulence. CMV is responsible for a viral infection with a high rate of asymptomatic excretion. Shedding of the virus takes place intermittently. CMV can be isolated in cell culture from urine, a pharynx swab, respiratory secretions, human milk, tears, saliva, semen, cervical secretions, and body fluids such as blood and amniotic fluid. CMV usually causes an asymptomatic infection and remains dormant throughout life but can be reactive. CMV frequently reactivates in critically ill persons.

B. CMV is one of the TORCH infections (Toxoplasmosis, Other infections including syphilis, Rubella, CMV, and Herpes simplex virus [HSV]).

C. CMV is primarily asymptomatic or produces mild flulike symptoms and can develop 9 to 60 days after primary infection. The lymph nodes and spleen may be enlarged, and they may have extreme fatigue that can last even after normalizations of lab values. CMV symptoms in immunocompromised clients are often more pronounced, including bone marrow suppression, pneumonitis, hepatitis, colitis, nephritis, and retinitis (discussed later).

INCIDENCE

A. In the United States, as many as 60% of the population have been exposed to the virus. However, the number is more than 90% in high-risk groups such as those with HIV and unborn babies whose mother got infected during pregnancy. The age of prevalence varies worldwide, but in developing countries it is generally acquired during childhood and the risk increases with age.

B. Congenital infection rates are about 0.2% to 2.5%; however, most infected newborns will be asymptomatic.

C. Milk ingestion. Preterm infants are at the greatest risk to acquire CMV from breast milk.

D. Individuals with HIV who have low CD4 counts (<50 cells/u/L) are at high risk for CMV.

PATHOGENESIS

A. CMV is a lytic virus that causes cytopathic effect in vitro and in vivo. Its hallmark is enlarged cells with inclusion bodies. The cells are described as "owl's eye." This virus is transmitted both horizontally (by direct person-to-person contact with virus-containing secretions) and vertically (from mother to infant before, during, or after birth). When a person is infected, the CMV DNA can be detected with polymerase chain reaction (PCR) in all different cell lineages and body systems. Once infected, CMV affects the epithelia cells of the salivary gland, causing persistent infection and shedding of the virus.

B. The incubation period for horizontally transmitted CMV infections in households is unknown. CMV antibodies can be found as early as 4 to 7 weeks after initial infection and may persist as long as 16 to 20 weeks. CMV infection is lifetime; there is no treatment, although the virus may remain dormant.

C. Symptomatic CMV disease can affect almost all organs of the body, resulting in unknown origin of fever, pneumonia, encephalitis, myelitis, hepatitis, colitis, retinitis, and neuropathy. In very rare cases, especially in immunocompetent people, the manifestations can include Guillain–Barré syndrome, meningoencephalitis, pericarditis, myocarditis, thrombocytopenia, and hemolytic anemia.

1. Congenital CMV disease: The most common clinical findings include petechiae, jaundice, microcephaly, small size for gestational age, and hearing loss in children.
2. CMV pneumonia: It is detected via culture, chest x-ray, and histopathology; 6% of clients with CMV infection develop pneumonia. Immunosuppressed clients can develop life-threatening CMV pneumonia and the higher rate and greatest severity are among lung transplant recipients.
3. CMV retinitis: It is most common with AIDS clients and typically progresses to decreased visual acuity or even blindness if untreated. Retinitis must be confirmed by an ophthalmologist.
4. CMV gastritis and colitis: There is a combination of symptoms of upper and lower gastrointestinal (GI) tract, mucosal lesions seen by endoscopy; detection is by culture, histopathology, immunohistochemistry, or in situ hybridization. It can infect from the mouth through the colon, causing ulcerative lesions, abdominal pain, hematemesis, colitis, and diarrheal illness.

PREDISPOSING FACTORS

A. Exposure to young children, especially those in day-care centers.

B. Sexual contact (cervicovaginal secretions and semen).

C. Blood transfusions.

D. Hospital or occupational exposure.

E. Pregnancy. ▶

The contributions of B. Denise Hemby and Theresa M. Campo to this chapter in previous editions are acknowledged here.

F. Transplacental transmission.
G. Breast milk.
H. Immunosuppressed state.
I. Inflammatory bowel disease.

COMMON COMPLAINTS

A. Mild flu-like symptoms.
B. Fever.
C. Overwhelming fatigue.
D. Pharyngitis.
E. Ulcerative lesions in the mouth.

OTHER SIGNS AND SYMPTOMS

A. Visual changes.
B. Retinitis.
C. Arthralgias.
D. Nausea, abdominal cramping, and vomiting (includes hematemesis).
E. Prolonged fever.
F. Mild hepatitis.
G. Headache.
H. Immunocompromised persons:
1. Bacterial: pneumonia, retinitis, myocarditis, and aseptic meningitis.
2. Anemia.
3. Thrombocytopenia.

SUBJECTIVE DATA

A. Review onset of presenting signs, symptoms, and their duration.
B. Review the client's history for recent upper respiratory infection (and mononucleosis-like symptoms).
C. Elicit information concerning contact with any person known to be CMV-infected.
D. Review history for other family members with similar symptoms.
E. Ask the client about any occupational exposure.
F. Review the client's exposure to children in day-care centers.
G. Review recent blood transfusions and/or organ transplantation.
H. Determine the client's history for risk factors or presence of HIV:
1. If known HIV, does the client know their last viral load CD4 count?
2. Is the client taking antiretroviral therapy (ART)? When were the antivirals started?
I. Ask family members if the client has been confused, lethargic, or withdrawn, or has exhibited personality changes (CMV encephalitis, dementia).

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure, and weight (document serial weight loss).
B. Inspect:
1. Inspect skin for jaundice and petechiae.
2. Conduct a detailed eye exam:
a. CMV infection may appear as yellow-white areas with perivascular exudates and hemorrhage, having a "cottage cheese and ketchup" appearance at either the periphery or the center of the fundus:
i. Differentiating suspected CMV retinitis and cotton wool spots is essential. Cotton wool spots appear as small, fluffy white lesions with indistinct margins and are not associated with exudates or hemorrhages.
b. Evaluate field of vision.
c. Perform ear and nasal exam.
d. Perform an oral/throat exam.
e. Conduct hearing test.
f. Evaluate thoroughly for lymphadenopathy.
C. Auscultate:
1. Auscultate heart and lungs.
D. Palpate:
1. Palpate abdomen.
a. Note organomegaly.
b. Mild to moderate abdominal pain and tenderness associated with GI colitis.
c. Pregnancy: Palpate fundal height and evaluate for suspected intrauterine growth restriction (IUGR).
E. Neurologic exam:
1. Assess all cranial nerves.
2. Sensation (deficits may occur without loss of vibratory sense and proprioception).
3. Deep tendon reflexes.
4. Motor skills and coordination.
5. Gait.

DIAGNOSTIC TESTS

A. Nucleic acid detection (NAT) from blood, plasma, or tissue specimen is the most sensitive.
B. Pp65 antigenemia measures viral antigen in blood leukocytes.
C. Viral culture of specimen from urine, cervix, vagina, nasopharynx, and saliva.
1. Results cannot distinguish between primary and recurrent disease. Urine contains high titers of the virus because CMV is relatively stable in urine.
D. Complete blood count (CBC) with differential.
E. Complete metabolic panel (CMP): total direct and indirect serum bilirubin.
F. Colonoscopy with biopsy.
G. Serology for CMV immunoglobulin G (IgG) indicates past or recent infection and immunoglobulin M (IgM) indicates recent primary, early in illness, reactivation, or reinfection of the virus. Only the recovery of the virus from a target organ provides unequivocal evidence that the disease is caused by CMV infection. However, when comparing the first and second specimen, a fourfold or greater rise in IgG-specific antibody titer is usually considered evidence of acute infection.
H. Amniocentesis for PCR to detect CMV DNA is the preferred diagnostic approach for detecting the infected fetus.
I. Immunocompromised clients, especially transplant recipients, may be monitored for viral surveillance weekly using surrogate markers for viremia-PCR.
J. Chest x-ray or chest CT scan if indicated (CMV pneumonitis/interstitial infiltrates/nodules).
K. CT scan is indicated to evaluate abnormalities of the brain in the presence of an abnormal neurologic examination, seizures, and microcephaly.
L. Sensorineural hearing evaluation.
M. Routine maternal screening for CMV infection is not recommended during pregnancy. Lab tests that are currently available generally cannot conclusively determine whether a primary CMV infection has occurred during the pregnancy. Screening is advised if mother has ongoing flu or mononucleosis type symptoms with confirmed fetal abnormalities.

DIFFERENTIAL DIAGNOSES

A. CMV.
B. Autoimmune hepatitis.
C. Human HSV-6 infection.
D. Epstein–Barr virus (EBV; mono).
E. Other TORCH infections.
F. HIV/AIDS.
G. Viral hepatitis.
H. Enteroviruses.
I. Fever of unknown origin (FUO).

PLAN

A. General interventions:
1. Provide support for the client and the family.
2. Contact social services if long-term support will be required for these infants/families.
3. Rest is vital.

B. Client teaching:
1. Clients with splenomegaly should avoid activity that may increase the risk of injury to the spleen, such as contact sports and heavy lifting.
2. Mothers infected with CMV should be discouraged from breastfeeding because CMV is secreted in breast milk.
3. Isolation is not required.
4. Use universal precautions, especially good handwashing.
5. Do not share eating or drinking utensils, drinks, or food with toddlers or young children.
6. Educate about the importance of ART in treating CMV.
a. Clients with CMV retinitis may require lifelong suppressive therapy to prevent blindness.
b. Report visual impairment, floaters, or deterioration immediately.

C. Pharmaceutical therapy. The goals of pharmacotherapy are to prevent outbreaks and complications and to reduce morbidity.
1. Valganciclovir (Valcyte): drug of choice for prevention of CMV in solid-organ transplant clients due to ease of administration, monitor for leukopenia as the first-line option, and used for prophylaxis.
a. Retinitis with AIDS: Start valganciclovir (Valcyte) 900 mg PO Q12H for 21 days; maintenance: 900 g PO daily.
b. Transplant: Start valganciclovir (Valcyte) 900 mg daily within 10 days of transplant for 200 days.
2. Ganciclovir (Cytovene): a mainstay except in CMV pneumonitis, colitis, and retinitis, and treatment of CMV, HSV, human herpesvirus (HHV)-6, HHV-7, and HHV-8; requires intravenous (IV) access and has major adverse reactions including fever, rash, diarrhea, neutropenia, anemia, thrombocytopenia, and leukopenia.
a. Colitis: Start ganciclovir (Cytovene) 5 mg/kg IV Q12H; once PO tolerated and CMV no longer detected in the blood can change to valganciclovir 900 mg PO Q12H for 21 to 42 days.
b. Clients receiving ganciclovir should have blood counts monitored closely due to dose-dependent bone marrow suppression. Monitor for leukopenia, neutropenia, anemia, or thrombocytopenia. Stop ganciclovir when neutrophil counts are less than 500/mm^3.
3. Cidofovir (Vistide): approved for CMV retinitis.
a. Retinitis: Start 5 mg/kg IV every week for 2 weeks; maintenance 5 mg/kg IV every 2 weeks.
4. Foscarnet (Foscavir; third-line option/alternative option in ganciclovir-resistant CMV): nephrotoxic; monitor renal function.
a. Retinitis: Start 60 mg/kg IV Q8H for 14 to 21 days; maintenance 90 to 120 mg/kg IV daily.
b. HSV: 40 mg/kg IV Q8H for 14 to 21 days.
5. CMV vaccines are currently in clinical trials.
6. For retinitis diagnosed in clients with AIDS infection with no drug resistance, the known recommendation is to treat with ganciclovir 2 mg intravitreally (into the vitreous humor of the eye) weekly by a specialist until lesion inactivity is achieved, or foscarnet 2.4 mg intravitreally weekly as second option and ganciclovir 5 mg/kg IV Q12H hours for 12 to 14 days, or valganciclovir 900 mg orally BID for 14 to 21 days.

FOLLOW-UP

A. Because retinitis is the most common manifestation of CMV disease, clients with central nervous system (CNS), GI, or pulmonary disease should be assessed with a dilated retinal examination to detect subclinical retinal disease.
B. Monitor CBC, serum creatinine, and electrolytes (especially calcium and magnesium) every week.
C. The client should be seen for reports of excessive bruising or bleeding, jaundice, or abnormal CNS functioning.

CONSULTATION/REFERRAL

A. Consultation with an infectious disease specialist is indicated for acute infection, especially for immunocompromised clients.
B. Antivirals have many adverse effects and are best managed by a physician who has experience using these drugs.
C. Consultation with a hematologist is needed in severe cases, especially with hemolytic anemia and thrombocytopenia.
D. Consultation with a neurologist is indicated for meningitis, encephalitis, polyneuritis, and Guillain–Barré syndrome.
E. Consultation with an ophthalmologist is needed on an emergent basis for a dilated retinal examination due to the risk of blindness. Serial dilated retinal examinations should be done after ART induction therapy and monthly thereafter.
F. Consultation with a perinatologist is indicated in pregnant clients. Evaluation may include ultrasonography, amniocentesis, and percutaneous umbilical blood sampling (PUBS).
G. Refer to a gastroenterologist for an endoscopic evaluation with tissue biopsies.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Between 55% and 85% of pregnant persons are immune to CMV, with a higher prevalence of immunity in lower socioeconomic populations.
2. Susceptible pregnant persons have a 2% to 2.5% risk of acquiring primary CMV during pregnancy. Those with prior immunity have a 1% chance of reactivation of latent infection.
3. Perinatal transmission of CMV occurs by four routes:
a. In utero transplacental infections.
b. Ascending infections from the cervix.
c. Exposure to infected secretions from the lower genital tract during delivery.
d. Ingestion of infected breast milk.

Because CMV is spread by intimate contact with infectious secretions, handwashing after exposure to secretions is particularly important for pregnant healthcare workers.

▶

B. Adults:

1. Occupational exposure: Risk often appears to be greatest for childcare personnel who care for children younger than 2 years of age.

2. Routine serologic screening for childcare staff is not currently recommended.

3. Pregnant personnel who may be in contact with CMV-infected clients should be counseled regarding the risk of acquiring CMV infection and about the need to practice good hygiene, particularly handwashing.

C. Geriatrics:

1. CMV infection increases the mortality rate in healthy older people based on an excess—a near doubling—of vascular deaths.

2. Although uncommon, CMV colitis in the elderly immunocompromised person is potentially life-threatening, particularly in persons with chronic renal disease:

a. The symptoms of CMV colitis are nonspecific.

i. The most common presentation is bloody diarrhea.

ii. Colonoscopy findings are variable and nonspecific; therefore, a biopsy is critical for the diagnosis.

3. CMV and HSV-1 (oral herpes of cold sore and fever blisters) were found to be associated with the development of Alzheimer disease secondary to the effects on the immune system. Monitor cognitive function, behavior problems, or any developing emotional changes/paranoias in geriatric clients with a history or active infection of CMV with HSV-1.

4. Immunosenescence is the age-related reduction of the immune system and is related to geriatric frailty. Studies indicate that elderly females with frequent CMV reactivations are susceptible to aggressive ongoing frailty. Assess these clients for fall risks, weakness, undernutrition, depression, and sleep disorders.

5. An association has been reported between CMV infection and systolic blood pressure. Healthy geriatric persons ≥70 years old with hypertension (HTN) who have been diagnosed with CMV need their blood pressures closely monitored and effectively controlled to reduce the risk of cardiovascular events (transient ischemic attack [TIA], cerebrovascular accident [CVA], myocardial infarction [MI]).

6. ART for older adults ≥50 years old who have CMV retinitis is accepted as safe and is recommended to prescribe regardless of CD4 count. After initiating ART in geriatrics with AIDS-related CMV, the viral load suppression will be greatest in clients ≥65 years old.

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ENCEPHALITIS

DEFINITION

A. Encephalitis is the inflammation of brain parenchyma caused by infectious agents or other toxins. It primarily involves the brain, but meninges are frequently involved. It presents as focal or diffuse neuropsychological dysfunction. Most cases are viral, but it can also be caused by bacterial, autoimmune, and fungal disorders. Encephalitis is different from meningitis but they share many clinical features, such as acute onset of febrile illness with altered mental status and photophobia, headache, or stiff neck. There are several specific types of encephalitis, with the most common cause being herpes simplex encephalitis (HSE) and arthropod-borne viruses. The mortality depends on the etiologic agents of the encephalitis. Clients with either syndrome present with fever, headache, and altered states of consciousness. In most cases of encephalitis, there is some concomitant meningeal inflammation, in addition to the cephalic component, a condition commonly referred to as meningoencephalitis.

INCIDENCE

A. The true incidence of encephalitis can be difficult to track because there are no standardized reporting policies, as well as the lack of strict enforcement. In the United States, several thousand cases of viral encephalitis are reported to the Centers for Disease Control and Prevention (CDC) each year. In the United States, the incidence of encephalitis is between 10 and 15 per 100,000 per year. Estimates on an annual incidence of about 20,000 new cases have been reported by the CDC. There are numerous viruses known to cause encephalitis, but the most common is HSE, with an overall incidence of 10% of all cases in the United States. The arbovirus group affects 150 to 3,000 people per year and the two most common are St. Louis encephalitis and California encephalitis (CE). St. Louis encephalitis is caused by a virus that is primarily found near the Mississippi River but can also be found throughout the United States. CE can be found in the rural areas of the Upper Midwest and Northeast but is not common to California as its name depicts.

1. HSE: HSE may occur in both healthy and immunocompromised adults and is potentially deadly if left untreated. Quick identification and treatment can be lifesaving. Herpes encephalitis is the most common form in the United States and has a mortality rate as high as 75% in untreated individuals. Survival and recovery from neurologic sequelae are related to the mental status at the time of acyclovir initiation.

2. Arthropod-borne viruses: There are more than 20 arboviruses that can cause encephalitis. These arboviruses are enveloped RNA viruses from different families. Some common arboviral encephalitides are the following:

a. St. Louis encephalitis virus (SLEV): has a mortality rate of 2% to 20% but is higher in clients 60 years and older. Long-term sequelae include behavioral disorders, memory loss, and seizures.

b. Eastern equine encephalitis (EEE) virus: rare and is the deadliest; it is found in areas of New England and surrounding areas. EEE is one of the most severe mosquito transmitted diseases in the United States with ▶

about 33% mortality and significant brain damage in survivors. Death usually occurs in 2 to 10 days after onset of symptoms.

c. Western equine encephalitis (WEE) virus: milder disease, in rural communities west of Mississippi River, west of the Rocky Mountains. It is associated with fewer deaths, less than 5%, and less morbidity. It is a summertime disease, common between April and September, with peaks in July and August. Heavy rainfalls or snowy seasons can increase the vector population.

d. Japanese encephalitis (JE) virus: endemic in rural areas. In the United States, JE develops mostly among military personnel, expatriates, and returning travelers. Outbreaks are rare in the U.S. territories of Guam and Saipan. It is a seasonal disease, occurring mostly in temperate areas starting from June to September. In subtropical areas, it can be seen as early as March and go until October.

PATHOGENESIS

A. HSE: believed to be the reactivation of herpes simplex virus (HSV) lying dormant in the trigeminal ganglia. The virus replicates outside the central nervous system (CNS) and enters by either hematogenous spread or by travel along neural pathways. Human herpesvirus (HHV)-6 is the causative agent.

B. SLEV: an enveloped, single-stranded, positive-sense RNA virus from the subgroup of Flaviviridae. It is transmitted via mosquito from wild birds to humans. The mosquitoes include *Culex pipiens, C. tarsalis,* and *C. quinquefasciatus.*

C. EEE: from the *Alphavirus* genus, Togaviridae family, and is transmitted to humans from the bite of *Culiseta melanura* mosquitos. The virus has a single-stranded, positive-sense RNA genome. The incubation period ranges from 4 to 10 days.

D. WEE: belongs to the *Alphavirus* genus and causes encephalitis and viral symptoms without a rash. It is spread primarily by the vector mosquito *C. tarsalis.* It is transmitted into the subcutaneous and cutaneous tissue from bites. It is usually subclinical and may mimic other inflammatory and viral syndromes.

E. JE: belongs to the *Flavivirus* genus. It is transmitted via the bite of infected *Culex* mosquitoes, especially *C. tritaeniorhynchus, C. vishnui, C. gelidus*, and *C. fuscocephala.* Incubation period averages 6 to 8 days, but can range from 4 to 15 days.

PREDISPOSING FACTORS

A. Exposure to vectors:
1. Mosquitoes.
2. Ticks.
3. Bats.
4. Raccoons.
5. Feral dogs/cats.
6. Sandflies.

B. HIV-positive.

C. Herpes.

D. Occupational exposure:
1. Laboratory workers.
2. Healthcare workers.
3. Veterinarians.

E. Recreational activities (camping/hunting).

F. Recent travel to endemic areas.

G. Recent vaccination.

COMMON COMPLAINTS

A. Clients may present focal or diffuse symptoms.
1. Altered mental status.
2. Personality changes (very common).
3. Lethargy.
4. Neck pain, stiffness.
5. Ataxia.
6. Cranial nerve defects.
7. Photophobia.
8. Generalized or focal seizures.
9. Decreased level of consciousness (LOC).

OTHER SIGNS AND SYMPTOMS

A. CNS symptoms:
1. Nuchal rigidity.
2. Irritability.
3. Hemiparesis: weakness or paralysis on one side of body.
4. Flaccid paralysis.
5. Seizures.
6. Exaggerated deep tendon reflexes.
7. Ataxia.
8. Nystagmus.

B. Photosensitivity.

C. Swollen or protruding eyes.

D. Malaise.

E. Nausea and/or vomiting.

F. Dysphagia with rabies.

SUBJECTIVE DATA

A. Review the onset, duration, and course of all symptoms.

B. Rule out recent history of chickenpox, rubeola, herpes, or other infections.

C. Ask the client about recent travel.

D. Question the client regarding recent mosquito or animal bites (rule out rabies).

E. Elicit a detailed sexual history.

F. Does the client have HIV?

G. Ask about recent recreational activities, including camping, spelunking, or hunting.

PHYSICAL EXAMINATION

A. Signs may be diffuse or focal.

B. Check temperature, pulse, respirations, and blood pressure.

C. Inspect:
1. Observe general overall appearance.
2. Conduct an eye exam.
3. Examine the skin for rash, vesicles, or bites.
   a. Maculopapular rash is seen in approximately half of clients with West Nile virus (WNV), measles, HHV-6, and Colorado tick fever.
   b. Erythema migrans: Lyme disease (LD).
   c. Grouped vesicles in a dermatomal pattern suggest varicella zoster.
   d. Classic herpetic skin lesions suggest herpes encephalitis.
4. Assess dehydration status.
5. Observe for seizure activity.
6. Observe for tremors of the eyelids, tongue, lips, and extremities, which may suggest the possibility of St. Louis encephalitis or West Nile encephalitis.

D. Auscultate:
1. Auscultate the lungs and monitor breathing pattern.
2. Auscultate the heart.

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E. Palpate:
1. Palpate the lymph nodes: preauricular, posterior auricular, submental and sublingual, anterior cervical chains, and supraclavicular nodes.
2. Palpate the mastoid bone.
F. Neurologic exam:
1. Assess LOC.
2. Assess the client for personality changes.
3. Assess for meningeal signs:
a. Signs of meningeal irritation include nuchal rigidity.
b. Positive Brudzinski and Kernig signs (Figures 19.1 and 19.2).
i. Brudzinski sign: Place the client supine and flex the head upward. Resulting flexion of both hips, knees, and ankles with neck flexion indicates meningeal irritation.
ii. Kernig sign: Place the client supine. Keeping one leg straight, flex the other hip and knee to a bent knee to form a 90° angle. Slowly extend the lower leg. This places a stretch on the meninges, resulting in pain and spasm of the hamstring muscle. Resistance to further extension can be felt.
4. Check deep tendon reflexes (exaggerated and/or pathologic reflexes).

DIAGNOSTIC TESTS

A. Complete blood count (CBC) with differential.
B. Complete metabolic panel (CMP).
C. Urine electrolyte levels.
D. Polymerase chain reaction (PCR) tests for viruses.
E. Serology for arboviruses.
F. Lumbar puncture for the following:
1. Cerebrospinal fluid (CSF) analysis (in autoimmune encephalitis up to one-third of clients have normal CSF):

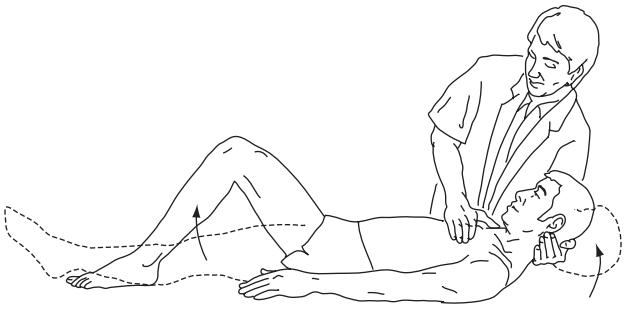


FIGURE 19.1 Brudzinski sign.



FIGURE 19.2 Kernig sign.

a. Pressure.
b. Cell count (elevated white blood cells/negative red blood cells).
c. Microorganisms.
d. Glucose (normal to low).
e. Protein (normal to elevated).
f. CSF culture (viral and bacterial): Detection of virus-specific immunoglobulin M (IgM) antibody in CSF is diagnostic.
g. CSF serology: etiology-dependent.
h. CSF PCR: etiology-dependent.
G. HSV cultures.
H. Viral cultures of CSF including HSV.
I. Blood cultures for bacterial pathogens.
J. Complement fixation antibodies to identify arbovirus.
K. Heterophile antibody and cold agglutinin testing for Epstein–Barr virus (EBV).
L. Serologic tests for *Toxoplasma.*
M. EEG.
N. CT scan: useful to rule out space-occupying lesions or brain abscess; can be normal in early encephalitis.
O. MRI: sensitive in detecting demyelination as well as autoimmune encephalitis.
P. Stool if enteroviruses are suspected.
Q. Throat swab viral detection.
R. Skin lesions and urine may be cultured for herpes simplex and cytomegalovirus (CMV).

DIFFERENTIAL DIAGNOSES

A. Encephalitis.
B. WNV.
C. SLEV.
D. EEE.
E. JE.
F. HSE.
G. WEE.
H. Venezuelan equine encephalitis.
I. Rabies.
J. Meningitis.
K. Brain abscess.
L. Syphilis.
M. Intracranial hemorrhage.
N. Head trauma.
O. Toxic ingestion.
P. Fungal meningitis.
Q. Rocky Mountain spotted fever (RMSF)/tick borne diseases.
R. Cerebral bacterial infections.
S. Toxoplasmosis.
T. LD.
U. CMV.
V. Nonparalytic poliomyelitis.
W. Epilepsy.

PLAN

A. General interventions:
1. If encephalitis is suspected, hospitalization is recommended for further diagnostic studies and evaluation, preferably managed in a neurology unit under isolation until etiology is diagnosed.
B. Client teaching:
1. Prevention for vector borne encephalitis involves mosquito and tick avoidance, use of insect repellents, and vaccination:

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a. Travelers planning prolonged stays (more than 30 days) in endemic areas during the transmission season, especially with activities such as bicycling, camping, or other unprotected outdoor activities in rural areas.

2. If rabies is suspected, the domestic animal should be observed for 10 days to detect rabid behavior. If there is no indication of rabies, the animal should be immunized. Animals that show rabid behavior or wild animals should be sacrificed and their brains submitted to the local/state health department for pathology testing.

C. Pharmaceutical therapy. Goal is to reduce morbidity and prevent complications.

1. Antivirals are used to treat viral encephalitis:

a. If unknown etiology, treat with acyclovir empirically until diagnosis is determined, if not contraindicated, with supportive care.

2. Herpes simplex virus encephalitis-treatment should be initiated as soon as possible; acyclovir 10 to 15 mg/kg intravenously (IV) Q8H for 10 days.

3. Antibiotics for bacterial etiology.

4. Anticonvulsants for seizures.

5. Other pharmaceutical therapies depend on specific causal agent.

6. Corticosteroids are anti-inflammatory agents for treatment of postinfectious encephalitis and acute disseminated encephalitis. They are also used to treat various allergic and inflammatory diseases. The use of large doses of steroids should be limited to 3 to 5 days. The benefit of use is considered controversial at this time and should be determined on an individual client evaluation.

7. If rabies is suspected, human rabies immune globulin should be given 20 U/kg dose.

FOLLOW-UP

A. Follow-up varies and is specific to the causal agent.

B. Long-term management of clients with neurologic sequelae includes rehabilitation services, home care, or nursing home placement for convalescent care.

CONSULTATION/REFERRAL

A. Refer the client to a physician and/or hospital. Consultations include a neurologist, infectious disease specialist, and neurosurgeon for managing elevated intracranial pressure.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. Travelers: Advise client to take mosquito bed nets and aerosol insecticide sprays to reduce the risk of mosquito bites at night.

2. For travelers to areas with high risk of JE vaccination, recommend:

a. Inactivated JE vaccine series:

i. Adults 18 to 65 years: two doses of 0.5 mL administered intramuscularly (IM) on days 0 and 7 or day 28.

ii. Children 2 months to <18 years of age: two doses of 0.5 mL administered IM on days 0 and 28.

iii. Children >2 months to 2 years: two doses of 0.25 mL administered IM on days 0 and 28.

B. Geriatrics:

1. Elderly clients may be at risk for a severe form of the disease. Mortality is significantly higher in persons older than age 70.

2. Encephalitis should be considered in all elderly clients who have progressive mental confusion.

3. WNV encephalitis occurs primarily in clients older than 65 years of age.

4. HSV is the main cause of encephalitis in individuals older than 60 years of age, in whom it has a more severe clinical course.

5. HSE in the elderly presents with atypical clinical symptoms:

a. Progressive amnestic cognitive disorder.

b. Behavioral changes.

c. Progressive mental confusion.

d. Focal neurologic deficits, which mimics stroke.

6. HSV encephalitis is a deadly virus of the brain, and geriatric clients have a higher incidence of the disease with the worst prognosis. Acyclovir antiviral treatment (if implemented on time) can effectively reduce morbidity complications and reduce the risk of mortality. Consider treating empirically while waiting for diagnosis confirmation.

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LYME DISEASE

DEFINITION

A. Lyme disease (LD) is the most common vector borne illness in the United States. The number of reported cases in the United States has tripled since the late 1990s. It is a multisystem illness usually caused by the spirochete *Borrelia burgdorferi* and is carried and spread by several types of ticks. In 2019, it was the sixth most commonly reported disease. LD is transmitted to humans via the bite of an infected tick. Ticks can attach to any part of the body, but are found in hard-to-see areas like the groin, armpits, scalp, and back. There is not a minimum attachment time, but the longer the attachment the greater risk or likelihood of acquiring the infection. Generally, it is 36 to 48 hours or longer before *B. burgdorferi* can be transmitted.

B. Morbidity from LD usually involves neurologic and cognitive dysfunction. There is a subset of clients treated for LD with a recommended 2- to 4-week antibiotic regimen who experience some post-LD symptoms. Posttreatment Lyme disease syndrome (PTLDS) includes cognitive disturbances, fatigue, joint/muscle pain, headaches, hearing loss, vertigo, mood disturbances, paresthesias, and difficulty sleeping.

C. There is no evidence that LD is transmitted from person to person or from pet to person, or spread via the air, food, or water, or via bites from mosquitoes, flies, fleas, and lice. *B. burgdorferi* can live in blood that is stored for donation; however, after completing the antibiotic treatment, blood donation may be considered.

INCIDENCE

A. There is an increasing number of Lyme and tickborne disease, with the number of counties in the north eastern and upper midwest United States at high risk for LD increasing 300% over the past 20 years. Current statistics estimate that around 476,000 people get LD each year in the United States. LD is the most common tick borne disease in the Northern Hemisphere. Age distribution is bimodal; the peak is in children aged 5 to 14 years and the second in adults aged 45 to 54 years.

B. LD has a seasonal influence that reflects the feeding patterns of ticks. Infections occur most often between May and November, with a peak incidence in June through August, with 75% of cases occurring during the summer months.

PATHOGENESIS

A. *B. burgdorferi*, a spirochete bacterium, is an infectious agent carried by several types of ticks. The ticks bite mice or deer that are infected with *B. burgdorferi*, and infect humans with their bites. The blacklegged tick or deer tick, *Ixodes scapularis*, spreads the disease in the northeastern, mid-Atlantic, and north-central United States. The western blacklegged tick, *I. pacificus*, spreads the disease on the Pacific Coast of the United States. The life cycle of blacklegged ticks is 2 years and it goes through four life stages: egg, six-legged larva, eight-legged nymph, and adult. Once hatched, the tick must have a blood meal to survive. Once infected, a tick can transmit infection throughout its life. Rodents and pets can also harbor deer ticks.

B. The spirochete enters the bloodstream at the time of tick feeding. The incubation period is 3 to 32 days, or about 1 to 3 weeks after the bite. Late manifestations occur several months to more than 1 year later.

PREDISPOSING FACTORS

A. People of all ages are affected.

B. Recreational exposure:

1. Hiking, golfing, hunting, soccer.

C. Gardening.

D. Exposure to rodents such as field mice and domestic pets, which may also carry the ticks.

COMMON COMPLAINTS

A. Fever, chills, "flulike symptoms."

B. Fatigue.

C. Headache.

D. Joint pain.

E. Muscle aches.

F. Swollen lymph nodes.

G. Rash or lesion at site of tick bite.

OTHER SIGNS AND SYMPTOMS

A. Stage 1: acute early localized; begins days or weeks (1–30 days) after infection:

1. Rash: erythema migrans—"bull's eye"—flat or slightly raised red area at the site of the tick bite.

It usually begins as a red macule at the site of the tick bite and spreads out to form a large annular lesion with red secondary outer rings, an intense red outer border (measuring at least 5 cm), and some clearing at the site of the bite. Appearance is a "bull's-eye" shape. The lesion is generally painless and not pruritic.

2. Body aches.
3. Fever or chills.
4. Swollen lymph nodes.

B. Stage 2: disseminated infection (early disseminated); develops 3 to 10 weeks after infection:

1. Fever, malaise, debilitating fatigue.
2. Headache.
3. Photophobia.
4. Mild neck stiffness.
5. Joint or muscle pain.
6. Migratory arthralgia.
7. Rash: diffuse erythema.
8. Itching.
9. Transient heart block:
   a. In 5% to 10% of cases, clients have cardiac involvement: a transient heart block ranging from asymptomatic, first-degree atrioventricular (AV) block to complete heart block with fainting; the cardiac phase lasts 3 to 6 weeks.
10. Bell's palsy:
    a. A unilateral or bilateral Bell's palsy is the most common cranial nerve deficit.
11. Mild encephalopathy.

C. Stage 3: chronic (late disease); occurs months to years after initial infection:

1. Prolonged arthritis: hallmark of stage 3.
   a. Approximately 60% of complaints evolve into frank arthritis. Onset of arthritis is variable but averages 6 months from the time of initial infection. It tends to involve the large joints; the knee is the most common (90%) site and the pattern continues to be oligoarticular.
2. Chronic neurologic deficits.
3. Distal paresthesia.
4. Radicular pain.
5. Memory loss.

SUBJECTIVE DATA

A. Review the onset, course, and duration of symptoms.

B. Ask the client about any recent outdoor activities, such as camping, hiking, gardening, or other activities. Less than half of the people infected remember a tick bite. A history of a tick bite is not necessary for diagnosis.

C. Have other family members had similar symptoms?

D. Review thorough history of medications.

E. Review any history of rash and course of spread.

F. Rule out late symptoms associated with LD, such as arthritis, memory loss, and distal paresthesia.

G. Has the client been previously treated for LD or Rocky Mountain spotted fever (RMSF)?

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.

B. Inspect:

1. Observe general overall appearance.
2. Observe rash pattern and type.
3. Inspect the skin; observe for target-like pattern.

C. Palpate:

1. Palpate the lymph nodes and mastoid bones.
2. Examine the joints for tenderness, swelling, and range of motion.

D. Auscultate:

1. Auscultate the heart and lungs.

E. Neurologic exam:

1. Evaluate for signs of meningeal irritation by means of Brudzinski sign and Kernig signs (see Figures 19.1 and 19.2):

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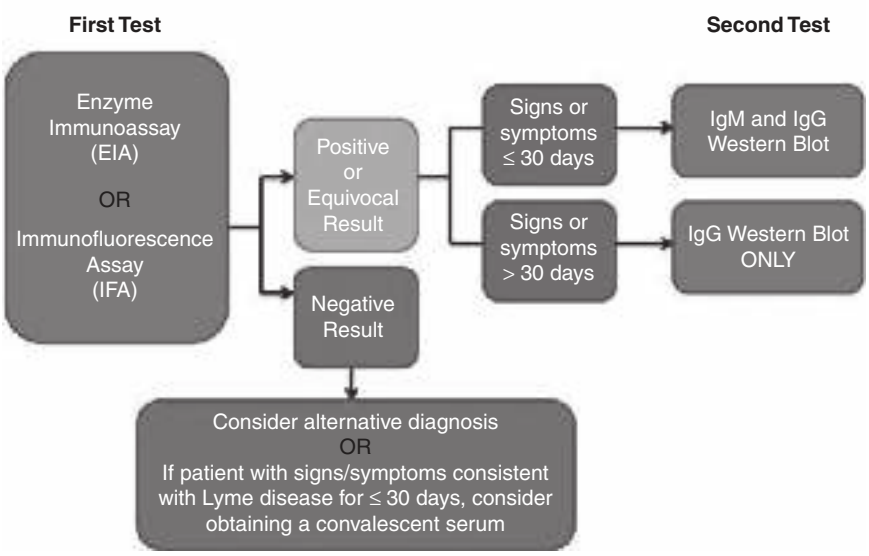


FIGURE 19.3 Two-tiered test for Lyme disease. IgG, immunoglobulin G; IgM, immunoglobulin M.

Source: From Centers for Disease Control and Prevention. (2015). Lyme disease: Two-step laboratory testing process. https://www.cdc.gov/lyme/diagnosistesting/labtest/twostep/index.html.

a. Brudzinski sign: Place the client supine, one hand on the client's chest, one hand behind the client's head, and flex the head upward. Flexion of lower extremities (hip and knees) is a positive sign and resulting flexion indicates meningeal irritation.
b. Kernig sign: Place the client supine. Keeping one leg straight, flex the other hip and knee to a bent knee to form a 90° angle, then completely extend the leg at the knee joint. This places a stretch on the meninges, resulting in pain. If the leg cannot be completely extended, this is a positive Kernig sign.

DIAGNOSTIC TESTS

A. Immunoblot tests (Figure 19.3).
B. Immunoglobulin M (IgM): acute, needs to be done in the first few weeks.
C. Immunoglobulin G (IgG): more reliable, can take 4 to 6 weeks for the body to produce enough to detect.

OTHER TESTS TO RULE OUT OTHER INFECTIONS

A. Complete blood count (CBC) with differential.
B. Comprehensive metabolic panel: alanine transaminase (ALT) and aspartate transaminase (AST; may be mildly elevated).
C. Sedimentation rate.
D. Arthrocentesis for joint effusion: chronic/late disease.
E. Polymerase chain reaction (PCR) testing.
F. Creatine phosphokinase.
G. Lumbar puncture indicated for the presence of neurologic symptoms or manifestations of meningitis.

DIFFERENTIAL DIAGNOSES

A. LD.
B. Insect or spider bite.
C. RMSF.
D. Ehrlichiosis.
E. Tularemia.
F. Cellulitis.
G. Arthritis.
H. Bacterial meningitis.
I. Chronic fatigue syndrome (CFS).
J. Viral syndrome.
K. Nummular eczema.
L. Tinea corporis (ringworm).

PLAN

A. General interventions:
1. Prophylactic treatment with antibioitics after a tick bite is not recommended.
2. Treatment with doxycycline for tick bites should be prescribed when skin exhibits inflammation.
3. Treat with doxycycline when client becomes symptomatic.
4. There is no evidence of "chronic Lyme disease." Some symptoms may last for several months; however, long-term treatment has not proven to be effective and can contribute to other adverse problems.
B. Client teaching: *See Client Teaching Guide for this chapter, "Lyme Disease and Removal of a Tick."*
1. Not all neurologic signs and symptoms may completely resolve (such as headache, photophobia, Bell's palsy, and third-stage symptoms).
2. Clients with active LD should not donate blood because spirochetemia occurs in early LD. Clients who have been treated for LD in the past can be considered for blood donation.
3. Currently, there is no vaccine for LD. The vaccine was withdrawn in early 2002. Previously vaccinated clients are not protected against LD.
4. Nonspecific symptoms may persist for months after treatment of LD. There is no evidence that the complaints represent ongoing active infection or need repeated antibiotics:
a. Headache.
b. Fatigue.
c. Arthralgias.

C. Pharmaceutical therapy:

NOTE: The Infectious Diseases Society of America (IDSA) 2006 guidelines state that for LD be given if a person meets the criteria:

1. Tick attached greater than 36 hours.
2. Tick identified.
3. Infection rate is greater than 20% in the local area.
4. No contraindications to doxycycline:
   a. For chemoprophylaxis in clients older than 8, one-time dose of doxycycline 200 mg.
5. Treatment for early LD with erythema migrans.
   a. Early localized LD, with erythema migrans:
      i. Doxycycline:
         1) Adults: 100 mg PO BID for 14 days. OR
      ii. Amoxicillin:
         1) Adults: 500 mg PO TID for 14 days. OR
      iii. Cefuroxime:
         1) Adults: 500 mg PO BID for 14 days.
   b. LD with neurologic involvement:
      i. Ceftriaxone (Rocephin).
         1) Adults: 2 g/d intravenously (IV) for 14 days. OR
      ii. Cefotaxime:
         1) Adults: 2 g IV Q8H for 14 days. OR
      iii. Penicillin G:
         1) Adults: 18 to 24 million U/d IV divided doses Q4H for 14 days. OR
      iv. Doxycycline:
         1) Adults: 200 to 400 mg/d PO/IV BID for 10 to 28 days.
6. The Jarisch–Herxheimer reaction (JHR); onset 24 to 72 hours after initiating antibiotics and can last for weeks. JHR occurs when there is accelerated phagocytosis of the spirochetes, causing a rise in cytokines. Symptoms include increased fever, muscle, joint, and body pain, headache, low blood pressure, and elevated heart rate. It is a normal part of treatment for late-stage/chronic LD. Symptoms should be closely monitored. Nonsteroidal anti-inflammatory drugs (NSAIDs) may be beneficial and the antimicrobial agent should be continued.

FOLLOW-UP

A. Follow-up depends on the stage of disease.

B. Repeat Lyme titer in 1 to 2 months to determine the need for continuation of antibiotic therapy.

CONSULTATION/REFERRAL

A. Referral to a physician is indicated when the client with strong clinical evidence of LD fails to respond to prescribed antibiotics.

B. Consultation with a neurologist is particularly important for clients with refractory neurologic deficits.

C. Consultation with a rheumatologist is needed for clients with persistent or debilitating arthritis or fibromyalgia occurring after LD.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Maternal–fetal transmission of infection with subsequent injury to the fetus has been reported. Antibiotic treatment should be instituted promptly in symptomatic clients.
2. Tetracyclines are contraindicated in pregnancy. Otherwise, therapy is the same as for nonpregnant persons.
   a. For localized early LD, amoxicillin 500 mg TID for 21 days.
   b. For disseminated early LD or any manifestation of late disease, penicillin G 20 million units daily for 14 to 21 days.
   c. For asymptomatic seropositivity, no treatment is necessary.
3. There is no causal relationship between maternal LD and congenital malformations.
4. There is no evidence that LD can be transmitted in breast milk.

B. Geriatrics:

1. There is an increased risk of LD complications for the elderly population secondary to age-related decline of immune systems. It is important that older adults understand the early signs of LD and are educated that the earlier treatment is initiated the better chance they have of overcoming the illness.
2. Older adults often have pets for companionship. Be sure to coach pet-owning clients on the importance of bathing and grooming their dogs, and to talk to their veterinarian about tick prevention measures.
3. Encourage older adults to plan exercise away from high grass and leaf litter, and to walk in the center of the trails/greenbelts. Emphasize to older adults how to use products that contain permethrin to treat boots, pants, and socks, and to tumble-dry clothes on high heat for 10 minutes to kill ticks after returning home from picnics and outdoor social functions.
4. Geriatric syndrome assessments:
   a. For older adults suffering from acute psychiatric behaviors or atypical chronic mental disease without a therapeutic response to treatment, LD should be considered and ruled out.

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MENINGITIS

DEFINITION

A. The following are the types of meningitis.

1. Meningitis is an acute inflammation of the meninges, the three layers of membranes that enclose the brain and spinal cord. Viral or bacterial infections are the most common causes of meningeal inflammations.
2. Bacterial meningitis is caused by pyogenic inflammation of the meninges and subarachnoid cerebrospinal fluid (CSF). If the infection is left untreated, it can lead to death or lifelong disabilities. There are two categories of ▶

infections involving the central nervous system (CNS): one involves the meninges (meningitis) and the other the parenchyma (encephalitis).

3. Bacterial meningitis is a severe infection with associated complications, including brain damage, hearing loss, neurologic/learning disabilities, and digit or limb amputations. Mortality rate varies in part with the organism and if it is a nosocomial or a community-acquired infection.

4. Viral meningitis, in most cases (85%), is benign, self-limiting, and may only need supportive care; however, in some instances, an antiviral may be indicated. Causes of meningitis can include viruses, bacteria, fungi, drugs, and parasites.

a. Viral meningitis is the most common type of meningitis.
   i. *Enterovirus* category: coxsackievirus A, coxsackievirus B, and echoviruses.
   ii. West Nile, influenza, mumps, measles, herpes, HIV, *Coltivirus* (Colorado tick fever), and lymphocytic choriomeningitis virus.

b. Bacterial: The most common organisms are:
   i. *Streptococcus pneumoniae, Neisseria meningitidis, Haemophilus influenzae, Listeria monocytogenes*, and group B *Streptococcus* (GBS).

c. Fungal meningitis: The most common fungi are:
   i. *Cryptococcus* (most common form of meningitis in people with HIV or cancer), *Histoplasma, Blastomyces, Coccidioides.*

d. Parasitic meningitis: The most common parasites are:
   i. *Angiostrongylus cantonensis, Baylisascaris procyonis, Gnathostoma spinigerum.*
   ii. *Naegleria fowleri,* a primary amebic meningoencephalitis (PAM), is a very rare form of parasitic meningitis. It is usually fatal.

e. Noninfectious meningitis: The most causes are:
   i. Cancers.
   ii. Systemic lupus erythematosus (SLE): often precipitated by drugs.
   iii. Drug-induced meningitis (DIAM) includes nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, antimicrobials, muromonab (OKT3), eculizumab (Soliris), intravenous immunoglobulin (IVIG), tumor necrosis factor (TNF)-alpha inhibitors, and antineoplastics. Drugs and diagnostic agents administered intraventricularly and intrathecally.
   iv. Neurosurgery and deep brain stimulation hardware.

f. Head trauma: Noninfectious insult, such as blood in the subarachnoid space, causes aseptic meningitis. Three baseline clinical features have been independently associated with an adverse outcome: hypotension, altered mental status, and seizures.

INCIDENCE

A. There are more than 10,000 cases of viral meningitis reported annually, but it is speculated that the number is much higher because the reporting is not as strong as it is with bacterial meningitis.

B. The Centers for Disease Control and Prevention (CDC) reports the incidence of inpatient hospitalization from viral meningitis can range from 25,000 to 50,000 per year, which is approximately 11 per 100,000 population.

C. The incidence of meningitis varies and is based on the etiologic factor; it is higher in developing countries due to the lack of preventive services (e.g., vaccinations).

D. In the United States, almost 4,100 cases and 500 deaths occur annually from bacterial meningitis. The overall incidence of bacterial meningitis in the United States has declined because of the *Haemophilus influenzae* type b vaccination.

E. In adults after age 60, the risk of meningitis starts to rise again due to underlying comorbid conditions and weakened immune systems.

F. Meningitis affects all races, but in the United States there is a higher reported rate in Blacks than Whites and Hispanics.

PATHOGENESIS

A. Meningitis is caused by an infectious agent that has established or colonized as a localized infection somewhere in the host. Sites include the nasopharynx, respiratory tract, skin, gastrointestinal (GI) tract, and genitourinary tract. The infectious agent can gain access to the CNS and cause meningeal disease by three major pathways:

1. Invasion into the bloodstream then getting into the CNS; this is the most common mode.

2. A retrograde neuronal pathway: olfactory and peripheral nerves.

3. Direct contiguous spread: sinusitis, otitis media, trauma, congenital malformations.

B. The incubation period is variable, depending on the pathogen/type of meningitis.

PREDISPOSING FACTORS

A. Peak demographics:
1. Adolescents.
2. Freshman college students living in dormitories.

B. Attendance in the military.

C. Sequela of Lyme disease (LD).

D. Odontogenic infection.

E. Sequela of otitis media, bacterial sinusitis, *H. influenzae* type B infection, and varicella.

F. Sickle cell disease, asplenia, Hodgkin disease, and antibody deficiencies.

G. Review history for STI and HIV infection.

H. Penetrating wound, head trauma, spinal tap, surgery, or anatomic abnormality.

I. Occupational exposure such as laboratory personnel.

J. Travel exposure.

K. Maternal infection and fever at the time of delivery.

L. Lumbar epidural steroid injections.

M. Immunosuppression.

COMMON COMPLAINTS

A. Only about 44% of adults with bacterial meningitis exhibit the classic triad. Symptoms can develop over several hours to 1 to 2 days.

1. Classic triad:
   a. Nuchal rigidity.
   b. Fever.
   c. Altered mental status (confusion).

OTHER SIGNS AND SYMPTOMS

A. Sudden onset of a severe, constant headache affecting the entire head that worsens with movement.

B. CNS symptoms (nuchal rigidity, nausea and/or vomiting, confusion, lethargy, decreased level of consciousness [LOC]).

C. Fever or chills.

D. Backache.

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E. Photophobia.
F. Difficulty swallowing.
G. Facial and eye weakness and sagging eyelids.
H. Seizures.
I. Rash: The type of rash—macular, maculopapular, petechial, or purpuric—is dependent on the virus.
J. Anorexia.
K. Nausea/vomiting.
L. Chronic meningitis: usually have subacute onset of symptoms, including fever, headache, and vomiting.

SUBJECTIVE DATA

A. Review the onset, course, and duration of symptoms, including a progressive petechial or ecchymotic rash.
B. Determine current or recent history of ear infections, upper respiratory infection (URI), sinus infection, and chickenpox exposure.
C. Ask the client about any recent dental procedures, extractions, and gum procedures.
D. Review the client's recent history of tick bite and any treatments.
E. Review the client's recent history of Hib immunization.
F. Evaluate a history of serious drug allergies.
G. Evaluate a history of recent head trauma/fracture.
H. Evaluate full history for ventriculoperitoneal shunt and other cranial surgery/procedures.
I. Review history for use of lumbar epidural steroid injections for pain.
J. Review all medications, including over-the-counter (OTC) and herbal products. Determine recent use of antibiotics.
K. Review for a history of illicit drug use, especially the intravenous (IV) route.
L. Review any recent travel locations.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.
B. Inspect:
1. Observe general overall appearance.
2. Examine the skin for the presence of a petechial or ecchymotic rash.
3. Complete an ear, nose, and throat exam.
4. Examine mouth and teeth for dental diseases and disorders.
5. Assess the client for dehydration.
6. Observe the client for seizure activity.
7. Assess level of pain.
8. Inspect for cranial nerve palsies.
C. Auscultate:
1. Auscultate heart and lungs; monitor breathing pattern.
D. Palpate:
1. Neck: Palpate the lymph nodes.
2. Palpate the mastoid bones.
3. Abdominal examination: hepatosplenomegaly.
E. Neurologic exam:
1. Perform a complete neurologic examination.
2. Evaluate for signs of meningeal irritation by means of Brudzinski and Kernig signs (see Figures 19.1 and 19.2). Positive Brudzinski and Kernig signs:
a. Brudzinski sign: Place the client supine and flex the head upward. Resulting flexion of both hips, knees, and ankles with neck flexion indicates meningeal irritation.
b. Kernig sign: Place the client supine. Keeping one leg straight, flex the other hip and knee to a bent knee to form a 90° angle. Slowly extend the lower leg. This places a stretch on the meninges, resulting in pain and spasm of the hamstring muscle. Resistance to further extension can be felt.

DIAGNOSTIC TESTS

A. Bacterial meningitis must be the foremost consideration in the differential diagnosis. In acute meningitis, a lumbar puncture (LP) and CSF exam should be done to identify the causative organism and, especially with bacterial meningitis, the antibiotics sensitivities.
B. LP to obtain CSF for analysis:
1. Opening pressure should be measured.
2. Cell count.
3. Gram stain and cultures, sensitivity.
4. White blood cells (WBCs) and absolute neutrophil count.
5. CSF protein and glucose.
C. Laboratory tests:
1. Complete blood count (CBC) with differential and platelets.
2. Complete metabolic panel (CMP).
3. Coagulation profile.
4. Blood cultures × 2.
D. Cultures of petechial or purpuric lesion scraping and synovial fluid.
E. MRI or CT scan, with and without contrast (a screening CT is not necessary in the majority of the clients).

DIFFERENTIAL DIAGNOSES

A. Bacterial meningitis must be the foremost consideration in clients with fever, headache, neck stiffness, and altered mental status.
B. Meningitis:
1. Bacterial etiology.
2. Viral etiology.
3. Fungal etiology.
4. Aseptic meningitis.
5. Chronic meningitis.
C. Brain abscess.
D. Brain neoplasms.
E. Encephalitis.
F. Febrile seizures.
G. LD.
H. Herpes.
I. Delirium tremens.
J. Gonorrhea.
K. Otitis media.
L. Dental abscess.
M. Chickenpox.
N. Sinusitis.
O. Mastoiditis.
P. Subarachnoid hemorrhage.

PLAN

A. General interventions:
1. Treatment for meningitis is initiated in the hospital setting. Delayed initiation of antibiotic treatment in clients with bacterial meningitis is strongly associated with death and poor outcomes. Begin empiric antibiotic coverage according to age and presence of overriding physical conditions. The treatment depends on the organisms. ▶

Treat aggressively because the progression of disease is often rapid. Empiric therapy should be initiated immediately after blood cultures are drawn after the LP is done and CSF has been sent for testing. The choice of agents is based on known predisposing factors. Once pathogen is identified and antimicrobial susceptibilities are known, the antibiotics should be modified for targeted treatment. The antibiotic used should attain adequate levels in the CSF and this depends on the solubility, protein-binding capacity, molecular size, and the client's degree of meningeal inflammation. Antibiotics that provide high CSF levels are penicillin, fluoroquinolones, cephalosporins, and rifampin. Duration of therapy is dependent on the causative pathogen.

2. Guidelines recommend adjunctive dexamethasone with suspected or proven community-acquired bacterial meningitis. First dose of dexamethasone (0.4 mg/kg IV Q12H for 2 days or 0.15 mg/kg Q6H for 4 days) given 15 to 20 minutes with the first dose of antibiotics. If IV antibiotics have already been started, dexamethasone can still be administered up to 4 hours after the start of the first dose. Recommendations also state that dexamethasone should be stopped if the client does not have bacterial meningitis or if the bacterium causing the meningitis is a species other than *H. influenzae* or *S. pneumoniae*. Other experts advise that adjunctive treatment should be continued irrespective of the causative bacterium.

3. Maintain hydration.

4. In addition to standard precautions, droplet precautions are recommended until 24 hours after initiation of effective antimicrobial therapy.

5. Chemoprophylaxis is recommended for people who have had close contact with the client with invasive meningococcal meningitis. Close contacts are defined as household members, childcare center contacts, and anyone directly exposed to oral secretions. Treatment with rifampin 600 mg PO Q12H for 2 days. Alternatives include ceftriaxone 250 mg intramuscularly (IM) for one dose or ciprofloxacin 500 to 750 mg in one dose.

6. Airline travelers with 8 hours of contact, who are seated directly next to an infected person, should receive prophylaxis.

7. Fungal meningitis is treated with a long course of high-dose antifungal medications usually given IV in the hospital setting.

8. Parasitic meningitis treatment is symptomatic relief of headaches rather than for the infection itself.

B. Client teaching:

1. Advise close contacts of the client with meningococcal and *H. influenzae* meningitis that prophylactic treatment may be indicated; they should check with their healthcare providers or the local public health department.

C. Pharmaceutical therapy:

1. Most cases of viral meningitis are benign and self-limiting. Client may only need supportive care, but in certain instances a specific antiviral may be required.

2. Bacterial meningitis, including *H. influenzae*, meningococcal, and staphylococcal meningitis, is a neurologic emergency and initiation of appropriate antibacterial therapy is important to reduce the risk of death. The most common organisms are *S. pneumoniae, N. meningitidis*, less often *H, influenzae* and GBS in healthy adults up to age 60, and *L. monocytogenes meningitis* in individuals at increased risk and in those over the age of 50:

a. Adults (>15 to 50 years) considered otherwise healthy:

i. Cefotaxime: 2 g IV every 4 to 6 hours.

ii. Ceftriaxone: 2 g IV Q12H.

iii. PLUS vancomycin: 750 to 1,000 mg IV Q12H or 10 to 15 mg/kg IV Q12H due to ceftriaxone resistance.

iv. PLUS ampicillin 2 g IV Q4H.

1) **For clients with penicillin or cephalosporin allergy, meropenem may be utilized.**

b. **Further treatment is determined based on culture results.**

3. For clients with disabling disease or alcoholism and age older than 50, the most common organisms are *S. pneumoniae*, coliforms, *H. influenzae, Listeria* species, *Pseudomonas aeruginosa*, and *N. meningitidis.*

a. Cefotaxime: 2 g IV Q4H; or ceftriaxone: 2 g IV Q12H + vancomycin 10 to 15 mg/kg IV Q12H.

b. Vaccinations: See Exhibit 2.2 in Chapter 2 for the CDC recommendations on all immunizations.

FOLLOW-UP

A. Follow-up is dependent on symptoms. Schedule return visit after completion of antibiotics, or 2 to 3 weeks after initial examination.

B. Sequelae associated with meningococcal disease occur in 11% to 19% of clients:

1. Hearing loss.

2. Neurologic disability.

3. Digit or limb amputations.

4. Skin scarring.

C. Bacterial meningitis is a notifiable disease to the local health department. All presumptive, probable, and confirmed cases should be reported.

CONSULTATION/REFERRAL

A. The client needs immediate consultation with a physician or to be sent to the ED if lethargic.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. The American Academy of Pediatrics (AAP) lists the following indications for selective intrapartum chemoprophylaxis in GBS-positive females:

a. Preterm labor and delivery.

b. Preterm rupture of membranes.

c. Rupture of membranes greater than 18 hours before delivery.

d. Intrapartum fever.

e. Multiple gestation.

f. Previous offspring with invasive GBS disease.

2. Maternal: Culture introital or vaginal and anorectal samples for GBS using a selective medium such as Todd Hewitt broth. Vaginal colonization of GBS occurs in 5% to 40% of pregnant clients, of which 40% to 70% transmit GBS to their offspring.

3. The most significant problem associated with pregnancy is exposure of the fetus to GBS in the maternal genital tract. In 2002, the CDC, the AAP, and the American College of Obstetricians and Gynecologists recommended universal perinatal screening for vaginal and rectal GBS colonization at 35 to 37 weeks' gestation.

4. Treat clients with positive urine culture only because recolonization occurs frequently. Antibiotics should be administered to females for preterm labor, premature

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rupture of membranes (including preterm), and positive GBS cultures (past or present).

B. Adults:

1. Immunization with the pneumococcal vaccination has been shown to reduce the incidence of bacterial meningitis.

C. Geriatrics:

1. The overall mortality rate is higher for elderly clients, in part due to the high prevalence of comorbid conditions.

2. Geriatrics at highest risk are those taking glucocorticoids, immunotherapy, radiation, and TNF-alpha antagonist therapy.

3. Research indicates that healthcare-associated meningitis is a life-threatening complication from invasive neurosurgical procedures, and older adults are particularly at higher risk. This may be due to the likelihood of geriatrics of having these procedures in conjunction with their tendency to have multiple comorbidities and age-related CSF abnormalities.

4. *L. monocytogenes* is a foodborne pathogen that is particularly lethal with geriatrics and commonly presents with syndromes of meningitis and septicemia.

5. Geriatric syndrome: Acute delirium requires a through workup to evaluate the potential for meningitis.

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MONONUCLEOSIS (EPSTEIN–BARR)

DEFINITION

A. Infectious mononucleosis is an acute, infectious viral disease caused by human herpesvirus 4 (HHV-4)/Epstein–Barr virus (EBV), with four classic symptoms: fever, pharyngitis, fatigue, and lymphadenopathy. The spread is via intimate contact between susceptible persons and asymptomatic EBV shedders through the passage of bodily fluids, primarily saliva. This virus is rarely spread through blood transfusion and genital secretions.

B. Oral shedding may occur for 6 months after the onset of symptoms prior to its latency phase.

C. Epstein–Barr is not related to chronic fatigue syndrome (CFS); however, the fatigue related to EBV may last several months.

INCIDENCE

A. Antibodies to EBV have been demonstrated in all population groups with a worldwide distribution. Approximately 90% to 95% of adults are EBV-seropositive.

PATHOGENESIS

A. EBV (HHV-4) is the primary agent of infectious mononucleosis. EBV persists asymptomatically for life in nearly all adults and is associated with the development of B-cell lymphomas, T-cell lymphomas, and Hodgkin lymphoma in certain clients.

B. The incubation period is 10 days in young children and 1 to 2 months, with an average of 11 days, in adults; it is communicable during the acute phase, which may be prolonged. Acute symptoms resolve in 1 to 2 weeks. Pharyngeal excretion may persist for up to 18 months following clinical recovery. It is estimated that once infected with EBV, the virus may be intermittently shed in the oropharynx for life.

C. EBV has also been isolated both in the cervix and in male seminal fluid, suggesting the possibility of sexual transmission.

D. EBV has also been noted in breast milk.

PREDISPOSING FACTORS

A. Exposure through oropharyngeal secretions (kiss, cough, shared food).

B. Roommates.

C. Intrafamilial transmission to siblings.

COMMON COMPLAINTS

A. Infectious mononucleosis is characterized by the following four symptoms:

1. Fever.

2. Tonsillar pharyngitis (with exudate, which may possibly have a white, gray-green, or necrotic appearance).

3. Fatigue: may be persistent and severe.

4. Lymphadenopathy (usually posterior cervical chains, typically symmetric).

OTHER SIGNS AND SYMPTOMS

A. Uvular edema, fairly specific finding.

B. Generalized aches.

C. Appetite loss.

D. Headache.

E. Hepatosplenomegaly: Mild hepatitis is encountered in approximately 90% of individuals; splenomegaly is noted in approximately 50%. Jaundice is uncommon.

F. Rash may be present in approximately one-third of pediatric clients and 10% of adults with cytomegalovirus (CMV) mononucleosis, generally occurring within the first week of symptoms:

1. Macular.

2. Papular.

3. Maculopapular can be caused by a large variety of infections and noninfectious agents.

4. Rubelliform.

5. Morbilliform.

6. Scarlatiniform.

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SUBJECTIVE DATA

A. Review signs, symptoms, course, and duration of symptoms (generally symptoms have a gradual onset but may be acute), specifically pharyngitis, fever, fatigue, and lymphadenopathy.
B. Assess the client for recent upper respiratory infection (URI) and sore throat.
C. Inquire about any contact with persons known to have mononucleosis and other infections such as strep infections.
D. Review the client's history for other family members with similar symptoms.
E. Carefully review medications. A mononucleosis syndrome with atypical lymphocytosis can be induced by drugs, including the following:
1. Phenytoin.
2. Carbamazepine.
3. Antibiotics such as isoniazid and minocycline.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure:
B. Inspect:
1. Conduct ear, nose, and throat exam, especially tonsils and palate:
a. Pharynx shows lymphoid hyperplasia, erythema, and edema.
b. Tonsillar exudate is present in about 50% of cases.
c. Tonsillar pillars may touch "kissing tonsils" and may lead to airway compromise.
d. Evaluate for petechiae at the junction of the hard and soft palate.
e. Evaluate for oral hairy leukoplakia (OHL) on the lateral portions of the tongue. OHL appears as white corrugated painless plaques that cannot be scraped from the surface. (EBV-related malignancies as well as HIV may present with OHL.)
C. Auscultate:
1. Auscultate the heart and lungs.
D. Palpate:
1. Palpate the lymph nodes, especially anterior and posterior cervical chains, axilla, and groin. Firm, tender, and mobile lymph nodes are indicative of mononucleosis; lymphadenopathy is usually symmetric and presents in the posterior cervical chain more than the anterior chain.
2. Palpate the abdomen, especially the spleen. Splenomegaly is noted in 50% of cases. Hepatomegaly and tenderness are noted in 10% of cases.
E. Percuss:
1. Percuss the abdomen, especially the spleen area.
F. Neurologic exam:
1. Evaluate for facial nerve palsy or symptoms of meningitis.

DIAGNOSTIC TESTS

A. White blood cell (WBC) count with differential and a heterophile test.
B. Throat swab for rapid strep; if negative, send for culture.
C. Monospot test—heterophile antibody test—may be negative early in course. Positivity increases during the first 6 weeks (may see false-positive results in those with autoimmune disorders and those with acute retroviral syndrome from HIV infection).
D. EBV immunoglobulin M (IgM) and immunoglobulin G (IgG):
1. Viral capsid antigen (VCA).
2. VCA IgM: early infection and disappears within 4 to 6 weeks.
3. VCA IgG: acute phase, peaks 2 to 4 weeks after onset, decreases slightly then persists for the rest of the life.
4. Early antigen (EA): EA IgG in acute phase, peaks 3 to 4 weeks after onset, falls to undetectable levels after 3 to 6 months.
5. EBV nuclear antigen (EBNA) IgG: not seen in acute infection, appears 2 to 4 months after onset of symptoms and persists through life.
E. Liver function tests: Abnormal liver function tests in a client with pharyngitis strongly suggest the diagnosis of infectious mononucleosis. Mild elevation is constant in early EBV; high elevation should suggest viral hepatitis.
F. Abdominal ultrasound if indicated for splenomegaly and to monitor spleen size post splenomegaly.
G. CT of abdomen if splenic rupture a concern.

DIFFERENTIAL DIAGNOSES

A. Mononucleosis.
B. Streptococcal pharyngitis (group A *Streptococcus*).
C. Viral syndrome.
D. Hodgkin disease.
E. Hepatitis.
F. CMV.
G. Secondary syphilis.
H. CFS.
I. Acute HIV.
J. Toxoplasmosis.
K. Adenovirus.
L. Rubella.
M. Acute HIV.
N. HHV-6 (roseola).
O. HHV-7 (herpesvirus).

PLAN

A. General interventions:
1. Supportive care, hydration, and antipyretics.
2. Make certain that the client does not have an upper airway obstruction from enlarged tonsils and lymphoid tissue.
3. Treat concurrent infections.
4. Isolation is not required with good handwashing and prevention of the spread of pharyngeal secretions.
5. Bedrest is unnecessary.
6. There is no commercially available vaccine to prevent EBV infection.
7. Splenic rupture is rare but potentially life-threatening, occurring in one to two cases per thousand. It occurs between the 4th and 21st day of symptomatic illness, but it can be the presenting symptom. Therefore, strenuous physical activity and contact sports are restricted for 3 to 4 weeks and up to 8 weeks in some clients with splenomegaly. The typical manifestations are abdominal pain and/or a falling hematocrit.
B. Pharmaceutical therapy:
1. Antibiotic therapy is reserved for concurrent infections such as streptococcal pharyngitis.
2. Administer analgesics, such as acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs) for fever, body aches, and malaise.
3. Corticosteroids may be considered in the presence of significant inflammation and swelling of the throat. ▶

FOLLOW-UP

A. Examine client every 1 to 2 weeks.

B. Initial monospot test may be negative; repeat in 7 to 10 days.

C. If splenomegaly is present, schedule an appointment to reevaluate the client prior to the release for contact sports. Splenomegaly puts clients at risk for rupture secondary to blunt trauma (e.g., sports and motor vehicle accidents).

D. Clients with the classic four symptoms of mononucleosis should also have a diagnostic test for strep because the presenting symptoms are so similar.

EMERGENT ISSUES/INSTRUCTIONS

A. Severe pain in the upper left abdomen is considered a medical emergency to evaluate a ruptured spleen.

B. *See Client Teaching Guide for this chapter, "Mononucleosis":*

1. Prolonged communicability may persist for up to 1 year.

CONSULTATION/REFERRAL

A. Consult a physician for marked tonsil enlargement and difficulty swallowing or symptoms lasting longer than 2 weeks. An emergent consultation with an otolaryngologist may be required.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Intrauterine infection with EBV is rare.

B. Geriatrics:

1. Typically, older clients do not always present with symptoms of sore throat, tender adenopathy, or leukocytosis.

2. The sexually active older adult population has an increased risk of exposure to EBV. However, common complaints from this population include tired, right-sided tenderness, myalgias, and occasionally slightly jaundice. Consider an EBV workup to rule out mononucleosis.

3. Studies revealed that clients receiving immunosuppressive therapy/medications for rheumatic conditions were susceptible to EBV, inducing lymphoid proliferations (B-cell lymphoma, Hodgkin lymphoma, and other variants). Recommendations are to evaluate older adults taking immunosuppressive agents for EBV and further explore other diagnostic measures specific to clinical manifestations.

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RHEUMATIC FEVER

DEFINITION

A. Rheumatic fever (RF) is an autoimmune inflammatory process that occurs as sequelae of a group A beta-hemolytic streptococcal (GAS) tonsillopharyngitis, with initial symptoms occurring 2 to 3 weeks after acute infection. RF is a preventable disease through detection and adequate treatment of streptococcal pharyngitis. It is characterized by nonsuppurative inflammatory lesions of the heart, joints, central nervous system (CNS), and subcutaneous tissue. RF is a clinical syndrome with no specific diagnostic test and varying manifestations, making a definitive diagnosis difficult. It consists of exudate and proliferative inflammatory lesions found in connective tissue involving the joints, blood vessels, heart, and subcutaneous tissue:

1. Revised Jones criteria, low-risk populations:

a. Major criteria: carditis (clinical and/or subclinical), arthritis (polyarthritis), chorea, erythema marginatum, and subcutaneous nodules.

b. Minor criteria: polyarthralgia, fever (≥101.3°F), sedimentation rate ≥60 mm and/or C-reactive protein (CRP) ≥3.0 mg/dL, and prolonged release (PR) interval (unless carditis is a major criterion).

2. Revised Jones criteria, moderate- and high-risk populations:

a. Major criteria: carditis (clinical and/or subclinical), arthritis (monopolyarthritis or polyarthritis, or polyarthralgia), chorea, erythema marginatum, and subcutaneous nodules.

b. Minor criteria: fever (≥101.3°F), sedimentation rate ≥30 mm and/or CRP ≥3.0 mg/dL, and prolonged PR interval (unless carditis is a major criterion).

3. Acute rheumatic fever (ARF) diagnosis (initial episode):

a. The diagnosis of an initial episode of ARF requires two major criteria, or one major plus two minor criteria.

4. ARF diagnosis (subsequent episode): Clients with a history of ARF or rheumatic heart disease (RHD) are at high risk for recurrent attacks if reinfected with group A streptococci:

a. With reliable history of ARF or established RHD, and in the face of documented group A streptococcal infection, two major, one major and two minor, or three minor manifestations may be sufficient for presumptive diagnosis.

b. When minor manifestations alone are present, the exclusion of other more likely causes of the clinical presentation is recommended before a diagnosis of an ARF recurrence is made.

c. The most significant complication of ARF is RHD, which occurs after repeated bouts of acute illness.

B. RF is the predominant cause of mitral stenosis.

INCIDENCE

A. RF has markedly decreased in the past 50 years and remains rare in the United States. However, a higher number of cases in Hawaii and American Samoa is seen. In developing countries, RF is enormous. It has been suggested that 15.6 million people worldwide have RF, with 470,000 new cases occurring annually.

1. About 20% diagnosed with RF have a positive history of pharyngitis or recall any upper respiratory symptoms within the preceding 3 months.

2. Ages: Most common age is 5 to 15 years, rarely in infants, preschoolers, and after the age of 35 years.

3. RF does not have a definite sexual predication, but certain manifestations are more common in females during puberty.

B. Cardiac involvement is the most serious complication and causes significant morbidity due to valvular fibrosis leading to stenosis and/or insufficiency. Of the 470,000 clients diagnosed with RF, 60% eventually will develop carditis. Morbidity due to congestive heart failure (CHF), strokes, and endocarditis is common among individuals with RHD, and about 1.5% of persons with rheumatic carditis die of the disease annually. Mitral stenosis and Sydenham chorea are more common in females who have gone through puberty. People who have had a previous attack of RF are at high risk for a recurrent attack, which worsens the damage to the heart.

PATHOGENESIS

A. RF is caused by a preceding infection with GAS and *Streptococcus pyogenes.* Nonsuppurative inflammatory lesions of the joints, heart, subcutaneous tissue, and CNS characterize ARF. The incubation period is between 1 and 5 weeks and 6 months after a group A strep pharyngitis.

PREDISPOSING FACTORS

A. Group A pharyngitis, untreated or inadequately treated.
B. Most common in developing countries, crowding, and poverty.
C. Sex: more common in females, 5 to 15 years.

COMMON COMPLAINTS

A. Sore throat.
B. Polyarthritis: involves large joints such as knees, ankles, elbows, and wrists.
C. Fever.
D. Abdominal pain.

OTHER SIGNS AND SYMPTOMS

A. Fatigue.
B. Appetite loss.
C. Sydenham chorea (more common in females).
D. Erythema marginatum.
E. Enlarged lymph nodes.
F. Carditis: development of new heart murmurs, cardiomegaly, and CHF.
G. Pericarditis, pericardial friction rub, and/or pericardial effusion.

SUBJECTIVE DATA

A. Review recent history:
  1. Sore throat and the onset, duration, severity, and treatment of symptoms.
B. Complete a drug history.
C. Prescribed antibiotics. Does the client take aspirin? **Use of aspirin can mask signs of inflammation and tends to prolong the course of the disease.**
D. Assess the client for signs and symptoms of rheumatic and scarlet fever.
E. Discuss the client's history of heart problems, chest pain, or shortness of breath.
F. Evaluate the onset and complaints of chorea: fidgety, clumsy, uncoordinated erratic facial movements, including grimaces, grins, and frowns. Evaluate tongue movements. Ask if the movements and other symptoms disappear with sleep.
G. Review symptoms of joint pain.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.
B. Inspect:
  1. Inspect joints for swelling and warmth.
  2. Observe for signs of chorea (symptoms noted earlier).
  3. Conduct a dermal exam, especially the trunk and proximal aspects of the extremities. Individual lesions of erythema marginatum are evanescent, moving over the skin in wavy patterns or with indented margins. The lesions may be macular and can develop and disappear in minutes, appearing to change shape while being examined.
  4. Complete an ear, nose, oral, and throat exam. Evaluate tonsillopharyngeal erythema with or without exudates. Observe for beefy, red, swollen uvula.
C. Auscultate:
  1. Auscultate heart. Note heart murmur, pericardial friction rub, or effusion. Characteristic murmurs of acute carditis include the high-pitched, blowing, holosystolic, apical murmur of mitral regurgitation and a high-pitched, decrescendo, diastolic murmur of aortic regurgitation heard in the aortic area. The features of CHF include tachycardia, a third heart sound, rales, and edema.
  2. Auscultate all lung fields. Note shortness of breath.
D. Palpate:
  1. Palpate the neck lymph nodes.
  2. Palpate the extremities: apical and radial pulses.
  3. Palpate the abdomen.
E. Neuromuscular examination:
  1. Have the client stick out their tongue for observation of a "bag of worms" when protruded.
  2. Have the client grip your hand; with chorea, they will be unable to maintain a grip; rhythmical squeezing results.
  3. Observe for the spooning sign: a flexion at the wrist with finger extension when the hand is extended.
  4. Observe for the pronator sign; the palms turn outward when held above the head.

DIAGNOSTIC TESTS

A. No single specific laboratory test can confirm the diagnosis of RF. The throat culture remains the criterion for confirmation of group A streptococcal infection.
  1. If a rapid antigen detection test is negative, obtain a throat culture.
  2. Because of the high specificity, a positive rapid antigen test confirms a streptococcal infection.
  3. Antistreptolysin O (ASO) titer is used to detect streptococcal antibodies against streptococcal lysin O. Elevated titer is proof of previous strep infections.
  4. Erythrocyte sedimentation rate (ESR) is usually elevated at the onset of RF.
  5. CRP is usually elevated at the onset of RF.
  6. Blood cultures to rule out infective endocarditis, bacteremia, and disseminated gonococcal infection.
  7. EKG: Note **prolonged PR interval.**
  8. Chest x-ray: Look for CHF and cardiomegaly.
  9. Echocardiogram may demonstrate valvular regurgitant lesions.
  10. Labs: Tests that may rule out differential diagnoses include rheumatoid factor (RF), antinuclear antibody (ANA), Lyme serology, and evaluation for gonorrhea.

DIFFERENTIAL DIAGNOSES

A. RF (Jones criteria).
B. Juvenile rheumatoid arthritis.
C. Rheumatoid arthritis.

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D. Gonococcal arthritis.
E. Septic arthritis.
F. Reactive arthritis.
G. Sickle cell anemia.
H. Infective endocarditis.
I. Lyme disease (LD).
J. Mixed connective tissue disease.
K. Systemic lupus erythematous.
L. Huntington chorea.

PLAN

A. General interventions:

1. Bedrest is a traditional part of RF therapy. It is important with carditis. Bedrest is needed throughout the acute illness and should continue until the ESR has returned to normal for 2 weeks.

B. Client teaching:

1. Reinforce the need to take the complete prescribed course of antibiotics for strep infections.

2. Adequate treatment for a streptococcal pharyngitis or skin infection is the best prevention against RF. Strep infections are contagious, but RF is not.

3. Chorea is usually managed conservatively in a quiet nonstimulatory environment. Carbamazepine is the primary option for anticonvulsant followed by valproic acid due to liver toxicity potential associated with valproic acid. Although there is no conclusive evidence of their efficacy, intravenous immunoglobulin (IVIG), steroids, and plasmapheresis have all been used successfully in refractory chorea.

4. Diuretics and angiotensin-converting enzyme inhibitors (ACEI) are the mainstay of carditis/heart failure.

5. RF with arthritis is managed with a nonsteroidal anti-inflammatory drug (NSAID), naproxen: adults 250 to 500 mg BID or ibuprofen adults 400 to 800 mg Q8H.

C. Pharmaceutical therapy:

1. Antibiotic treatment in clients who present with ARF is necessary regardless of the throat culture. The drug of choice is penicillin, but erythromycin or sulfadiazine may be used in clients who are allergic to penicillin. Primary prophylaxis reduces the risk of RF and is given when group A streptococcal pharyngitis is confirmed.

a. RF with carditis and clinically significant residual heart disease may require antibiotic treatment for a minimum of 10 years after the last episode; prophylaxis is required until the client is aged at least 40 to 45 years and is often continued for life.

b. RF with carditis and no residual heart disease aside from mild mitral regurgitation requires antibiotic treatment for 10 years or until 25 years of age (whichever is longer).

c. RF without carditis aside from mild mitral regurgitation requires antibiotic treatment for 5 years or until 18 to 21 years of age (whichever is longer).

i. Benzathine penicillin G (Bicillin L-A):

1) Primary prophylaxis for treatment of group A streptococcal pharyngitis: 1.2 million units intramuscular (IM) in a single dose.

2) Secondary prevention is 1.2 million units IM every 4 weeks.

ii. Penicillin VK, for strep:

1) Adults: 500 mg PO Q12H for 10 days.

iii. Amoxicillin:

1) Adults: 875 mg orally in two divided doses for 10 days.

iv. Erythromycin base:

1) Adults: 250 to 500 mg orally QID for 10 days.

v. Azithromycin:

1) Adults: 500 mg orally once a day for 5 days.

vi. Sulfadiazine:

1) Initial load is 2 to 4 g PO.

2) Maintenance 2 to 4 g/d divided three to six times per day.

3) Prophylaxis for recurrent RF:

a) Greater than 30 kg: 1 g/d.

b) Less than 30 kg: 500 mg/d.

2. Salicylates, or NSAIDs, and corticosteroids are the mainstay of anti-inflammatory treatment of RF.

a. **Avoid anti-inflammatory drugs until the diagnosis is confirmed, as they mask symptoms essential to the diagnosis; give acetaminophen in the interim**. Codeine is the analgesic of choice for mild arthritis symptoms.

i. Naproxen 250 to 500 mg BID to a maximum of 1,250 mg in adults.

ii. Aspirin is used in clients with moderate to severe arthritis and carditis without heart failure. 325 to 650 mg PO every 4 to 6 hours as needed. Enteric coated: 650 to 1,300 mg PO Q8H; not to exceed 3.9 g/d.

iii. If tolerated, anti-inflammatory therapy should be continued until all joint symptoms have resolved. Most clients require treatment for 1 to 2 weeks; treatment up to 8 weeks may be necessary.

iv. Prednisone is used in severe carditis and heart failure. High-dose prednisone is administered for 2 to 3 weeks, then tapered. Intravenous (IV) corticosteroids are reserved for fulminate cases.

FOLLOW-UP

A. The client should be closely observed until all acute symptoms are resolved and there is return to normal health. Most can be treated as outpatient.

B. Household contacts should have throat cultures performed. Those with positive results should also receive a full course of antibiotic therapy, even if asymptomatic.

C. In patient treatment may be needed in clients with severe constitutional symptoms, chorea, or carditis with CHF.

D. Clinical or laboratory manifestation of rheumatic inflammation may recur upon cessation of anti-inflammatory therapy.

E. Gradual tapering of corticosteroids is required due to rebound.

F. Salicylates are usually continued for 1 month following corticosteroid discontinuance.

G. Clients with carditis should be followed every 6 months by a cardiologist, with an echocardiography every 1 to 2 years.

EMERGENT ISSUES/INSTRUCTIONS

A. Pancarditis that causes CHF, heart blocks, or pericardial effusion needs immediate inpatient care and cardiology consults.

CONSULTATION/REFERRAL

A. Consultation with ENT (ear, nose, and throat) specialist for clients with intact tonsils and recurrent symptomatic strep infections for consideration of tonsillectomy. ▶

B. Consultation with a cardiologist to manage heart blocks and CHF.
C. Consultation with a neurologist to confirm the diagnosis of chorea.

INDIVIDUAL CONSIDERATIONS

A. Adults:
1. Cases are rare for persons older than age 40.
B. Pregnancy:
1. Penicillin G is pregnancy category B. Fetal risk is not confirmed in humans but has been shown in some studies in animals.
2. Erythromycin is pregnancy category B.
3. Aspirin products are pregnancy category D. As fetal risk has been shown in humans, use only if the benefits outweigh the risk to the fetus.
C. Geriatrics:
1. Mitral stenosis caused by RHD builds mitral fibrotic/calcifications of the chordae tendineae over time. Studies indicate that elderly persons ≥65 years of age are not considered candidates for percutaneous commissurotomy due to the extent of degenerative changes to the valvular structure. Best medical practice guidelines suggest that palliative treatment is most suitable for this population. However, for older adults insistent to have the procedure, refer them to a cardiologist for further evaluation and shared decisions.
2. Studies reveal that clients receiving immunosuppressive therapy/medications for rheumatic conditions are susceptible to Epstein–Barr virus (EBV), inducing lymphoid proliferations (B-cell lymphoma, Hodgkin lymphoma, and other variants). Recommendations are to evaluate geriatrics taking immunosuppressive agents for EBV and further explore other diagnostic measures specific to clinical manifestations.
3. Rehabilitation for geriatric population with rheumatic diseases and/or receiving palliative care secondary to rheumatic conditions may seem challenging because of managing multiple comorbidities. Research indicates that an effective approach was to implement the following priorities: pain relief, restoration of joint function, maintenance of independence, and preservation of energy and quality of life (specifically for geriatrics who have experienced a decrease in physical/social activities secondary to rheumatic heart conditions).
4. Involve families and surrogates in providing fundamental goals for restoration that promote the geriatric client's independent activities. This could include fostering the use of necessary technical aids, engaging in exercises, and giving assistance with translation communication to meet personal needs for those who suffer from cognitive/expressive impairment.

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ROCKY MOUNTAIN SPOTTED FEVER

DEFINITION

A. Rocky Mountain spotted fever (RMSF) is a serious bacterial tickborne disease that can be deadly if not treated early. The cause is the organism *Rickettsia rickettsii* and is spread by several species of ticks in the United States, with the most common tick being *Dermacentor variabilis* (American dog tick). The organism is endemic in parts of North, Central, and South America, especially in the southeastern and south. RMSF has been described as a "wolf in sheep's clothing" and "the great imitator" of other disease processes. The hallmark is a petechial rash beginning on the palms of the hands and soles of the feet.
B. Although its clinical manifestations and treatment are well known, RMSF still causes significant mortality and morbidity. The current mortality rate is 1.4%, mostly due to delay in diagnosis and treatment. In the United States, RMSF is a reportable disease. Mortality rate in untreated cases is 20% to 25%. Mortality rates can be as low as 5% with proper antibiotic therapy and as high as 70% in untreated elderly people.

INCIDENCE

A. RMSF is the most common cause of fatal tickborne illness in the United States. Although RMSF is more common in rural and suburban locations, it does occur in urban areas. The highest incidences are in the southeastern U.S. tick belt (the South Central region) and selected areas of the Northeast. In the northern United States, infections commonly occur in the spring. However, in the South, cases may occur at any time of the year, including winter.
B. People of all ages can be infected. Caucasians have twice the incidence of African Americans. African Americans have a higher case-fatality rate, possibly due to the difficulty of distinguishing the rash in highly pigmented skin.
C. The incidence of RMSF is highest among adults aged 60 to 69 years (3.1 cases/million persons) and children aged 5 to 9 (estimated 3.3 cases/million persons).
D. RMSF is the only tickborne disease that can directly cause heart failure secondary to myocarditis.

PATHOGENESIS

A. *R. rickettsii* is the infectious agent and is transmitted by a tick vector. *R. rickettsii* is a small, gram-negative, obligate, intracellular coccobacillus. The organism is spread through the body via blood and the lymphatic system. The bacteria also infects rodents, squirrels, and chipmunks. Up to one-third of clients with proven RMSF do not recall a recent tick bite or tick contact. RMSF is not transmitted by person-to-person contact.
B. Ticks can become infected by transmission from an infected animal during feeding or from mother to offspring. Infection can occur through contact with tissue and fluids from the tick. The incubation period ranges from 2 to 12 days after either the ▶

tick bite or contamination, but usually occurs around 1 week or 7 days and depends on the amount of contamination from the tick.

C. Principal recognized vectors:
- **1.** *D. variabilis* (American dog tick).
- **2.** *D. andersoni* (Rocky Mountain wood tick).
- **3.** *Amblyomma americanum* (Lone Star tick).
- **4.** *Rhipicephalus sanguineus* (brown dog tick).

PREDISPOSING FACTORS

A. Outdoor activities (hunting, hiking, camping).

B. Tick bite: The tick must attach and feed for 6 to 10 hours before transmitting the infection.

C. Age is not a predisposing factor, but the disease is more common in children and young adults.

D. Exposure to heavy brush areas.

E. Contact with dogs and other animals with ticks.

F. Transmission has occurred on rare occasion by blood transfusion.

COMMON COMPLAINTS

A. In the early phase, most clients have nonspecific signs and symptoms. The classic triad of fever, headache, and rash may be present in less than 5% in the first 3 days but increases to 60% to 70% by the second week after tick exposure:
- **1.** Fever (>102°F).
- **2.** Headache: frequently severe.
- **3.** Myalgias.
- **4.** Rash: Rash usually (90%) occurs between 3 and 5 days of illness. The typical RMSF rash begins as a pink maculopapular eruption on the ankles and wrists. The rash then spreads both centrally and to the palms of the hands and soles of the feet. By the fourth day, the rash spreads centripetally and becomes petechial and papular. Hemorrhagic, ulcerated lesions may follow. In a small percentage, the onset of rash is delayed (past 5 days) and/or is atypical (e.g., confined to one body region). Urticaria and pruritus are not characteristic of RMSF, and their presence makes the diagnosis unlikely.

OTHER SIGNS AND SYMPTOMS

A. Periorbital edema: key finding in children.

B. Deep nonproductive cough at the time of the rash.

C. Edema.

D. Bleeding.

E. Conjunctivitis with or without photophobia.

F. Retinal abnormalities.

G. EKG abnormalities.

H. Seizures.

I. Dehydration.

J. Malaise.

K. Nausea without vomiting.

L. Altered mental status.

POTENTIAL COMPLICATIONS

A. Disseminated intravascular coagulation (DIC).

B. Noncardiogenic pulmonary edema.

C. Acute renal failure (ARF).

D. Myocarditis/heart failure: usually a cause of death.

E. Skin necrosis and gangrene (fingers, toes, elbows, ears, and scrotum).

F. Seizures.

G. Encephalopathy.

H. Peripheral neuropathy.

I. Hearing loss.

J. Cerebellar and vestibular dysfunction.

K. Bowel and bladder incontinence.

SUBJECTIVE DATA

A. Review the onset, course, and duration of symptoms.

B. Elicit information about a recent tick bite or removal.

C. Ask the client about any recent outdoor activities, such as camping, hiking, gardening, or other activities and travel, especially to endemic areas. Less than half of the people infected remember a tick bite.

D. Rule out similar symptoms in other family members.

E. Review thorough history of medications.

F. Review any history of rash and the course of spread.

G. Elicit a history of mental or neurologic changes, including seizures.

H. Rule out other symptoms such as arthritis, memory loss, and distal paresthesia.

I. Review the client's recent history of blood transfusion.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure. Elevated temperature (fever >102°F) is the most common symptom.

B. Inspect:
- **1.** Conduct an ear, nose, and throat exam.
- **2.** Inspect the skin, especially on the wrist, palms, ankles, and soles of the feet. The face is generally spared from the rash. Rash is a major diagnostic sign that can appear on the first day of infection. Maculopapular rash appears 2 to 6 days after onset of fever.
- **3.** Note the presence of petechiae. The rash may blanch and appear macular in the early phase.
- **4.** Conduct an eye examination; evaluate periorbital edema and petechial conjunctivitis.

C. Auscultate:
- **1.** Perform complete heart evaluation.
- **2.** Auscultate all lung fields.

D. Palpate:
- **1.** Palpate all lymph nodes.
- **2.** Palpate the mastoid bones.

E. Neurologic examination:
- **1.** Assess level of consciousness (LOC).
- **2. Evaluate the client for signs of meningeal irritation, such as nuchal rigidity and positive Brudzinski and Kernig signs (see Figures 19.1 and 19.2).**

DIAGNOSTIC TESTS

A. Indirect immunofluorescence antibody (IFA): antibody titers: A fourfold rise in antibody titer is the diagnostic gold standard for RMSF:
- **1.** Antibodies typically appear 7 to 10 days after the onset of the illness.
- **2.** 85% will not have detectable antibody titers during first week of illness.

B. Complete blood count (CBC) with differential. Platelet count: As the illness progresses, thrombocytopenia becomes more prevalent and may be severe. White blood cell (WBC) can be normal.

C. Coagulation panel: prothrombin time (PT)/partial thromboplastin time (PTT)/international normalized ratio (INR).

D. Chest x-ray.

E. EKG.

F. Comprehensive metabolic panel: low sodium, elevated blood urea nitrogen.

G. Liver function studies mildly elevated.

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H. Bilirubin.
I. Skin biopsy: 3-mm punch biopsy.
J. Lumbar puncture may be indicated if there is suspected meningitis and/or other neurologic signs.
K. Rickettsial blood cultures are highly sensitive and specific; however, they require specialized laboratories.

DIFFERENTIAL DIAGNOSES

A. RMSF.
B. RMSF is commonly mistaken for an undifferentiated viral illness during the first few days of illness. Consider RMSF if other signs and symptoms support a diagnosis, even if a rash is not present.
C. Viral meningitis.
D. Ehrlichiosis.
E. Tularemia.
F. Anaplasmosis.
G. Lyme disease (LD).
H. Mononucleosis.
I. Atypical measles.
J. Viral hepatitis.
K. Parvovirus B19 (fifth disease).

PLAN

A. General interventions:
1. Early treatment is necessary; never delay initiation of antimicrobial treatment to confirm clinical suspicion of the disease. This is a life-threatening disease. Antibiotics are less likely to prevent fatal outcome from RMSF if started after day 5 of symptoms. Close monitoring is necessary.
2. Antibiotic therapy (see "Pharmaceutical Therapy" of this section). If penicillin or a cephalosporin is administered empirically in the first few days of the illness, the subsequent rash may be incorrectly diagnosed as a drug reaction.
3. Hospitalization should be considered for most clients. Mild cases may be treated as outpatient. RMSF can progress rapidly.
B. Client teaching: *See Client Teaching Guide for this chapter, "Rocky Mountain Spotted Fever and Removal of a Tick."*
1. RMSF is not transmissible by person-to-person contact; therefore, isolation is not necessary.
2. Relapse of the illness may occur; the client should report recurrence of symptoms immediately.
3. Clients who report tick bites should be advised to inform their healthcare provider if any systemic symptoms, especially fever and headache, occur in the following 14 days.
4. All pets should be treated for ticks.
C. Pharmaceutical therapy:
1. Drug of choice for adults: doxycycline 100 mg orally or intravenously (IV) Q12H for 5 to 7 days.
2. Clients younger than 8 years: Doxycycline is not recommended for mild to moderate infection:
a. Older than 8 years and less than 45 kg: loading dose 4.4 mg/kg/d PO/IV BID for 1 day.
b. Maintenance dose 2.2 to 4.4 mg/kg/d PO/IV BID.
i. Clients should be treated for at least 3 days after fever subsides and until there is evidence of clinical improvement. Minimum course of therapy is 5 to 7 days.
c. If pregnant or allergic to doxycycline: chloramphenicol.
d. Most experts consider the risk of morbidity from rickettsia diseases greater than the minimal risk of dental staining from one short course of doxycycline.
3. Alternative drug therapy: chloramphenicol (Chloromycetin):
a. Chloramphenicol requires frequent serum platelet counts and CBCs.
b. Not available in oral form in the United States.
c. Use of chloramphenicol is associated with a higher risk of fatal outcome and persons treated with it have a greater risk of death compared with doxycycline.
4. Prophylactic therapy with doxycycline or another tetracycline is not recommended following tick exposure.

FOLLOW-UP

A. See the client 24 to 48 hours after initial visit and again at the end of antibiotic therapy (unless following lab for chloramphenicol therapy). RMSF progresses rapidly. Approximately 10% of outpatients are subsequently admitted to the hospital.
B. Several rickettsial diseases, including RMSF, are nationally notifiable diseases and should be reported to state and local health departments.

EMERGENT ISSUES/INSTRUCTION

A. The client must be seen for any alteration in mental status, stiff neck, severe headaches, nausea and vomiting, severe weakness, dizziness, or high fever.
B. Hospitalization is indicated in clients who are severely ill or have complications, such as seizures, hypotension, or marked gastrointestinal (GI) symptoms.

CONSULTATION/REFERRAL

A. Consult a physician for any suspected signs of RMSF because the client is in danger of vascular collapse and disseminated intravascular collapse.
B. Consultation with an infectious disease expert is advised.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Tetracyclines should not be used in pregnancy.
B. Geriatrics:
1. If older adult clients have dexterity challenges, encourage them to return to the clinic for assistance in removing ticks.
2. In older clients and in severe or fatal cases of RMSF, the rash tends to appear later and with less frequency.
3. Age and thrombocytopenia at presentation have been independently associated with the development of ARF.
4. ARF development increases the odds ratio of dying by a factor of 17.
5. Geriatric syndrome assessments:
a. Delirium is often a predominating symptom in the geriatric population. Obtain a full history for delirious clients and do not immediately assume the cause is from a transient ischemic attack (TIA), urinary tract infection (UTI), or age-related cognitive changes. Clues for RMSF during history evaluations would be outside activities, travel, fishing, and pet owners.

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TOXOPLASMOSIS

DEFINITION

A. Toxoplasmosis is caused by an intracellular protozoan parasite, *Toxoplasma gondii.* Cats are the primary host in which *T. gondii* can complete its reproductive cycle. Humans are the intermediate host. Toxoplasmosis is acquired through contact with infected cat feces (cleaning a cat's litter box), by eating raw or undercooked meat (usually pork, lamb, goat, or wild game meat), by gardening, or by eating soil-contaminated fruit or vegetables. In less developed countries, contaminated unfiltered water is an important source of infection. Once ingested by humans or any other warm-blooded animal, the parasite transforms into a tissue-infective stage in the intestine, migrates through the intestinal wall, and is carried via the blood to other tissues, including the central nervous system (CNS). Mother-to-child transmission typically occurs when a pregnant client is newly infected during or just prior to their pregnancy. The organism can also be transmitted when a previously uninfected person receives an organ or blood transfusion from an infected donor.

B. Emphasis should not be placed on prior exposure to cats because clients can acquire *Toxoplasma* without direct contact with felines. Toxoplasmosis is one of the TORCH infections (**T**oxoplasmosis, **O**ther infections including syphilis, Rubella, **C**MV [cytomegalovirus], and Herpes simplex virus [HSV]). Toxoplasmosis causes CNS disease in clients with AIDS and has perinatal consequences. Latent infection can persist for the life of the host.

C. There are three genotypes of *T. gondii:* types I, II, and III. Genotype II is generally responsible for congenital toxoplasmosis in the United States.

D. There is no vaccination available for prevention of toxoplasmosis.

INCIDENCE

A. Toxoplasmosis has a worldwide distribution and is responsible for considerable morbidity and mortality in the United States. Serologic evidence of infection is more frequently seen in Black and Hispanic persons, and among persons who are foreign-born, have low educational level or socioeconomic status, or have occupations involving exposure to soil. There is no association with cat ownership. The incidence of seropositivity in adults ranges from 9% (United States) to 85% (Europe). Adults most commonly acquire toxoplasmosis by environmental exposure, that is, through ingestion of infectious oocysts, usually from soil contamination with feline feces.

B. Once a person is infected, the parasite lies dormant in neural and muscle tissue and will never be eliminated. Studies have now revealed associations between *T. gondii* antibody seropositivity (indicating infection) and the presence of various psychiatric disorders in humans (e.g., schizophrenia, bipolar illness, suicide attempts, episodes of self-directed violence, and memory impairment in elderly persons). Approximately 50% of all pregnant persons in the United States have been previously infected and are immune, whereas those who keep cats as pets have a higher seropositivity rate. Congenital infection can cause pregnancy loss (miscarriage or stillbirth) or severe disease in the newborn, including developmental delays, blindness, and epilepsy. However, many newborns with congenital toxoplasmosis are asymptomatic at birth. Nevertheless, even if asymptomatic at birth, illness will develop in many infected infants later in their life, most often ocular disease but also neurologic symptoms and developmental disabilities.

C. Approximately 30% of clients who acquire the infection during pregnancy transmit the infection to the fetus; the risk of transmission from mother to child is lowest when infection occurs during the first trimester (10%–15%) and higher as pregnancy progresses to the third trimester (60%–90%).

D. In the United States, it is estimated that toxoplasmosis is the second leading cause of death attributable to a foodborne illness. It is also the fourth leading cause of hospitalizations from contaminated food and the leading contributor to loss of quality-adjusted life years. *T. gondii* infects an estimated 1.1 million persons each year in the United States.

E. In clients with late-phase HIV infection and AIDS, toxoplasmosis is the leading cause of focal CNS disease.

PATHOGENESIS

A. *T. gondii* is the causative agent and an intracellular protozoan parasite. It is worldwide in distribution; cats, birds, and domesticated animals serve as reservoirs. *T. gondii* is recognized as a major cause of opportunistic infection in AIDS.

B. The incubation period is estimated to be on average 7 days (4- to 21-day range).

C. Routine screening for toxoplasmosis in pregnancy is not currently recommended.

1. Maternal toxoplasmosis infection is acquired orally.
2. Fetal infection results from transmission of parasites via the placenta (vertical transmission).
3. Neonatal infection may also occur during vaginal delivery.

PREDISPOSING FACTORS

A. Food sources:

1. Eating raw or undercooked meats, especially lamb, goat, pork, and wild game meat.
2. Drinking unpasteurized goat milk.
3. Eating raw shellfish from contaminated waters.

B. Exposure to contaminated soil or gardens, kitty litter, and cats.

C. Immunocompromised state:

1. HIV-infected/AIDS.
2. Cancer therapy.
3. Transplant recipients.
4. Prescribed immunosuppressive drugs.

D. *T. gondii* has been documented as being acquired from blood or blood product transfusion and organ (e.g., heart) or bone marrow transplant from a seropositive donor with latent infection.

E. Poor sanitary conditions.

F. Consumption of contaminated unfiltered water.

G. Occupational exposure:

1. Working with meat.
2. Landscaping.

H. Travel to underdeveloped country.

COMMON COMPLAINTS

A. 80% to 90% of acute *T. gondii* infectious hosts are asymptomatic.

B. Bilateral, symmetrical, nontender cervical adenopathy.

C. 30% of symptomatic clients have generalized lymphadenopathy.

OTHER SIGNS AND SYMPTOMS

A. Usually subclinical infection:

1. Fever.
2. Arthralgia, malaise, and myalgia.

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3. Headache.
4. Sore throat/pharyngitis.
5. Generalized lymphadenopathy.
6. Skin rash: diffuse nonpruritic maculopapular rash.
7. Hepatosplenomegaly.
8. Chorioretinitis: ocular pain and loss of visual acuity (most frequent, permanent manifestation of toxoplasmic infection).

B. Pregnancy:
1. Intrauterine growth restriction (IUGR) or low birth weight.
2. Hydrocephaly.
3. Microcephaly.
4. Anemia.

C. CNS toxoplasmosis: Clinical manifestations are dependent on areas of the brain compromised by infection. Symptoms can progress gradually or abruptly with seizures and motor deficits.
1. Headache, dull and constant, is an almost universal symptom of cerebral lesions in AIDS clients.
2. Fever.
3. Lethargy.
4. Altered mental state.
5. Seizures.
6. Weakness.
7. Hemiparesis.
8. Cranial nerve disturbances.
9. Sensory abnormalities.
10. Movement disorders.
11. Neuropsychiatric manifestations: Toxoplasmosis is a significant factor in the causation of mental retardation and blindness.

SUBJECTIVE DATA

A. Review onset, course, and duration of symptoms.
B. Determine if the client is pregnant.
C. Review presence of indoor cat and contact with cat litter.
D. Rule out other illness with review of symptoms such as pharyngitis (mononucleosis).
E. Elicit initial site of rash and progression to other body areas.
F. Determine any new medications and contact exposures.
G. Rule out other family members with similar symptoms.
H. Review HIV status.
I. Review ingestion of raw/rare or undercooked meats, raw shellfish, and unpasteurized goat's milk.
J. Review recent travel to an underdeveloped country (untreated water).
K. Review occupational exposure.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.
B. Inspect:
1. Conduct ear, nose, and throat exam and careful funduscopic exam.

A funduscopic exam may reveal yellow-white areas of retinitis with fluffy borders. Diagnosis of ocular toxoplasmosis is based on observation of characteristic retinal lesions in conjunction with Toxoplasma-specific serum immunoglobulin G (IgG) or immunoglobulin M (IgM) antibodies.

2. Complete a dermal exam.

C. Auscultate:
1. Auscultate lungs and heart.

D. Palpate:
1. Palpate all lymph nodes, especially cervical nodes. Lymph nodes are usually smaller than 3 cm in size and nonfluctuant.
2. Palpate the abdomen.
3. Palpate the joints.

E. Neurologic exam:
1. Conduct a mental status evaluation.

DIAGNOSTIC TESTS

A. A panel of tests (the *Toxoplasma* serologic profile [TSP]) consisting of the Sabin–Feldman dye test (DT), double-sandwich immunoglobulin M enzyme-linked immunosorbent assay (IgM ELISA), IgA ELISA, IgE ELISA, and differential agglutination (AC/HS), and avidity test. The most commonly used tests for measurement of IgG antibody are DT, ELISA, immunofluorescence assay (IFA), and the modified direct agglutination tests. Polymerase chain reaction (PCR) amplification is used for detection of DNA in body fluids and tissue for diagnosis of congenital, ocular, and cerebral and disseminated toxoplasmosis.
1. DT: high titers; positive IgM, IgA, and IgE ELISA and an acute pattern in the AC/HS test highly suggestive of a recently acquired infection.
2. IgG antibodies: screening for prior exposure; does not indicate severity of illness; usually appears within 1 to 2 weeks of acquisition of the infection, peaks within 1 to 2 months, declines at various rates, and usually persists for life.
3. IgM antibodies: detectable for months after initial infection and can evaluate acute infection, pregnancy and newborns; immunosorbent agglutination assay for newborns is diagnostic of congenital disease.
4. IgE antibodies: detected by ELISA for acutely infected adults, assists in confirming diagnosis of toxoplasmic encephalitis and ocular toxoplasmosis, and detection of congenitally infected infants and children with congenital toxoplasmic chorioretinitis.
5. PCR: often performed on amniotic fluid at 18 weeks' gestation to determine if infant is infected.

B. Complete blood count (CBC) with differential.
C. HIV (rule out).
D. Pregnancy test if indicated.
E. CT scan or MRI (usually superior to CT scan).
F. Emerging tests include IgG/IgM immunoblot tests, enzyme linked immunosorbent spot assay and interferon-gamma release assay tests.
G. Confirmatory testing through the reference laboratory at Palo Alto Medical Foundation Toxoplasma Serology laboratory is advised for diagnosis of acute toxoplasmosis.

DIFFERENTIAL DIAGNOSES

A. Toxoplasmosis.
B. Epstein–Barr virus (EBV).
C. CMV retinitis.
D. Cat-scratch disease (CSD).
E. Tuberculosis.
F. Syphilis.
G. Sarcoidosis.
H. Hodgkin disease.
I. Lymphoma.
J. Viral syndrome.
K. HIV.
L. Mononucleosis.
M. *Pneumocystis carinii* pneumonia.

N. Varicella zoster.
O. Fungal infection of eye.

PLAN

A. Client teaching: *See Client Teaching Guide for this chapter, "Toxoplasmosis."*
1. Handwashing is the single most important measure to reduce transmission of *T. gondii.*
B. Pharmaceutical therapy:
1. Treatment is rarely necessary because most cases of clinical illness resolve spontaneously; the exception is during pregnancy.
2. Treatment is usually given for 2 to 4 weeks if the disease is clinically evident or symptoms are severe or persistent. If ocular lesions are present, the client should be immediately referred to ophthalmology for evaluation and treatment.
a. Nonpregnant adults: one of two regimens are typically prescribed:
i. Pyrimethamine: 200 mg loading dose then 50 mg PO daily; *plus* sulfadiazine 1,000 mg four times a day; *plus* leucovorin calcium (folinic acid) 10 to 25 mg PO daily. Pyrimethamine penetrates the brain parenchyma efficiently even in the absence of inflammation. Leucovorin reduces the likelihood of development of hematologic toxicities associated with pyrimethamine therapy.
ii. Pyrimethamine: 100 mg loading dose, then 25 to 50 mg PO daily; *plus* clindamycin 600 mg PO Q6H; *plus* leucovorin calcium (folinic acid) 10 to 25 mg PO daily. (Pyrimethamine plus clindamycin plus leucovorin is the preferred alternative regimen for clients who cannot tolerate sulfadiazine or do not respond to first-line therapy.)
b. Maternal and fetal: Spiramycin is recommended for the first and early second trimesters or pyrimethamine/sulfadiazine and leucovorin for late second and third trimesters.
c. Persons with compromised immune systems:
i. Pyrimethamine: 200 mg loading dose, then 50 mg PO daily; *plus* sulfadiazine 1,000 mg four times a day; *plus* leucovorin calcium (folinic acid) 10 to 25 mg PO daily.
ii. Trimethoprim sulfamethoxazole PO. May be considered as a non pyrimethamine alternative in clients with AIDS if unable to tolerate usual adult therapy noted earlier.
iii. **Administering dapsone with pyrimethamine appears to provide effective chemoprophylaxis in HIV clients seropositive for *T. gondii* who have CD4 cell counts lower than 200. However, rash, fever, and hemolytic anemia are common adverse effects, often necessitating cessation of therapy.**
iv. For clients with AIDS, after primary therapy, lifelong prophylaxis for recurrence of toxoplasmosis is required for as long as they are immunosuppressed.

FOLLOW-UP

A. Follow up in 1 week to evaluate for secondary complications.
B. Pyrimethamine is a folic acid antagonist that can cause dose-related bone marrow suppression with resultant anemia, leukopenia, and thrombocytopenia. Sulfadiazine is another folic acid antagonist; it works synergistically with pyrimethamine and can cause bone marrow suppression and reversible acute renal failure (ARF). Clients should return for laboratory monitoring (CBC and platelet counts) weekly.

CONSULTATION/REFERRAL

A. Consultation is needed for all clients; comanage with a physician/specialist.
B. Refer the client to an obstetrician if pregnant.
C. Ophthalmologist: Medication recommendation depends on the size of the eye lesion, the location, and the characteristics of the lesion (active acute vs. chronic not progressing).

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Routine prenatal screening is not the standard of care secondary to costs. Many clients have antibodies prior to pregnancy that protect the fetus.
2. Transplacental infection increases the incidence of first-trimester spontaneous abortion, IUGR, preterm birth, neonatal anomalies, and stillbirth.
3. Pyrimethamine is a folic acid antagonist and should not be given in the first trimester.
4. Sulfadiazine should not be given in the third trimester secondary to the increased incidence of jaundice in the neonate.
5. Transmission of toxoplasmosis in breast milk has not been demonstrated. Pyrimethamine is excreted in the breast milk; however, the World Health Organization (WHO) and the American Academy of Pediatrics (AAP) classify it as compatible with breastfeeding.
B. Clients with AIDS:
1. Clinical CNS toxoplasmosis occurs in 3% to 15% of individuals with AIDS in the United States, and in some European countries and Africa the occurrence is 50% to 75% in clients. CNS disease occurs during advanced HIV infections when CD4+ counts are less than 200 cell/µL. The probability of reactivation is as high as 30% among clients with a CD4+ count of less than 100 cells/µL.
2. Standard treatment therapy:
a. Sulfadiazine: 1,000 mg QID for clients less than 60 kg or 1,500 mg QID if greater than 60 kg.
b. Pyrimethamine 200 mg loading dose, then 50 mg daily for clients less than 60 kg or 75 mg greater than 60 kg.
c. Leucovorin should be administered to prevent pyrimethamine-induced toxicity with dosages 10 to 25 mg daily.
d. Corticosteroid treatment may be warranted in cases of impending brain herniation. However, they may complicate the interpretation of response to antitoxoplasmosis therapy.
3. Primary therapy for 6 weeks followed by long-term suppression therapy at reduced doses. Long-term therapy can be stopped if the client has persistent elevation in CD4 counts greater than 200 cells/µL and resolution of lesions on MRI.
C. Geriatrics:
1. Many adults are unaware that parasites and diseases their pets harbor may be transmitted to humans. Remind pet owners to keep routine visits with their veterinarian. Cats are common companions of older adults and are potential sources of minor skin infections to systemic diseases such as toxoplasmosis. Explain to older adults that toxoplasmosis as well as parasites (e.g., roundworm, ringworm, hookworm, giardiasis) are common human illnesses contracted from pet animals. Prevent pet cats from interacting with stray animals and encourage indoor pets.

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2. Animal parasites and diseases could become a serious illness to elderly populations with age-related or medical treatment-related immunosuppression.

3. Continue discussions at annual visits and/or as needed visits to keep geriatrics reminded of preventive measures from pet diseases.

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VARICELLA (CHICKENPOX)

DEFINITION

A. Varicella, commonly called chickenpox, is a viral disease that largely occurs during childhood, with 90% occurring in children younger than 10 years. Chickenpox that affects "healthy" children is a self-limiting illness. Morbidity is associated with adults and immunocompromised individuals. Manifestation is characterized as generalized, pruritic, vesicular rash. Varicella zoster virus (VZV) remains dormant in the sensory nerve roots for life. Adults may have a more complicated course than children with more widespread rash, prolonged fever, and increased risk of complications like varicella pneumonia.

B. VZV (shingles) is the reactivation of the virus and the risk increases with age. The symptoms include pain, vesicular rash, sensory loss, and neurologic complications. Diagnostic clues are sensory symptoms: pain and vesicular rash that do not cross midline. Postherpetic neuralgia (PHN) is a prolonged complication.

C. Pregnant individuals who get VZV are at an increased risk for serious complications. If contracted during the first or early third trimester, the baby has a risk (0.4%–2.0%) of being born with congenital varicella syndrome, scarring on the skin, abnormalities in limbs, brain, and eyes, and low birth weight.

INCIDENCE

A. In 1995, the vaccine for varicella was introduced and since then the incidence has significantly declined. As of November 2016, according to the Centers for Disease Control and Prevention (CDC), VZV causes about four million cases, 10,600 hospitalizations, and 100 to 150 deaths each year.

PATHOGENESIS

A. VZV, herpesvirus 3, is a member of the herpesvirus family. Humans are the only source of infection for this highly contagious virus. Person-to-person transmission occurs primarily by direct contact with a client with varicella or zoster, by airborne droplet, from respiratory secretions, and from direct contact with vesicular zoster lesions. In utero infections also occur from transplacental passage of the virus during maternal varicella infection.

B. The incubation period lasts 10 to 21 days. It is communicable 1 to 2 days to 1 week before onset of macular rash and the client remains infectious for 4 to 5 days after lesions crust over (about 1 week). It takes from 10 to 21 days after exposure for someone to develop varicella.

C. VZV remains dormant in the dorsal root ganglia of the sensory cranial and spinal nerves.

PREDISPOSING FACTORS

A. Exposure to someone with the varicella virus.

1. Direct contact with skin lesions or by respiratory tract secretions.

2. Direct contact with persons with shingles can induce chickenpox in susceptible healthcare workers.

B. Compromised immune system.

COMMON COMPLAINTS

A. The triad of rash, malaise, and low-grade fever can signal disease onset.

1. Skin lesions or rash: Characteristic rash is pruritic, vesicular exanthem occurring in crops that begin on the head and neck and progress to involve the trunk and extremities. Blisters collapse within 24 hours to 1 week and crust over to form scabs. Skin eruptions appear almost anywhere on the body, including the scalp, penis, and inside the mouth, nose, throat, and vagina.

B. Itching.

C. Nausea.

D. Anorexia.

E. Headache.

F. Sore throat.

OTHER SIGNS AND SYMPTOMS

A. Adults may develop varicella pneumonia. (The risk of pneumonia is higher in smokers than nonsmokers.)

B. Cough.

C. Headache.

D. Respiratory symptoms include cough and chest discomfort. Respiratory symptoms usually develop shortly after cutaneous eruption. Respiratory failure in pregnancy can be rapid.

E. Abdominal pain lasting 1 to 2 days.

SUBJECTIVE DATA

A. Review the onset, duration, and course of symptoms.

B. Elicit exposure information when noting characteristic rash, the time it started, spread of the rash or lesions, and characteristic changes.

C. Review any pulmonary or nervous system problems, such as seizures, that occur as complications.

D. Determine if the person has HIV, is immunocompromised, or is pregnant.

E. Determine the caregiver's immunity status to varicella.

F. Review the client's immunization history.

G. Review all medications, including over-the-counter (OTC) and herbal products.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.

B. Inspect:

1. Conduct a dermal exam, especially the hairline.

2. Inspect the buccal mucosa.

3. Conduct an ear, nose, and throat exam, and a detailed eye exam.

C. Auscultate:

1. Auscultate the heart and lungs.

D. Palpate:

1. Palpate the neck and lymph nodes. ▶

DIAGNOSTIC TESTS

A. Diagnosis is usually determined by clinical diagnosis with the appearance of skin eruptions, and laboratory tests are not necessary. Lab testing is recommended to confirm varicella as a cause, determine susceptibility, establish an individual's immunity, or establish varicella as cause of death.

B. Enzyme-linked immunosorbent assay (ELISA) is used to determine if a person has antibodies to VZV from past infection or vaccinations.

C. Varicella immunoglobulin G (IgG) and immunoglobulin M (IgM):

1. IgG antibodies help identify nonimmunity.

2. IgM antibodies suggest acute or recent infection.

3. + IgG with –IgM indicates immunity from either vaccination or past infection.

4. – IgG with –IgM indicates no immunity.

D. Pregnancy test if indicated.

E. Tzanck smear if diagnosis is questionable; it has about 60% sensitivity.

F. Culture lesion for herpes simplex.

G. Chest x-ray if experiencing pulmonary symptoms to rule out pneumonia.

DIFFERENTIAL DIAGNOSES

A. Varicella (chickenpox).

B. Herpes simplex.

C. Viral exanthems.

D. Scabies.

E. Impetigo.

F. Coxsackievirus.

G. Drug reaction.

H. Secondary syphilis.

I. Measles.

J. Rubella.

K. Rocky Mountain spotted fever (RMSF).

PLAN

A. General interventions:

1. Avoid contact with persons infected with chickenpox. Clients with varicella should avoid contact with others. Healthcare workers should be immune to varicella.

2. Strict isolation should be enforced. Varicella is contagious 1 week before outbreak and until the lesions crust over (about 1 week). Isolation is a precaution until the vesicles dry.

3. Order oatmeal baths (Aveeno) for comfort.

4. Supportive medication for comfort; see "Pharmaceutical Therapy."

B. Client teaching:

1. Lesions that can be covered pose little risk to a susceptible person because transmission usually occurs from direct contact with the fluid from the lesion. Clothing or a dressing should cover lesions until they have crusted.

2. Scarring can occur from secondary infection of lesions; encourage good handwashing and no scratching.

C. Pharmaceutical therapy:

1. The goal is to reduce morbidity and prevent complications. Treatment is based on age, immune state, duration, and presentation. Three medications can help reduce pain and symptoms, but caution should be taken with altered renal function.

a. Acyclovir (Zovirax). Dosage forms and strengths: intramuscular (IM) 50 mg/mL; suspension 200 mg/5 mL; tablet 400 mg and 800 mg:

i. Varicella zoster: adults greater than 40 kg: 800 mg PO Q6H for 5 days; 10 to 15 mg/kg intravenously (IV) Q8H for 7 to 10 days.

ii. Herpes zoster: adults 800 mg PO Q4H for 7 to 10 days.

b. Valacyclovir (Valtrex). Dosage forms and strengths: tablet 500 mg and 1 g; herpes zoster: 1 g PO Q8H for 7 days:

i. Varicella zoster: older than 2 years: 20 mg/kg PO Q8H for 5 days (not to exceed 1 g PO Q8H).

ii. Herpes zoster: 1 g PO Q8H for 7 to 10 days.

c. Famciclovir (Famvir). Dosage forms and strengths: tablet 125, 250, and 500 mg:

i. Herpes zoster: adults 500 mg PO Q8H for 7 days.

2. Antihistamines are helpful in the symptomatic treatment of pruritus.

a. Diphenhydramine HCl (Benadryl): adult dosage 25 to 50 mg PO every 4 to 6 hours orally or topical cream 1% to 2% three to four times daily to affected areas for itching.

3. Antipyretics are helpful for comfort/ pain:

a. Acetaminophen 500 to 1,000 mg every 4 to 6 hours as needed for pain.

4. The use of corticosteroids for clients with shingles to prevent PHN is controversial.

5. Treatment for PHN includes gabapentin, pregabalin, tricyclic antidepressants, phenytoin, carbamazepine, cimetidine, and topical capsaicin.

6. Immunizations:

a. Shingrix was approved by the Food and Drug Administration (FDA) in 2017 as the recommended shingles vaccine by the Advisory Committee on Immunization Practices in the United States. The CDC recommended healthy adults ≥50 years of age may receive two doses 2 to 6 months apart. Shingrix provides clients with protection against PHN and shingles. Protection remains above 85% the first 4 years postvaccination. Clients can receive influenza and pneumococcal vaccines at the same visit.

b. Studies revealed that Shingrix was 97% effective in shingles prevention for older adults 50 to 69 years and 91% prevention-effective among adults ≥70 years old. Zostavax is no longer available for vaccine use in the United States as of November 18, 2020.

FOLLOW-UP

A. No follow-up is necessary in uncomplicated cases.

B. Have the client return to the office for any secondary skin infections, conjunctival involvement, central nervous system (CNS) problems such as encephalitis and meningitis, or pneumonia.

CONSULTATION/REFERRAL

A. Refer the client to a physician if pregnant; varicella pneumonia in pregnancy is a medical emergency.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Varicella vaccine should not be administered to pregnant individuals:

a. Clients are advised not to get pregnant for at least 1 month following the varicella immunization.

b. A pregnant mother or household member is not a contraindication for immunization for a child in the household.

2. Reporting of inadvertent immunization when the varicella zoster-containing vaccine is given during pregnancy is encouraged: 1-800-986-8999 or via at www.merckpregnancyregistries.com/varivax.html.

3. Maternal complications of active varicella infection may include preterm labor, encephalitis, and varicella pneumonia. The mortality rate in gravid females is 10%. The profound maternal hypoxia that occurs in varicella pneumonia is associated with increased risk of spontaneous abortion and stillbirth.

4. Fetal complications of active varicella infection may include intrauterine growth restriction (IUGR), limb reduction defects, and eye defects.

5. Acyclovir is a category B drug based on FDA drug classification in pregnancy. IV acyclovir is recommended for pregnant clients with serious complications of varicella.

B. Adults:

1. Shingles (reactivated chickenpox) appears as grouped vesicular lesions distributed in one to three sensory dermatomes, sometimes accompanied by pain localized to the area. Systemic symptoms are few.

2. A recommendation for varicella vaccination includes persons at high risk for exposure, which include adolescents and adults who live in households with children.

3. Immunocompromised: IV antiviral therapy is recommended for immunocompromised clients, including clients being treated with chronic corticosteroids. Therapy initiated early in the course of illness, especially within 24 hours of rash onset, maximizes efficacy.

C. Geriatrics:

1. Herpes zoster incidence increases with age and clinicians must encourage vaccines to geriatric populations. Studies indicate that older populations experience herpes zoster incidences four times more than clients ≤29 years of age.

2. The thoracic and lumbosacral areas are more prominent locations for a shingles outbreak in geriatric females than geriatric males and the trigeminal area increased in both older adult sexes.

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WEST NILE VIRUS

DEFINITION

A. West Nile virus (WNV) is a viral illness caused by arthropod-borne viruses transmitted to humans from infected mosquitoes, particularly *Aedes, Culex*, or *Anopheles.* The mosquitoes become infected by feeding on infected animals, especially birds. WNV has been found in a variety of birds, but more commonly in crows, hawks, owls, and jays. These birds become infected by eating on dead infected birds and animals. Usually people who get infected with WNV have no or mild symptoms (fever, headache, body aches, rash, or swollen glands). If WNV enters the brain, it can be life-threatening. WNV is a nationally notifiable condition. All cases should be reported to local public health departments to help authorities recognize outbreaks and implement control measures. Once a client recovers from WNV disease, they are thought to have a lifelong immunity to the disease. There is no immunization for WNV. The two categories of WNV disease are nonneuroinvasive and neuroinvasive.

1. Nonneuroinvasive disease is less severe and often presents as a febrile illness.

2. Neuroinvasive disease leads to encephalitis, meningitis, and flaccid paralysis and requires much more intensive treatment.

INCIDENCE

A. WNV is a reportable disease that allows the Centers for Disease Control and Prevention (CDC) to maintain significant surveillance data and statistics. WNV was first reported in the United States in New York City 20 years ago. A total of 50 states and the District of Columbia and Puerto Rico reported WNV infections in people, birds, or mosquitoes in 2018. A total of 21,869 confirmed or probable cases of WNV disease in people have been reported to CDC, and of these 112,835 (59%) were reported as neuroinvasive disease (such as meningitis or encephalitis). Neuroinvasive disease increases hospitalization rates (>85%) and in those over 70 years of age 98% required hospitalization. Almost 90% of cases occur during July through September or summer months.

B. Approximately 1 in 150 clients develop severe neurologic disease. About 1 out of 10 people who develop severe illness affecting the central nervous system (CNS) will die.

C. WNV was first noted in North America in 1999. WNV is endemic in the Middle East, Africa, and Asia.

D. Human WNV infections usually begin in midsummer and decline in September, correlating with peak mosquito activity. Mosquito bites are most likely to occur during peak feeding times during dawn and dusk. Prolonged contact or multiple mosquito bites increase the risk of developing WNV.

PATHOGENESIS

A. Mosquitoes are infected with WNV when feeding on an infected animal host. Wild birds are the most common source of infection, although other animals such as horses and chickens can be animal hosts. The virus is delivered to the human via mosquito bite when the mosquito's saliva is deposited in the human skin cells. The virus replicates in the skin cells and migrates to the lymph nodes and various organs. Infected immune cells are able to transverse the blood–brain barrier and infect the brain parenchyma, leading to encephalitis and meningitis. The typical incubation period for WNV is 3 to 14 days.

▶

PREDISPOSING FACTORS

A. Time of year: late summer and early fall.
B. Region: living in or traveling to a region with a higher number of reported cases.
C. Work or recreational outdoor exposure.
D. Homelessness.
E. Age: most serious disease occurring in the elderly and infants.
F. Blood transfusion.
G. Organ transplant.

COMMON COMPLAINTS

A. Nonneuroinvasive WNV:
1. Up to 80% of persons are asymptomatic.
2. Fever (>100.4°F or 38°C).
3. Headache.
4. Myalgia.
5. Arthralgias.
6. Fatigue.
7. Pharyngitis.
8. Gastrointestinal (GI) symptoms: nausea, vomiting, and diarrhea.
9. Maculopapular rash: usually noted on chest, back, and arms; occurs in 20% to 50% of clients. The presence of the rash represents a decreased risk of neuroinvasive disease.

OTHER SIGNS AND SYMPTOMS

A. Abdominal pain.
B. Visual disturbances and eye pain: chorioretinal lesions, conjunctival injection, inflammatory vitritis, retinal hemorrhage.
C. Irregular heart rhythm.

POTENTIAL COMPLICATIONS

A. Neuroinvasive disease:
1. Meningitis: stiff neck, photophobia, focal neurologic deficits, and higher fever (to 104°F).
2. Encephalitis: mental status changes, stupor, confusion, coma, movement disorders, focal neurologic deficits, personality changes, higher fever (to 104°F), and seizures.
3. Flaccid paralysis: cranial nerve palsy, vertigo, dysarthria, dysphagia, and respiratory failure.
4. Other rare complications:
a. Cardiac dysrhythmia.
b. Myocarditis.
c. Rhabdomyolysis.
d. Optic neuritis.
e. Uveitis.
f. Chorioretinitis.
g. Orchitis.
h. Pancreatitis.
i. Hepatitis.

SUBJECTIVE DATA

A. Review the onset, course, and duration of symptoms, especially fever, rash, headache, and myalgia.
B. Ask the client to describe any neurologic symptoms, changes in mental status, stiff neck, photophobia, and seizures.
C. Review symptoms of other family members or coworkers who are also ill.
D. Ask the client to recall what activity brought about or preceded their symptoms.
E. Ask the client what steps they have taken to treat symptoms at home.
F. Review the client's medical history for any chronic illnesses.
G. Review all medications, including over-the-counter (OTC) and herbal products.
H. Review history of mosquito bites and any preventive steps taken, such as use of insect repellent.
1. Outdoor work activities.
2. Outdoor recreational activities.
3. Review recent travel.
I. Evaluate living conditions for exposure risks.

PHYSICAL EXAMINATION

A. Clients presenting with neurologic symptoms should be quickly assessed and referred to a neurologist for immediate evaluation of symptoms.
B. Check temperature, pulse, blood pressure, and respirations.
C. Inspect:
1. Observe general overall appearance, noting weakness, difficulty breathing, or changes in affect, speech, or level of consciousness (LOC).
2. Assess hydration status.
3. Ophthalmic examination: Assess the presence of papilledema.
4. Inspect face for muscle weakness or drooping.
5. Dermal inspection for presence of a maculopapular rash, especially on the abdomen, back, and arms.
D. Auscultate:
1. Auscultate heart for dysrhythmias.
2. Auscultate all lung fields for adventitious breath sounds.
E. Palpate:
1. Palpate skin for signs of dehydration: poor skin turgor.
2. Palpate the neck and lymph nodes: preauricular, posterior auricular, submental and sublingual, anterior cervical chain, and supraclavicular nodes.
3. Palpate abdomen for tenderness and/or organomegaly.
F. Mental status and neurologic examination:
1. Administer mental status exam to look for confusion, stupor, and changes in LOC.
2. Complete cranial nerve testing: Focus on visual fields, extraocular movement (EOM) of a transient downbeat nystagmus, facial muscle movement and strength, and gag reflex.
3. Muscle strength testing and testing for sensation in all extremities.
4. Test reflexes:
a. Deep tendon reflexes.
b. Babinski reflex is performed by running the reflex hammer up the midline of the sole of the foot from heel to the base of the toes (both feet are tested):
i. A normal reaction is for the toes to either remain still or else to curl downward.
ii. A positive Babinski is noted when the big toe points upward and the other toes fan out.
5. Assess for meningeal signs:
a. Signs of meningeal irritation include nuchal rigidity.
b. Assess for positive Brudzinski and Kernig signs (see Figures 19.1 and 19.2).
i. Brudzinski sign: Place the client supine and flex the head upward. Resulting flexion of both hips, knees, and ankles with neck flexion indicates meningeal irritation.

▶

ii. Kernig sign: Place the client supine, keeping one leg straight; flex the other hip and knee to a bent knee to form a 90° angle. Slowly extend the lower leg. This places a stretch on the meninges, resulting in pain and spasm of the hamstring muscle. Resistance to further extension can be felt.

DIAGNOSTIC TESTS

A. WNV immunoglobulin M (IgM) antibody capture ELISA (MAC-ELISA) is the gold standard diagnostic test.

1. The presence of WNV-specific IgM in blood or cerebrospinal fluid (CSF) provides good evidence of recent infection but may also result from cross-reactive antibodies after infection with other flaviviruses or from nonspecific reactivity. According to product inserts for commercially available WNV IgM assays, all positive results obtained with these assays should be confirmed by neutralizing antibody testing of acute- and convalescent-phase serum specimens at a state public health laboratory or CDC.

2. If serum is collected within 8 days of illness onset, the absence of detectable virus-specific IgM does not rule out the diagnosis of WNV infection and the test may need to be repeated on a later sample.

3. WNV immunoglobulin G (IgG) antibodies generally are detected shortly after IgM antibodies and persist for many years following a symptomatic or asymptomatic infection. Therefore, the presence of IgG antibodies alone is only evidence of previous infection, and clinically compatible cases with the presence of IgG, but not IgM, should be evaluated for other etiologic agents.

B. If there is concern that the illness could have been caused by another type of flavivirus, an additional test, the plaque reduction neutralization test (PRNT), is used to identify false-positive MAC-ELISA test results. PRNTs can also confirm acute infection by demonstrating a fourfold or greater change in WNV-specific neutralizing antibody titer between acute- and convalescent-phase serum samples collected 2 to 3 weeks apart.

C. Complete blood count (CBC) with differential.

D. For clients with neuroinvasive disease:

1. Lumbar puncture (LP), for viral culture and tests to detect viral RNA on CSF or tissue specimens. Positive tests for WNV will show pleocytosis, increased lymphocytes, increased protein, and normal glucose. The MAC-ELISA test should be performed on CSF when LP is completed.

2. Imaging: MRI of the brain and spinal cord is the preferred imaging test. MRI may show meningeal inflammation and bilateral lesions.

3. EEG: EEG may show generalized slowness.

DIFFERENTIAL DIAGNOSES

A. Non-neuroinvasive WNV:

1. WNV.

2. Other viral causes of febrile illness.

B. Neuroinvasive WNV:

1. Other viral causes of encephalitis: St. Louis equine encephalitis, California encephalitis (CE), Western and Eastern encephalitis.

2. Acute poliomyelitis.

3. Postpolio syndrome.

4. Guillain–Barré syndrome.

5. Multiple sclerosis.

6. Vertebrobasilar stroke.

PLAN

A. Nonneuroinvasive disease:

1. Client teaching:

a. Educate about expected signs and symptoms of WNV acute febrile illness.

b. Explain signs and symptoms that would require immediate medical evaluation, such as mental status changes, stiff neck, and neurologic symptoms.

c. Instruct the client to rest as needed. The client may expect fatigue to continue for up to 3 weeks.

d. Instruct that WNV disease will be reported to the health department for surveillance purposes. Special populations may be subject to follow-up.

e. Prevention strategies for WNV include ways to avoid mosquito bites.

i. Use mosquito repellent containing N,N-diethyl-meta-toluamide (DEET), picaridin, FR3535, or oil of lemon eucalyptus.

ii. Mosquitoes are most active from dusk to dawn; stay indoors during these peak times.

iii. Wear long sleeves and pants.

iv. Remove mosquito breeding sites; dump standing water in flower pots and buckets.

v. Change the water in bird baths weekly.

f. Finding dead birds may be a sign that WNV is circulated between birds and mosquitoes.

i. Do not handle a dead bird with bare hands.

ii. Report finding dead birds to the local health department for testing.

2. Dietary management:

a. There are no specific dietary recommendations for WNV.

b. Encourage the client to drink plenty of fluids to prevent dehydration.

c. If the client is experiencing nausea and vomiting, meals should be light and nonspicy and should be taken in smaller amounts more frequently.

3. Pharmaceutical therapy:

a. There are no antiviral treatments for WNV.

b. Utilize supportive therapy such as acetaminophen for fever, antiemetics for nausea and vomiting, and antidiarrheal medications for diarrhea.

4. There are no WNV vaccines licensed for use in humans.

B. Neuroinvasive disease:

1. Clients presenting with neurologic symptoms should be quickly assessed and referred to a neurologist for immediate evaluation and treatment of acute symptoms.

FOLLOW-UP

A. Follow-up would be determined by the client's needs, severity of acute symptoms, and risk of complications.

B. WNV disease is a reportable disease to the health department for surveillance purposes.

CONSULTATION/REFERRAL

A. Neuroinvasive disease: Refer client to a neurologist immediately:

1. For longer term needs from neuroinvasive disease, the client may benefit from a referral to a neuropsychologist, rehabilitation specialist, physical therapist, occupational therapist, and/or speech therapist.

B. Consider an infectious disease specialist if there is difficulty with identifying the infectious agent.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. There has been no causal relationship found between WNV during pregnancy and fetal abnormalities. A small number of infants, born to individuals who developed WNV within 3 weeks prior to delivery, were found to have symptomatic WNV disease shortly after birth.

2. If WNV is diagnosed during the last few weeks of pregnancy, a detailed examination of the newborn should be completed and steps taken to monitor for signs and symptoms of the disease in the days and weeks after the birth.

3. All cases of WNV in pregnant individuals should be reported to the health department so that the cases can be followed to determine the outcome of the pregnancies.

4. The virus has been found in breast milk, and in rare cases breastfeeding has been linked to development of WNV in infants. The benefits of breastfeeding outweigh the risks of WNV disease; therefore, mothers should be encouraged to breastfeed.

5. Pregnant individuals can use insect repellent products containing DEET without adverse effects.

B. Geriatrics:

1. Adults older than age 60 are more likely to suffer more serious illness and are more likely to develop neuroinvasive disease. Close attention should be given to the mental status exam and neurologic examination of the elderly.

2. WNV is particularly dangerous for the geriatric population because it can cross the blood–brain barrier and rapidly decline cognition function and mental behavior. Experimental research suggested that older populations (compared with younger) have a 20-fold higher WNV-viral level in brains and a less potent WNV-specific antibody response, which could worsen clinical outcomes and increase mortality from WNV infection.

3. Frank encephalitis occurs more in the elderly and manifests with fever, altered mental status, seizures, focal neurologic deficits, or movement disorders such as tremor parkinsonism.

4. Implement education to cognitively impaired geriatric clients and families/surrogates regarding permethrin-treated clothing that will continue to protect the client after multiple washings. However, do not use permethrin directly on thin, aging skin.

5. Assist physically challenged geriatrics with finding resources to install/repair all screened windows and doors. Guide them to use appropriate insect sprays (outdoors/indoors) to kill mosquitoes in humid areas: laundry rooms, underneath sinks, patio furniture, carports, porches, and inside garages.

6. Many retired older adults and geriatric populations enjoy bird-watching and outdoor activities. Assure this population that, although birds may carry WNV, there is no evidence that a person can become infected from handling a dead/alive infected bird. However, use sanitary precautions when handling any animal (dead/alive).

7. Prevention: Avoid mosquito-populated areas, outdoor activities at dusk and dawn times, and areas with standing water. Use mosquito repellent that contain picaridin or DEET recommended in risk areas. A WNV vaccine has been created for horses but not for humans to date.

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CLIENT TEACHING GUIDE

LYME DISEASE AND REMOVAL OF A TICK

PROBLEM Ticks are vectors for Lyme disease and Rocky Mountain spotted fever. You have been diagnosed with Lyme disease.

CAUSE Lyme disease is caused by a spirochete from ticks.

PREVENTION

A. Avoid wooded and brushy areas with high grass and leaf litter.

B. Wear light-colored clothes to make ticks easier to spot. Wear long sleeves and tuck pants into the socks to form a barrier. Tumble clothes in a dryer on high heat for 10 minutes to kill ticks on dry clothes after you come indoors.

C. Stick to hiking trails. Avoid contact with overgrown foliage. Ticks prefer dense woods with thick growth of shrubs and small trees, as well as along the edge of the woods.

D. Check for ticks after each outdoor activity, especially hairy regions of the body (underarms, in and around the ears, belly button, behind the knees, between the legs, waist and beltline, and hair and scalp), where ticks often attach. Check for ticks prior to bathing.

E. Bathe or shower as soon as possible after coming indoors to wash off and make it easier to find ticks that are crawling on you.

F. Remove ticks promptly (see instructions that follow).

G. Inspect pets daily and remove ticks when present.

H. Some manufacturers offer permethrin-treated clothing that is effective for up to 20 washings. This clothing is not recommended for children.

I. Use Environmental Protection Agency (EPA)-registered insect repellents containing DEET, picaridin, IR3535, oil of lemon eucalyptus (OLE), para-menthane-diol (PMD), or 2-undecanone. EPA's helpful search tool can help you find the product that best suits your needs. Always follow product instructions. The EPA website is available at www.epa.gov/insect-repellents.

1. Do not use insect repellent in babies younger than 2 months old.
2. Do not use products containing OLE or PMD in children under 3 years old.

PRECAUTIONS

A. Do not hold a lighted match or cigarette to the tick. Do not apply gasoline, kerosene, or oil to the tick's body.

B. Old treatments, including using nail polish and petroleum jelly, should not be used to remove ticks.

TICK REMOVAL

A. Use fine-tip tweezers to grasp the tick as close to the skin's surface as possible.

B. Wash your hands and the tweezers with soap and water prior to beginning.

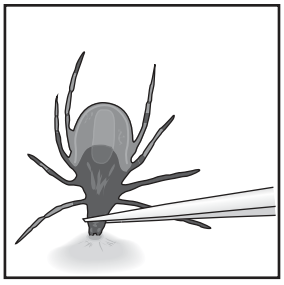


FIGURE 1 Grasp the tick's body close to the skin using a fine-tip tweezer.

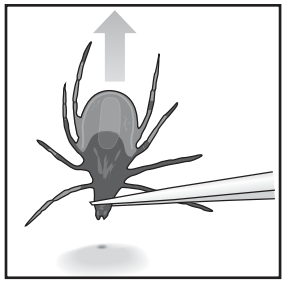


FIGURE 2 Avoid squeezing the body. Remove by pulling the tick straight upward without using twisting motions.

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CLIENT TEACHING GUIDE

LYME DISEASE AND REMOVAL OF A TICK (*CONTINUED*)

C. Grasp the tick with a fine-tip tweezer close to the skin. Remove by gently pulling upward with steady, even pressure without using any twisting motions (see illustrations that follow). If the mouth parts break off and remain in the skin, remove them with the tweezers.
D. If unable to remove the mouth parts easily with clean tweezers, leave it alone and let the skin heal.
E. Clean the bite area and your hands with rubbing alcohol or soap and water.
F. Do not crush or squeeze the tick during removal or with your fingers.
G. Dispose of a live tick by putting it in alcohol, placing it in a sealed bag/container, wrapping it tightly in tape, or flushing it down the toilet.

TREATMENT PLAN

Activity: As tolerated.
A. After removal, monitor for signs and symptoms of tickborne disease up to 30 days.
B. If you are prescribed doxycycline, avoid exposure to the sun because a rash may develop.

Diet: Eat a regular diet.

Medications:
A. Acetaminophen or Ibuprofen may be taken for body aches and any fever.
B. Antibiotics are effective against Lyme disease. Most common are doxycycline, amoxicillin, or cefuroxime.

EMERGENT ISSUES/INSTRUCTIONS

A. Go to the hospital for any alteration in mental status, change in behavior, stiff neck, severe headaches, seizure, nausea and vomiting, severe weakness, dizziness, or high fever.

You Have Been Prescribed: _____

You Need to Take: _____

Take all of your antibiotics even if you feel better.

You Need to Notify the Office If You Have:
A. No signs of improvement with antibiotic therapy.
B. Red rash that develops, especially if it looks like a target's bull's eye (See Figure 3.)
C. Development of a high fever.
D. Other: _____

Phone: _____



FIGURE 3 Bull's eye target rash.

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CLIENT TEACHING GUIDE

MONONUCLEOSIS

PROBLEM Mononucleosis (mono) is an acute, infectious, viral disease. Mono causes fever, sore throat, fatigue, and swollen lymph glands.

CAUSE Epstein–Barr virus causes mono and is spread to other persons through body fluids, primarily saliva, for example, kissing, sharing food, and coughing without covering your mouth.

PREVENTION

A. Avoid contact with persons diagnosed with mono.
B. Cover your mouth and nose when you cough or sneeze to prevent the spread of infection.
C. Use tissues to blow your nose and throw them away.
D. If you do not have a tissue, use your upper sleeve or the "elbow sneeze" using the bend of your arm instead of using your hand.
E. Wash your hands or use hand sanitizer.

TREATMENT PLAN There is no specific cure. For sore throat, gargle with warm saltwater.

Activity:

A. Mono makes you very tired; rest in bed, then gradually return to normal activity.
B. You should not do any physical activity that puts you at risk for blunt trauma, especially contact sports (football, soccer, basketball, etc.), unless you have been cleared by your healthcare provider.

Diet: Eat a high-calorie diet. Drink plenty of liquids (at least eight glasses a day).

Medications:

A. Analgesics, such as acetaminophen or ibuprofen, may be taken for fever, body aches, and malaise.
B. Antibiotic therapy is reserved for concurrent infections such as streptococcal pharyngitis.
C. Corticosteroids may be considered in the presence of significant inflammation and swelling of the throat.
D. You may be given other medications by your provider.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Severe pain in the upper left abdomen; rupture of the spleen is a medical emergency, so go immediately to the hospital.
B. Fever more than 102°F over 24 to 48 hours.
C. Swallowing or breathing difficulty from a severe throat inflammation.
D. Rash: A rash may follow if antibiotics are given.
E. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

ROCKY MOUNTAIN SPOTTED FEVER AND REMOVAL OF A TICK

PROBLEM Ticks are vectors for Lyme disease and Rocky Mountain spotted fever.

CAUSE Rocky Mountain spotted fever is caused by a bacterium, *Rickettsia rickettsii*, carried by ticks, most commonly the "dog tick."

PREVENTION

A. Avoid wooded and brushy areas with high grass and leaf litter.
B. Wear light-colored clothes to make ticks easier to spot. Wear long sleeves and tuck pants into the socks to form a barrier. Tumble clothes in a dryer on high heat for 10 minutes to kill ticks on dry clothes after you come indoors.
C. Stick to hiking trails. Avoid contact with overgrown foliage. Ticks prefer dense woods with thick growth of shrubs and small trees, as well as along the edge of the woods.
D. Check for ticks after each outdoor activity, especially hairy regions of the body (underarms, in and around ears, belly button, behind the knees, between the legs, waist and beltline, and hair and scalp), where ticks often attach. Check for ticks prior to bathing.
E. Bathe or shower as soon as possible after coming indoors to wash off; this makes it easier to find ticks that are crawling on you.
F. Remove ticks promptly (see the following instructions).
G. Inspect pets daily and remove ticks when present.
H. Some manufacturers currently offer permethrin-treated clothing that is effective for up to 20 washings. This clothing is not recommended for children.
I. Antibiotic therapy to prevent Rocky Mountain spotted fever is not recommended for tick exposure. Instead, tell your healthcare provider if any symptom, especially fever and headache, occurs in the following 14 days.
J. Use Environmental Protection Agency (EPA)-registered insect repellents containing DEET, picaridin, IR3535, oil of lemon eucalyptus (OLE), para-menthane-diol (PMD), or 2-undecanone. EPA's helpful search tool can help you find the product that best suits your needs. Always follow product instructions. The EPA website is available at www.epa.gov/insect-repellents.
    1. Do not use insect repellent in babies younger than 2 months old.
    2. Do not use products containing OLE or PMD in children under 3 years old.

PRECAUTIONS

A. Do not hold a lighted match or cigarette to the tick. Do not apply gasoline, kerosene, or oil to the tick's body.
B. Old treatments, including using nail polish or petroleum jelly, should not be used to remove ticks.

TICK REMOVAL

A. Use fine-tip tweezers to grasp the tick as close to the skin's surface as possible.
B. Wash your hands and the tweezers with soap and water prior to beginning.
C. Grasp the tick with a fine-tip tweezer close to the skin. Remove by gently pulling upward with steady, even pressure without using any twisting motions (see illustrations that follow).
D. If the mouth parts break off and remain in the skin, remove the mouth parts with the tweezers.
E. If unable to remove the mouth easily with clean tweezers, leave it alone and let the skin heal.
F. Clean the bite area and your hands with rubbing alcohol or soap and water.
G. Do not crush or squeeze the tick during removal or with your fingers.

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CLIENT TEACHING GUIDE

ROCKY MOUNTAIN SPOTTED FEVER AND REMOVAL OF A TICK (*CONTINUED*)

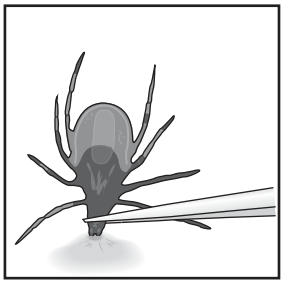


FIGURE 1 Grasp the tick's body close to the skin using a fine-tip tweezer. Avoid squeezing the body.

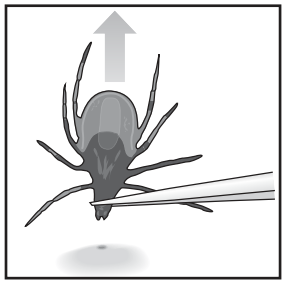


FIGURE 2 Remove by pulling the tick straight upward without using twisting motions.

H. Dispose of a live tick by putting it in alcohol, placing it in a sealed bag/container, wrapping it tightly in tape, or flushing it down the toilet.

TREATMENT PLAN

Activity: If the tick is removed, monitor for signs and symptoms of illness for up to 30 days.

Diet: Eat regular diet. Drink 8 to 10 glasses of water daily.

Medications:

A. Acetaminophen (Tylenol) or ibuprofen (Motrin, Advil, etc.) may be taken for body aches and fever.
B. You may be given an antibiotic for secondary infection, if needed. Finish all antibiotics, even if you are feeling better.

You Have Been Prescribed: _____

You Need to Take: _____

Take all of your antibiotics even if you feel better.

EMERGENT ISSUES/INSTRUCTIONS

A. Go to the hospital for any alteration in mental status, change in behavior, stiff neck, severe headaches, seizure, nausea and vomiting, severe weakness, dizziness, or high fever.
B. Go to the hospital if you have chest pain, shortness of breath, swelling, hearing loss, or skin changes.

You Need to Notify the Office If You Have:

A. No improvement while on antibiotic therapy.
B. Development of a high fever.
C. Rash that increases instead of improving.
D. Decreased urinary output.
E. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

TOXOPLASMOSIS

PROBLEM Toxoplasmosis is an infection acquired through contact with infected cat feces or from eating raw or undercooked meat.

CAUSE *Toxoplasmosis* is caused by a parasite and acquired through contact with infected cat feces (cleaning a cat's litter box), by eating raw or undercooked meat (usually pork, lamb, goat, or wild game meat), gardening, or by eating soil-contaminated fruit or vegetables.

PREVENTION

A. Proper and thorough handwashing, especially after gardening or handling raw meats.
B. Wash fruits and vegetables before eating.
C. Wash all kitchen surfaces that come into contact with uncooked meats.
D. Avoid uncooked eggs and unpasteurized milk.
E. Avoid drinking unfiltered water in any setting.
F. Meat should be thoroughly cooked at 152°F or higher, or frozen for 24 hours in a household freezer before eating (smoked meats and meats cured in brine are considered safe). Avoid tasting meat while cooking.
G. Wear gloves for gardening and landscaping, especially where cats have access.
H. Wear gloves for handling kitty litter, and wash hands after contact with cats. Change kitty litter daily.
I. Keep outdoor sandboxes covered.
J. Domestic cats can be protected from infection by feeding them commercially prepared cat food and preventing them from eating undercooked kitchen scraps and hunting wild rodents.
K. If you are not pregnant and have toxoplasmosis, you should not get pregnant for at least 6 months.
L. Have cats regularly examined by a veterinarian routinely and if infection occurs.

TREATMENT PLAN

A. If you are pregnant, you may be referred to a specialist.
B. If you have AIDS, you will be referred to a specialist.

Activity: There is no activity restriction for toxoplasmosis.

Diet: There is no special diet for toxoplasmosis. Avoid uncooked eggs and unpasteurized milk. Meat should be thoroughly cooked.

Medications: Depending on your risk, you may be prescribed one or two medications. You may be required to take the medication for several weeks.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Headache, dull and constant, with no relief from acetaminophen (Tylenol).
B. Abnormal speech.
C. Seizures.
D. Loss of visual acuity.
E. Poor concentration, forgetfulness.
F. Personality changes.
G. Other: _____

Phone: _____

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CHAPTER 20

SYSTEMIC DISORDERS GUIDELINES

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CANCER MANAGEMENT

DEFINITION

A. Cancer is a disease in which cells in the body divide in an abnormal fashion, invading normal cells and tissues. These abnormal cells proliferate and spread to other parts of the body by means of the blood and of the lymphatic systems. Cancer cells generally begin in one area of the body, causing "cancer" in that particular organ of the body. However, with invasion of other tissues/organs, the cancer cells can spread to other organs, causing "cancer" in other parts of the body.

INCIDENCE

A. There are over 100 different types of cancer. The different cancers are broken down into five broad categories.

1. Carcinoma: cancer of the skin and/or tissue covering major internal organs.
2. Sarcoma: cancer of the bone, cartilage, muscle, fat, connective tissue, and blood vessels.
3. Leukemia: cancer of the bone marrow and blood forming tissues.
4. Lymphoma and myeloma: cancer of the immune system.
5. Central nervous system: cancer of the brain and spinal cord system.

B. It has been estimated that in the year 2020 there were approximately 1.8 million new cases of cancer reported and approximately 606,520 cancer-related deaths expected in the United States. The cancers that are expected to be the most frequent include the following:

1. Bladder cancer.
2. Breast cancer.
3. Lung cancer.
4. Melanoma.
5. Colon/rectal cancer.
6. Non-Hodgkin lymphoma.
7. Endometrial cancer.
8. Pancreatic cancer.
9. Kidney (renal cell) cancer.
10. Prostate cancer.
11. Leukemia.
12. Thyroid cancer.

C. The most common cancers in females are breast, lung, and colorectal can cers. Th e most comm on cancers in males are prostate, lung, and colorectal cancers.

PATHOGENESIS

A. Cell division occurs by the body producing new cell formations in a controlled order, producing the appropriate number of cells to keep the body healthy. Once the cell grows and divides properly, the cell will die and new cells will replace the old cells. However, when cancer occurs, cell division does not occur properly, and mutations occur that affect normal cell growth and division. The existing cells do not die while new cells are being produced, and the overproduction/accumulation of cells forms a mass, which is known as a tumor. Tumors may be benign or malignant. Depending on anatomic location, benign tumors are not hazardous to the body. Malignant tumors are cancerous and may spread to other parts of the body, known as metastasis.

PREDISPOSING FACTORS

A. Predisposing risk factors for each type of cancer depend on the type of cancer present.

COMMON COMPLAINTS

A. Many complaints and side effects may occur prior to the diagnosis of cancer as well as during cancer treatment. Warning signs of cancer are the following:

1. Change in bowel or bladder habit.
2. A sore that does not heal.
3. Unusual bleeding or discharge.
4. Thickening or lump in breast, testicles, or elsewhere.
5. Indigestion or difficulty swallowing.
6. Obvious change in the size, color, shape, or thickness of a wart, mole, or mouth sore.
7. Nagging cough or hoarseness.
8. Coughing up blood.
9. Persistent headaches.
10. Unexplained loss of weight or appetite.
11. Chronic pain in bones.
12. Persistent fatigue, nausea, or vomiting.
13. Persistent low-grade fever, either constant or intermittent.
14. Repeated instances of infection.
15. Unexplained bruising.

B. Common complaints associated with cancer and treatment side effects:

1. Nausea and vomiting.
2. Constipation.
3. Diarrhea.
4. Fever and neutropenia.

The contributions of Julie Adkins, Beverly R. Byram, Cheryl A. Glass, Robertson Nash, and Pat Obulaney to this chapter in prior editions are acknowledged here.

SUBJECTIVE DATA

A. Inquire about onset, duration, and course of presenting symptoms.
B. What measures has the client used to relieve the symptoms?
C. What makes the symptoms worse or better?
D. What medications, over-the-counter (OTC) or prescription, have been used to treat the symptoms? What were the results?
E. Over the past week, how have symptoms changed?
F. Is client sleeping well? If not, what have they tried to do to assist sleeping?
G. Has client lost or gained weight over the past few weeks?
H. Is the client experiencing fever, cough, dyspnea, tightness in the chest, dysuria, or diarrhea?

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure, and weight.
B. Inspect:
  1. Observe overall appearance.
  2. Inspect skin for color and hydration.
C. Palpate:
  1. If abdominal symptoms exist, palpate abdomen for tenderness and masses.
  2. Evaluate for tissue swelling.
D. Percuss:
  1. Percuss abdomen.
E. Auscultate:
  1. Auscultate heart and lung sounds.
  2. Auscultate abdomen for bowel sounds.
F. Neurologic exam:
  1. Assess deep tendon reflexes (DTRs) and mental status.

DIAGNOSTIC TESTS

A. Screening tests for specific cancers:
  1. Mammogram starting at 40 years of age.
  2. Colonoscopy every 10 years or flexible sigmoidoscopy or double contrast barium enema every 5 years.
  3. Pap smear starting at 21 years of age.
B. Complete blood count (CBC) with differential.
C. Comprehensive metabolic profile (CMP).
D. Bone mineral density (BMD): Consider assessing for osteoporosis in clients who are at risk for fracture. Long-term use of steroids, certain chemotherapy treatment, androgen deprivation therapy, and advanced age contribute to an increase risk of bone loss leading to osteoporosis.

DIFFERENTIAL DIAGNOSES

A. Cancer being the primary diagnosis, the secondary diagnosis is made according to the presenting symptoms, which may include the following:
  1. Dehydration.
  2. Fatigue.
  3. Nausea and vomiting.
  4. Constipation.
  5. Diarrhea.
  6. Osteoporosis.
  7. Anemia.
  8. Neutropenic fever.

PLAN

A. General interventions for the common side effects of cancer treatment may include the following:
  1. Fatigue (National Comprehensive Cancer Network [NCCN] website: www.nccn.org/professionals/physician_gls/f_guidelines.asp):
    a. Limit naps to not interfere with nighttime sleep quality. Balance activity and rest.
    b. Encourage daily exercise or activities as tolerated. Short periods of exercise and activity help to increase energy levels and also help with anxiety/depression.
    c. Encourage the client to ask family and friends for help when needed. During this time, the client should not feel guilty for needing help with certain activities.
    d. Encourage the client to keep a schedule and block off times when they will need to perform certain activities. Saving energy for these tasks/time blocks will help the client complete the task and feel better when performing the task.
    e. Methylphenidate is helpful in the management of cancer-related fatigue.
  2. Nausea and vomiting (NCC website: www.nccn.org/professionals/physician_gls/f_guidelines.asp):
    a. Several medications are used to prevent and treat nausea and vomiting. Some medications may be used prior to the start of chemotherapy to prevent nausea and vomiting. If the client is prescribed around-the-clock dosing of their antiemetic drug, encourage them to take even if not experiencing nausea. The medication used may depend on the type of chemotherapy being prescribed. Stress the importance of the client notifying the oncologist about the severity of nausea/vomiting and treatment success with medications being prescribed.
    b. Other suggestions to improve nausea and vomiting include the following:
      i. Eat small, frequent meals during the day. Eating meals at a slower pace can also improve symptoms.
      ii. Bland foods are best tolerated. Avoid spicy foods, alcohol, and caffeine.
      iii. Wear comfortable clothing that does not restrict breathing or interfere with comfort around the abdomen.
      iv. Encourage frequent oral hygiene to eliminate bad tastes in the mouth.
      v. Eat foods that have ginger in them, such as ginger tea. Ginger has been shown to reduce nausea.
      vi. Hydration must be maintained. Encourage the client to drink plenty of liquids in whatever form they are able to tolerate. Water, Gatorade, Pedialyte, and other bland liquids are suggested. Some clients may also tolerate liquids that are cold; however, drinking liquids that are close to room temperature may be easier for some clients to keep down.
  3. Constipation/diarrhea:
    a. Constipation can be experienced as a side effect of some chemotherapy medications and opioids.
    b. Encourage the client to notify their oncologist if severe constipation is experienced.
    c. Encourage good dietary habits, such as increasing liquids and eating foods high in fiber for prevention/treatment of constipation.
    d. Healthy dietary choices include foods high in protein (eggs, meat, fish, poultry, peanut butter, beans), an assortment of fruits and cooked vegetables, and foods low in fat and lactose. Avoid fatty/fried foods, raw vegetables, and dried fruits.
    e. Daily exercise is recommended to improve constipation.
    f. All clients receiving opioids need to be placed on a stool softener and laxative. ▶

g. Diarrhea may occur as a side effect and can cause dehydration and loss of electrolytes from the body. Hydration should be maintained by drinking plenty of water, Gatorade, or Pedialyte. OTC medications may be used, such as loperamide (Imodium). If medications are needed to control the diarrhea, the primary provider or oncologist should be notified. If diarrhea is not controlled by diet and OTC medications, prescription medications may be given. For severe diarrhea not controlled by dietary changes and oral medications, intravenous (IV) hydration and medications should be prescribed.

4. Depending on anatomic location, bone loss can be a side effect in clients treated with radiation. Radiation breaks down the bone-building cells and increases the risk of fracture. Steroids are given to some clients with cancer. Steroids are known to increase the production of osteoclasts, which are cells that break down bone tissue. Androgen deprivation therapy, used to manage certain breast and prostate cancer, can lead to osteoporosis. Some chemotherapy combinations can result in early menopause. Therefore, all at-risk clients should have a baseline bone density screening test (dual-energy x-ray absorptiometry [DEXA] scan) and screen fracture risk using the Fracture Risk Assessment Tool (FRAX®) score. Vitamin D and calcium should also be taken daily. Counsel the client regarding daily weight-bearing activities to increase bone strength. See Chapter 22, "Rheumatologic Guidelines," for evaluation and treatment of osteoporosis.

5. Anemia should also be evaluated in clients receiving medications for cancer. Anemia can be caused by the cancer, radiation if bone marrow in the field, chemotherapy, iron deficiency, or a combination of these. Symptoms of anemia include fatigue, pale skin, shortness of breath, tachycardia, dizziness, and so forth. Majority of anemia in cancer clients is anemia of chronic disease. See the "Anemia" section of this chapter.

6. Neutropenia occurs when chemotherapeutic agents destroy maturing neutrophils. The client will be at greatest risk for neutropenia 7 to 14 days after last treatment and the neutrophils usually begin to recover within 21 to 28 days. Preventive measures to reduce the risk of infection include the use of granulocyte colony stimulating factors (G-CSF; e.g., filgrastim), good handwashing, avoiding crowds, and staying away from people who recently received attenuated vaccinations. Immunization recommendations are inactivated influenza vaccine annually. Newly diagnosed adult cancer clients are immunized against pneumococcal infection if never received. Client receives pneumococcal conjugate vaccine (PCV13), followed by pneumococcal polysaccharide vaccine (PPSV23) 8 weeks later (www.cdc.gov/vaccines/vpd/pneumo/hcp/who-when-to-vaccinate.html).

a. Neutropenic fever is defined as a single oral temperature of 38.3°C or a temperature o f greater tha n 38°C sustained for more than 1 hour in the neutropenic client. Fever requires prompt assessment and intervention/referral. A CBC with differential should be drawn. A chest x-ray and urinalysis are warranted based on clinical presentation (NCCN website: www.nccn.org/professionals/physician_gls/pdf/infections.pdf).

B. Caregiver support, advance directives, and palliative care should also be addressed. See Chapter 5, "Geriatric Syndromes," for information on this content.

C. Reference websites for clinicians and clients:

1. The NCCN comprises 25 designated cancer centers that have world-class, state-of-the-art research programs. The National Cancer Institute (NCI) is a U.S. federal agency for cancer research and clinician and family information. NCCN mobile apps are available for free for smartphones and tablets from the App Store for iPad or iPhone (search NCCN) or from the Google Play Store for the Android tablet or smartphone (search NCCN):

a. Clinician resources: NCCN guidelines: www.nccn.org/professionals/physician_gls/f_guidelines.asp.

2. NCCN client and caregiver resources:

a. NCCN guides for clients: www.nccn.org/patients/guidelines/cancers.aspx. Guidelines by cancer type, including breast cancer, caring for adolescents and young adults, colon cancer, melanomas, and others. The site also covers lung cancer screening.

b. Dictionary of terms: www.cancer.gov/publications/dictionaries/cancer-terms/.

c. Cancer diagnosis and staging guide: www.cancer.gov/about-cancer/diagnosis-staging.

3. NCI:

a. Prostate cancer treatment: www.cancer.gov/types/prostate/hp/prostate-treatment-pdq.

4. American Cancer Society:

a. Colon/rectum cancer: www.cancer.gov/types/colorectal/hp/colon-treatment-pdq.

b. Breast cancer: www.cancer.gov/types/breast/hp.

c. Ovarian cancer: www.cancer.gov/types/ovarian.

d. Cervical cancer: www.cancer.gov/types/cervical/hp.

5. National Breast Cancer Foundation, Inc:

a. Breast cancer: www.nationalbreastcancer.org/about-breast-cancer.

FOLLOW-UP

A. Follow-up will be based on the presenting symptoms and testing results. Refer to the NCCN guidelines for management of cancer survivors (NCCN website: www.nccn.org/professionals/physician_gls/pdf/survivorship.pdf).

CONSULTATION/REFERRAL

A. Refer the client to an oncologist or physician if side effects are not controlled with treatments offered.

B. For extreme cases of dehydration/illness or neutropenic fever, hospitalization may be required.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Geriatric clients who are immunocompromised deteriorate more quickly. The threshold for hospitalization and/or referral to the specialist should be lower for these clients along with more aggressive treatment options.

2. Oncology clinical guidelines recommend the Gupta & Zauderer Geriatric Assessment tool in evaluating the health status of geriatrics for those who are at increased risk for chemotherapy toxicity. The following is a summary:

a. Physical function: instrumental activities of daily living (ADLs), Karnofsky functioning status, number of falls in the past 6 months.

b. Comorbidities: subscale of Older Americans Resources and Services (OARS), multidimensional assessment of social resources, economic resources, mental and physical health, and ADLs.

c. Nutrition: body mass index (BMI), percentage of unintentional weight loss in the past 6 months.

d. Cognitive: Blessed Orientation Memory Concentration (BOMC) test.
e. Psychosocial: social activity limitations, social support systems.
f. Polypharmacy: total number of medications, high-risk medications.

3. Oncology research demonstrated using the Geriatrics Assessment Tool in the care of elderly clients suffering with cancer significantly improved provider–client communication regarding age-related concerns and increased client's satisfaction with cancer management.

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FEVERS OF UNKNOWN ORIGIN

DEFINITION

A. Fever of unknown origin (FUO) is classified into four distinct categories:

1. Classic FUO is a fever of 101.0°F (38.3°C) or greater, lasting for greater than 3 weeks with no identified cause after 3 days of hospital evaluation or three outpatient visits.

2. Healthcare-associated FUO is a fever in a hospitalized client receiving acute care and with no infection present or incubating at admission if the diagnosis remains uncertain after 3 days of appropriate evaluation.

3. Immune-deficient FUO is a fever in clients with neutropenia and other immunodeficiency if the diagnosis remains uncertain after 3 days of appropriate evaluation, including negative cultures after 48 hours.

4. HIV-related FUO is a fever for >3 weeks in outpatients with confirmed HIV infection or >3 days with confirmed HIV infection if the diagnosis remains uncertain after appropriate evaluation.

INCIDENCE

A. In adults, infections account for 17% to 35% of the cases, whereas cancer accounts for 10% to 20% of FUO cases. Autoimmune disorders occur with equal frequency in adults and children. Infection, cancer, and autoimmune disorders combined account for 20% to 25% of FUO in individuals who have been febrile for 6 months or longer. Various miscellaneous diseases account for another 25%. Approximately 16% to 39% of FUO remain undiagnosed but have a benign course, with symptoms eventually resolving.

B. In developing countries, an infectious etiology is most prevalent, whereas in developed countries FUO is more likely related to noninfectious inflammatory disease.

PATHOGENESIS

A. There are five categories of FUO causes:

1. Infection:

a. Localized: includes abscesses, endocarditis, osteomyelitis, urinary tract infection (UTIs), sinusitis, *Clostridium difficile* colitis.
b. Generalized: includes bacterial (bartonellosis, Q fever), viral (HIV, cytomegalovirus [CMV], Epstein–Barr virus [EBV]), fungal (histoplasmosis, coccidioidomycosis), mycobacterial (tuberculosis [TB]), and parasitic (malaria, toxoplasmosis).

2. Neoplasms: Most common are myelodysplastic syndrome (MDS), lymphoma, and leukemia.

3. Autoimmune disorders: Most common are adult Still disease, temporal arteritis, polymyalgia rheumatic, systemic lupus erythematosus (SLE), and polyarteritis nodosa.

4. Miscellaneous causes include drug fever, hyperthyroidism, thyroiditis, inflammatory bowel disease, sarcoidosis, Whipple disease, familial Mediterranean fever, recurrent pulmonary emboli, alcoholic hepatitis, factitious fever, and others.

5. Undiagnosed FUO.

PREDISPOSING FACTORS

A. Upper respiratory infection.
B. UTI.
C. Viral illnesses.
D. Drug allergy, especially to antibiotics.
E. Connective tissue disease.

COMMON COMPLAINTS

A. The client feels "sick all over," with malaise and fatigue.
B. The client has chills all over the body with high fever.

OTHER SIGNS AND SYMPTOMS

A. Tachycardia.
B. Sensation of warmth or flushing; night sweats.
C. Piloerection.
D. Myalgia.
E. Mild inability to concentrate, confusion, delirium, or even stupor.
F. Labial herpes simplex outbreak, or fever blisters.
G. Fever, drenching night sweats, and weight loss of 10% or more in the past 6 months are symptoms of concern for neoplasm.

SUBJECTIVE DATA

A. Review the onset, course, and duration of symptoms.
B. Review family, occupational, and social history.
C. Review sexual practices, including monogamy and oral, rectal, and vaginal sexual habits, recreational habits, and any new changes.
D. Elicit information regarding use of intravenous (IV) drugs.
E. Review if the client has had this illness before. How was it treated?
F. Review travel during the past month.
G. Review any new hobbies, changes at home or work, or other new events.
H. Review the client's history for contact with any friends or family members who have been sick and do not seem to be getting any better, including exposure to TB.
I. Review the client's history for eating any uncooked meat and dietary changes during the past month.
J. Review for risk factors for thrombophlebitis (see Chapter 13, "Cardiovascular Guidelines").
K. Review fever pattern, duration, and magnitude. Note response to antipyretics. Acute fevers are less than 7 days in duration. Chronic or persistent fevers are greater than 2 weeks in duration.
L. Review immunization status.
M. Review medications both current and recent, including herbals and over-the-counter (OTC) drugs. Chemotherapy or immunosuppressive medications place clients at higher risk for infection. Any history of antimicrobial use for current fever episode? ▶

N. Any prosthetic devices.
O. Recent invasive surgeries or procedures including dental work.
P. History of blood or blood product transfusions.
Q. Zoonotic exposure or insect bites.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure, weight, and height. Is the client tachycardic or heart rate within normal limits?
B. Inspect:
  1. Conduct funduscopic exam to rule out retinopathy, Roth spots, and choroidal tubercles.
  2. Examine the ears, nose, and throat. Inspect the mouth.
  3. Observe the skin and mucous membranes.
  4. Assess nails for clubbing and splinter hemorrhages.
C. Palpate:
  1. Palpate the neck, axilla, and groin for lymphadenopathy.
  2. Elderly: Palpate the scalp for tender arteries of temporal arteritis.
  3. Palpate the thyroid gland.
  4. Palpate the abdomen for organomegaly, masses, tenderness, guarding, rebound, suprapubic tenderness, and costovertebral angle (CVA) tenderness.
  5. Palpate the musculoskeletal system for bone or joint swelling, tenderness, and increased warmth; check lower extremities for evidence of phlebitis, asymmetrical swelling, calf tenderness, and palpable cord.
D. Percuss:
  1. Percuss the sinuses for tenderness, and transilluminate for evidence of sinusitis.
  2. Percuss the chest for consolidation.
  3. Percuss the abdomen.
E. Auscultate:
  1. Auscultate the heart for murmurs and rubs.
  2. Auscultate the lungs for rales, consolidation, and effusion.
F. Neurologic exam:
  1. Assess for signs of meningeal irritation (Brudzinski and Kernig signs), photosensitivity, and presence of focal deficits.
  2. Conduct mental status exam.
G. Genitorectal exam, if applicable:
  1. Females: Conduct pelvic exam for cervical discharge, adnexal masses, lesions, and pelvic inflammatory disease (PID) symptoms.
  2. Males: Conduct prostate and testicular exam for tenderness and masses; check penis for discharge, rash, and lesions.
  3. Both: Conduct rectal exam for discharge, tenderness, and masses; check stool for occult blood specimen.
  4. Check the skin for rashes or wounds.

DIAGNOSTIC TESTS

A. Complete blood count (CBC) with differential.
B. Erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and ferritin.
C. Urinalysis and urine culture.
D. Complete metabolic panel.
E. Liver function test.
F. Blood cultures. Three sets of blood cultures from different sites and at different times; incubation 5 days.
G. Consider serology for suspected infections or pathology (refer to Chapter 19, "Infectious Disease Guidelines"). Suspected infections include EBV, Q fever, Lyme disease or other tickborne diseases, hepatitis, syphilis, HIV, and CMV. Based on geographic location, consider testing for endemic fungal infections.
H. Suspected collagen disease: antinuclear antibodies (ANA) and rheumatoid factor.
I. Suspected TB: tuberculin skin test or interferon-gamma release assay (IGRA), sputum, and urine cultures.
J. Immunologic studies: enzyme-linked immunosorbent assay (ELISA), western blot test, and antistreptolysin O (ASLO) titer.
K. Suspected mononucleosis: heterophile antibody test.
L. Suspected *Salmonella*: Widal test.
M. Suspected thyroiditis: thyroid profile.
N. Suspected malaria or relapsing fever: direct examination of blood smears.
O. Q fever serology if risk factors include exposure to farm animals.
P. Imaging: depends on suspected infection:
  1. Chest, sinus radiographic films.
  2. Gastrointestinal (GI) studies: proctosigmoidoscopy, evaluation of gallbladder function.
  3. CT scan of abdomen and pelvis, abdominal ultrasonography.
  4. MRI is better than CT scan for detecting lesions of the nervous system.
Q. Suspected embolism: ventilation–perfusion (V/Q) scan.
R. Suspected endocarditis or atrial myxoma: echocardiography.
S. Radionuclide studies: gallium scan and radium-labeled immunoglobulin useful in detecting infection and neoplasm.
T. Laparotomy in the deteriorating client if the diagnosis is elusive despite extensive evaluation. Any abnormal finding should be aggressively evaluated: Headache necessitates a lumbar puncture to rule out meningitis; biopsy any skin from an area of rash to look for cutaneous manifestations of collagen vascular disease or infection. Enlarged lymph nodes should be aspirated or biopsied and examined for cytologic features to rule out neoplasm and send for culture.
U. Fluorodeoxyglucose (FDG)-PET/CT: performed to evaluate for lymphoma or other forms of cancer.

DIFFERENTIAL DIAGNOSES

A. Fever patterns can help the practitioner with diagnosis.
  1. FUO.
  2. Systemic and localized infections.
  3. Neoplasms.
  4. Autoimmune disorders.
  5. Thrombophlebitis.
  6. Miscellaneous causes (see "Definition" earlier in this section).
  7. Fever in clients older than 65 years:
    a. Autoimmune disorders including temporal arteritis, rheumatoid arthritis, and polymyalgia rheumatica.
    b. Infections including TB, infective endocarditis, intra-abdominal abscesses, and complicated UTIs.
    c. Malignancies especially hematologic malignancies.
    d. Drug-related fever.

PLAN

A. General interventions:
  1. Observe the client taking own temperature to document the presence of a fever to make sure the temperature is not self-induced. ▶

B. Client teaching:

1. Instruct the client to keep a record of temperatures, preferably rectal, at least morning and evening.

2. Reassure the client that there is nothing abnormal about temperatures in the range of 97.0°F to 99.3°F.

3. Instruct the client on use of physical cooling aids, such as cool towels or dampening clothing with tepid water.

4. Explain to the client that immersion in an ice water bath may be indicated for hyperthermic emergencies.

5. Instruct client on the importance of staying well-hydrated. Increase fluid intake.

C. Pharmaceutical therapy:

1. Start therapeutic trials if a diagnosis is strongly suspected:

a. Antituberculous drugs for TB.

b. Tetracycline for brucellosis.

c. Corticosteroids for temporal arteritis.

d. If the client shows no clinical response in 2 weeks, stop therapy and reevaluate.

2. Symptomatic antipyretic therapy: Salicylates or acetaminophen Q4H if not contraindicated. Nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen can be also be used.

FOLLOW-UP

A. Follow up in 24 to 48 hours.

B. Indications for admission to the hospital: Fever remains elevated beyond 101°F for weeks and ambulatory diagnostic efforts have been unsuccessful.

CONSULTATION/REFERRAL

A. Consult with a physician for diagnosis and comanagement if indicated.

B. Refer the client to a specialist if unable to differentiate definitive diagnosis.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Refer the client for perinatal consultation.

2. High fevers early in the first trimester have been associated with an increase in neural tube defects.

3. Maternal fevers may cause fetal tachycardia.

B. Geriatrics:

1. Common causes of FUO include TB, Hodgkin lymphoma, abscesses, and temporal arteritis.

2. Elderly clients commonly present with atypical symptoms.

a. Infection atypical presentation may include normal or below normal temperature, absence of tachycardia, or slightly elevated white blood cell count.

3. Elderly clients frequently present with altered mental status.

4. Elderly clients are at higher risk for developing dehydration with fever. Monitor closely for signs and symptoms of dehydration.

5. Review immunization status. Educate the client on the importance of being immunized for pneumococcus (PVS13 and PPVS23), COVID-19, and influenza annually.

6. Geriatrics' FUO must not be mistaken with end-of-life fever that often occurs during the final weeks or days of life. Although the origin is unknown, research suggests it is due to infection, neoplasm, or neurologic injury. The first-line agents for treatments are acetaminophen and NSAIDs for comfort palliative measures.

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HUMAN IMMUNODEFICIENCY VIRUS

DEFINITION

A. AIDS is a chronic, life-threatening condition caused by HIV. HIV is a retrovirus that targets helper T (CD4) cells and contains a viral enzyme called reverse transcriptase that allows the virus to convert its RNA to DNA, then integrate and take over the cell's own genetic material. Once taken over, the new cell begins to produce new HIV retrovirus. This process kills the CD4 cells that are the body's main defense against illness. This interferes with the body's ability to fight off infections—bacteria, viruses, and fungi—that cause disease. AIDS is the term used to define a severely compromised immune system.

EPIDEMIOLOGY

A. In 2019, 36,801 people were diagnosed with HIV in the United States. This represents a decrease of 9% from 2015. It is estimated that 1,189,700 people in the United States had HIV at the end of 2019. Approximately 87% of those individuals knew they had HIV. Of those diagnosed in 2019, 65% report male to male sexual contact as their risk factor, 23% report heterosexual sex as their risk factor, while another 7% report intravenous drug use as their primary risk factory. Additionally, Black people and Latinx people are disproportionately affected.

B. The widespread use of antiretroviral therapy has altered the course of HIV disease. In 2012, HIV was reclassified as a chronic illness. Approximately two-thirds of deaths in HIV-positive persons related to conditions other than HIV.

RISK FACTORS

A. Men who have sex with men.

B. Heterosexual contact with an infected partner, including commercial sex workers.

C. Needle sharing by intravenous drug users (IVDU).

D. Perinatal infection: mother-to-child transmission.

E. Open wound and mucous membrane exposure to body fluids of an infected person.

PATHOGENESIS

A. HIV belongs to a subgroup of retroviruses called lentiviruses or "slow" viruses. The course of infection of the virus is characterized by a long interval between infection and the onset of serious symptoms. CD4 cells are the primary target of HIV.

B. Primary HIV infection is followed by a burst of viremia during which the virus is easily detected in peripheral blood per HIV polymerase chain reaction viral load. During the "window period," the first 2 to 6 weeks following infection, persons may test negative for HIV on fourth-generation antigen/antibody tests. During this time, the person can be highly infectious to sexual partners. In this time of early infection with high viral load, the CD4 cells can decrease by 20% to 40%. Within 2 to 4 weeks after exposure to the virus, up to 70% of infected individuals experience a flulike illness related ▶

to acute infection. The immune system fights back to reduce the HIV levels with killer T cells (CD8) that attack and kill the infected cells. The individual's CD4 cell count may rebound by 80% to 90%. A person can remain symptom-free for a long time, often years. During this time, there is low-level replication of HIV but ongoing deterioration of the immune system. Enough of the immune system remains intact to prevent most infections. The individual is infectious during this time.

C. Advanced HIV occurs when a sufficient number of CD4 cells are destroyed and when production of new CD4 cells cannot match destruction. Clients exhibit fatigue, fever, weight loss, and susceptibility to opportunistic infections (OIs).

D. An HIV-infected person can live an average of 8 to 10 years before developing clinical symptoms. HIV disease is not uniformly expressed in all people. A small portion of individuals develop advanced HIV and have significant illness and even death within months of infection. Approximately 5% of those who are infected, known as "long-term nonprogressors," have no signs of disease after 12 or more years.

E. Most AIDS-defining conditions are marked by a CD4 count of less than 200 cells or the appearance of one or more of the OIs. Bacteria, viruses, or fungi that would not cause illness in a healthy immune system cause OI. These infections are often severe and sometimes fatal.

COMMON COMPLAINTS

A. Fatigue: often severe.
B. Fever: longer than 1 month.
C. Night sweats: drenching.
D. Loss of appetite.
E. Weight loss.
F. Rash.

OTHER SIGNS AND SYMPTOMS

A. Lymphadenopathy: enlarged lymph nodes often involving at least two noncontiguous sites.
B. Anemia.
C. Neutropenia.
D. Thrombocytopenia.
E. Cough.
F. Dyspnea.
G. Asymptomatic whitish patches on sides of tongue: hairy leukoplakia.
H. Thrush: oral candidiasis.
I. Odynophagia: esophageal candidiasis, cytomegalovirus (CMV), esophagitis.
J. Chronic vaginal candidiasis.
K. Skin changes: rashes, dry skin, and seborrheic dermatitis.
L. Purplish, nonblanching nodules found on the skin, mucous membranes, and viscera: Kaposi sarcoma.
M. Muscle wasting.
N. Chronic diarrhea: longer than 1 month.
O. Hepatosplenomegaly.
P. Cardiomyopathy.
Q. Chronic bacterial infections, including community-acquired pneumonias.
R. Tuberculosis (TB).
S. Sexually transmitted infections (STIs).
T. Peripheral neuropathy.
U. Dementia.

SUBJECTIVE DATA

A. Review symptoms: onset, course, and duration.
B. Ask about previous HIV testing: dates and reasons for testing.
C. Medical history review: hospitalizations, comorbidities, immunizations, normal weight, pain, chronic lymph node disorders, and any changes of skin overlying lymph nodes.
D. Surgical history review.
E. Sexual history: number of partners in the past year, number of lifetime partners, any previous partner known to be HIV-positive or have STIs, and any previous partners known to have been incarcerated:
  1. Females: history of abnormal Pap smears, contraception, and condom use with partner.
  2. Males: men having sex with men (MSM), heterosexual, bisexual, receptive anal intercourse, and condom use.
F. Mental health history: past and current mental health diagnosis and treatment.
G. Substance use: tobacco, alcohol, and drugs.
H. History of IVDU: needle sharing and when they last used drugs.
I. Transfusion of blood product history prior to 1985.
J. Lived or traveled outside of the United States: When and for how long?
K. Assess the presence of persistent fever with no localizing symptoms.
L. Assess the client's support system: Who knows about their diagnosis?

PHYSICAL EXAMINATION

A. Height, weight, blood pressure, pulse, respiratory rate, and temperature. Calculate the client's body mass index (BMI) at each office encounter.
B. General observation: general appearance, including fat distribution and signs of wasting.
C. Inspect:
  1. Skin: Evaluate for rashes, seborrhea, folliculitis, moles, Kaposi sarcoma, warts, herpes, dry skin, skin cancer, fungal infections, molluscum contagiosum, jaundice, and needle marks.
  2. Head and neck; eyes, ears, nose, and throat (HEENT):
    a. Assess visual acuity.
    b. Retina exam for CMV.
    c. Evaluate sclera for icterus.
    d. Oral exam for thrush, hairy leukoplakia, mucosal Kaposi sarcoma, gingivitis, aphthous ulcers, and dental health.
D. Auscultate:
  1. Pulmonary auscultation for air movement and abnormal breath sounds.
  2. Cardiac evaluation for normal and abnormal heart sounds.
E. Palpate:
  1. Palpate the thyroid.
  2. Lymphatic evaluation of regional versus generalized swelling: specific location, size, and texture of nodes.
  3. Palpate the abdomen to evaluate the presence of hepatosplenomegaly, masses, tenderness, pain, or rebound tenderness.
F. Rectal/vaginal examination:
  1. Males and females: Inspect for presence of ulcers and warts in the vagina, on the penis/testes perineum, and in the rectum.
  2. Females: Perform bimanual examination and Pap smear with HPV testing, obtain specimens for STI testing, and perform a digital rectal exam and anal Pap smear, if indicated.
  3. Males: Perform testicular exam and digital rectal exam; consider an anal Pap, if indicated. ▶

G. Neurologic examination:
  1. Assess mental status.
  2. Assess cranial nerves, including gait, strength, and deep tendon reflexes (DTRs); evaluate proprioception, vibration, pinprick, temperature, and sensation in the distal extremities.
H. Psychiatric examination:
  1. Screen for depression.

DIAGNOSTIC TESTS

A. HIV antigen/antibody tests.
B. Complete blood count (CBC) with differential, including platelets.
C. Complete chemistry profile.
D. Fasting lipid profile.
E. Venereal Disease Research Laboratory and rapid plasma reagin.
F. Serologies for toxoplasmosis.
G. CD4/CD8 cells and CD4/CD8 ratio.
H. HIV RNA viral load.
I. HIV resistance genotype.
J. HLA-B 5701 (risk for abacavir hypersensitivity reaction syndrome).
K. Hepatitis serologies: hepatitis A virus (HAV) serology (antibody), hepatitis B virus (HBV) serology (HBsAb, HBsAg, HBcAb), and hepatitis C virus (HCV) serology (antibody).
L. Cultures for STIs: anal and vaginal (culture pharynx if history indicates oral sex).
M. Urinalysis.
N. Pap smear with human papillomavirus (HPV) testing.
O. TB screening.

DIFFERENTIAL DIAGNOSES

A. HIV.
B. Other diseases that lead to immune suppression or are related to symptoms.
C. Cancer.
D. Chronic infections.
E. TORCH infections:
  1. T: toxoplasmosis
  2. O: others (syphilis, hepatitis B)
  3. R: rubella
  4. C: cytomegalovirus
  5. H: herpes simplex
F. Syphilis.
G. TB.
H. Endocarditis.
I. Infectious enterocolitis.
J. Bowel disorders: antibiotic-associated colitis, inflammatory bowel disease, or malabsorptive symptoms.
K. Endocrine diseases.
L. Neuropathy.
M. Alcoholism.
N. Liver disease.
O. Renal disease.
P. Thyroid disease.
Q. Vitamin deficiency.
R. Chronic meningitis.

PLAN

A. General interventions:
  1. Refer and comanage the client with HIV/AIDS clinician.
  2. Identify and refer for treatment of substance abuse.
  3. Explain to the client that each visit will include a review of history, a physical exam, and laboratory studies to assess health status.
  4. Discuss health habits: smoking, nutrition, and exercise.
  5. Discuss treatment plan:
    a. highly active antiretroviral therapy (HAART).
    b. Therapy for OI and malignancies.
    c. Prophylaxis for OI: pneumocystis pneumonia (PCP) and *Mycobacterium avium* complex (MAC).
    d. Management of side effects of medications and comorbidities.
    e. Immunizations.
B. Client teaching: *See Client Teaching Guide for this chapter, "Reference Resources Regarding HIV/AIDS."*
  1. Discuss living with HIV disease, including daily adherence to antiretrovirals.
  2. Discuss transmission prevention strategies: safer sex practices, condom use, Undetectable = Untransmittable, and partner preexposure prophylaxis (PrEP).
  3. Provide contact information for AIDS social service organizations (ASO).
  4. Discuss the client's concerns, including notification of sexual partner(s) and needle-sharing partner(s).
C. Pharmaceutical therapy:
  1. Treatment with HAART: The Department of Health and Human Services (DHHS) guidelines on the treatment of HIV/AIDS recommended HAART for all individuals infected with HIV regardless of CD4 cell count.
  2. Polypharmacy is one of the biggest challenges in caring for the aging population. The average HIV-negative client older than 50 years old takes an average of five medications. These added to three to five or more HIV medications complicate monitoring for medication interaction and side effects. Comorbidities, existing medications, and potential organ toxicities should all be considered in choosing a HAART regimen.
  3. HAART (Public Health Service Task Force Guidelines for the Use of Antiretroviral Agents in HIV-1 Infected Adults and Adolescents) classes:
    a. Nucleoside/nucleotide reverse transcriptase inhibitors (NRTIs).
    b. Nonnucleoside reverse transcriptase inhibitors (NNRTIs).
    c. Protease inhibitors (PIs).
    d. Integrase inhibitors (INIs).
    e. Fusion inhibitors (FIs).
    f. C-C chemokine receptor type 5 (CCR5) antagonists.
    g. Postattachment inhibitors.
  4. Prophylaxis and treatment for OIs: may discontinue prophylaxis if sustained immune reconstitution on HAART:
    a. PCP: Prophylaxis if CD4 count is less than 200:
      i. First-line: Trimethoprim-sulfamethoxazole (TMP-SMZ; Bactrim DS), one tablet three times weekly or one tablet daily (if toxoplasmosis is positive).
      ii. Alternatives: dapsone, atovaquone, and aerosolized pentamidine.
    b. MAC: Prophylaxis is no longer indicated if client is starting HAART immediately. If not starting HAART and CD4 <50, recommend prophylaxis.
      i. First line: zithromax 1,200 mg weekly.
      ii. Alternative: clarithromycin.
  5. Managing the side effects of HAART, complications of HIV therapy, and aging population: There are multiple complications of long-term HIV infection and treatment with HAART. Age-related decrease in renal and hepatic ▶

function should be considered when choosing a HAART regimen. These problems can lead to higher levels of HIV medications and increased toxicities. There are no guidelines for dose adjustment based on age, but renal and hepatic function should be monitored on an ongoing basis and adjustments made accordingly. Other aspects of HIV management include immunizations (live virus vaccines contraindicated unless CD4 count >200—varicella; measles, mumps, rubella [MMR]):

a. Hepatitis A and B series.
b. Tetanus.
c. Pneumococcal vaccines.
d. Yearly influenza vaccines.
e. Tdap booster as needed.
f. Shingles vaccine (inactivated recombinant vaccine).

6. Complications associated with both HIV-positive and with older populations requiring diligent monitoring:

a. Osteoporosis: HIV/AIDS clients are at high risk for decreased bone density.
b. Hypogonadism is common in males with AIDS.
c. Neuropsychiatric disorders: Depression, anxiety, and substance use are common in the HIV population. There is increased risk for HIV-related dementia.
d. Peripheral neuropathy is a common side effect with older HIV medications.
e. Cardiovascular disease (CVD): HIV-related disorders such as insulin metabolism, diabetes, lipodystrophy, dyslipidemia, and hypertension. CVD complications are among the leading causes of death in HIV disease.
f. Malignancies: Liver, anal, cervical, and lung cancers are common in people living HIV/AIDS.

FOLLOW-UP

A. Schedule return visit 4 to 6 weeks after initial visit to discuss staging HIV/AIDS and living with HIV/AIDS.

1. More frequent visits are needed with a new diagnosis and initiation of HAART. Once HIV is suppressed, less frequent visits are required, generally every 4 to 6 months.

B. TB screening at least one, and then based on client risk.

C. Pap smear:

1. Client <30 years of age: annual cytology if normal. After three annual normal cytology results, next Pap smear indicated in 3 years.

2. Client 30 years or older: cytology and HPV cotesting. If normal and HPV-negative, next Pap smear indicated in 3 years.

3. If abnormal, follow guidelines by the American Society of Colposcopy and Cervical Pathology (ASCCP).

D. Pap smears for clients as indicated.

E. Urinalysis yearly if taking a tenofovir-containing regimen.

F. STI screening initially, then based on risk.

G. Other annual screening as per the U.S. Preventive Services Task Force (USPSTF) and HIV/AIDS treatment guidelines (AIDSinfo.NIH.gov/guidelines).

CONSULTATION/REFERRAL

A. Refer the client to a specialist in HIV for management of continued care and pharmacologic therapy.

B. Refer the client to a nutritionist for baseline evaluation and diet counseling.

C. Perform vision screening yearly.

D. Require dental care every 6 months.

E. Make hepatology referral if chronic, active hepatitis B and/or C. Truvada is approved for HBV treatment.

INDIVIDUAL CONSIDERATIONS

A. PrEP is a prevention method for HIV-negative individuals:

1. Truvada and Descovy are Food and Drug Administration (FDA)-approved oral tablets for use as PrEP among heterosexual and MSM HIV-negative individuals who are males (Truvada only for females). One tablet is taken daily or episodically to reduce the risk of infection.

2. Apretude is FDA-approved for use as PrEP. It is an injectable medication given every 1 to 2 months to prevent HIV infection in HIV-negative individuals.

3. Those taking PrEP should be monitored for potential side effects.

B. Postexposure prophylaxis (PEP):

1. PEP: Depending on exposure type and severity, postexposure antiretroviral treatment (ART) should be started as quickly as possible and be taken for 4 weeks:

a. Expert consultation should be obtained as quickly as possible (National HIV/AIDS Clinician's Consultation Center PEPline, 1-888-448-4911).
b. Follow with occupational health for regular clinical assessment and labs.
c. Perform HIV testing with HIV RNA, viral load, if an illness compatible with seroconversion illness occurs (fever, lymphadenopathy, pharyngitis, and rash).
d. Advise transmission precautions during the first 3 to 6 months postexposure (use condoms, refrain from donating blood, etc.).

C. Pregnancy:

1. Refer or work very closely with the HIV obstetrics specialist:

a. Prenatal HIV antiretroviral therapy.
b. Intrapartum issues, for example, rupture of membranes and mode of delivery.
c. Antiretrovirals (PEP) for the neonate.
d. Ruling out HIV in the infant born to an HIV+ mother.

D. Geriatrics:

1. Seniors are at increased risk for STIs due to a weaker immune system, hormone changes, and increased vaginal friability.

2. Medicare offers free STI screening and treatment for seniors.

3. Clinicians need to incorporate a full sexual history when assessing senior clients:

a. Lack of sexual education: Older adults are less likely to perceive themselves at risk. Seniors may feel sex education is only directed to youth and those wishing to prevent pregnancy.
b. Medications for erectile dysfunction have contributed to an increase in sexual activity throughout the older years of female clients.
c. Postmenopausal clients, without concern for pregnancy, may not feel that condoms are required with sexual intercourse.

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IMMUNE THROMBOCYTOPENIA

DEFINITION

A. Immune thrombocytopenia (ITP) is an autoimmune disorder in which an immunoglobulin G (IgG) autoantibody is formed that binds to platelets. Platelets help your blood clot by clumping together to plug small holes in damaged blood vessels. The platelet count is less than 150,000 platelets per mL.

1. Newly diagnosed ITP defined as duration of <3 months.

2. Persistent ITP defined as duration of 3 to 12 months; more common in children.

3. Chronic ITP defined as duration >12 months.

INCIDENCE

A. Persistent ITP commonly occurs in childhood, frequently precipitated by a viral infection. Peak age of incidence is between 20 and 50 years. There is a 2:1 female-to-male predominance; however, the prevalence in males and females is equal in the elderly 65 or older. The adult form is usually a chronic disease (>12 months) and seldom follows a viral infection.

PATHOGENESIS

A. ITP is due to production of antiplatelet antibodies that lead to peripheral destruction and sequestration of platelets. It is not clear which antigen on the platelet surface is involved. Platelets are not destroyed by direct lysis even if the antiplatelet antibody binds complement. Destruction takes place in the spleen, where splenic macrophages with Fc receptors bind to antibody-coated platelets.

PREDISPOSING FACTORS

A. Infections including hepatitis C, Epstein–Barr virus, and cytomegalovirus.

B. Chronic alcoholism.

C. Sepsis.

D. AIDS.

E. Immune disorders, such as systemic lupus erythematosus (SLE).

F. Drug use.

G. Chronic lymphocytic leukemia (CLL).

H. Pregnancy.

I. *Helicobacter pylori*.

COMMON COMPLAINTS

A. Purple spots and bruises on skin.

B. Nosebleeds.

C. Mouth and gum bleeding.

OTHER SIGNS AND SYMPTOMS

A. Purpura, petechiae, and hemorrhagic bullae in the mouth.

B. Tendency to bleed easily: easy bruising.

C. Menorrhagia: abnormal heavy menstruation.

D. No systemic illness: The client feels well and is not febrile.

SUBJECTIVE DATA

A. Determine when the client or caregiver first noticed symptoms; note if the symptoms have changed or progressed.

B. Rule out pregnancy as the cause of nosebleeds.

C. Ask if the client is feeling well except for the bleeding or bruising.

D. Determine if the client has a fever.

E. Obtain medication history for over-the-counter (OTC) and prescribed medications.

F. Determine the client's history of immune disorders, recent infections, alcoholism, and pregnancy.

G. Establish usual weight and any recent weight loss.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure, and weight.

B. Inspect.

1. Observe general appearance.

2. Conduct dermal exam for purpura and petechiae.

3. Conduct eye exam; check sclera for hemorrhages.

4. Examine the mouth for dental caries and poor hygiene, mouth petechiae, and hemorrhagic bullae.

C. Palpate:

1. Normally, the spleen should not be palpable.

2. Palpate the abdomen, liver, and spleen.

3. Palpate the cervical, axillary, and groin lymph nodes for adenopathy.

D. Percuss:

1. Percuss the abdomen, liver, and spleen.

E. Auscultate:

1. Auscultate the heart and lungs.

2. Auscultate the abdomen for bowel sounds and bruits.

DIAGNOSTIC TESTS

A. Platelet count: The major concern during the initial phase is risk of cerebral hemorrhage when platelet count is less than 5,000 platelets/fiL.

B. Complete blood count (CBC) with differential and peripheral smear: Peripheral smear shows normal white blood cells (WBCs) and red blood cells (RBCs); platelets low in count with large size.

C. Bleeding time is prolonged.

D. Tests for HIV and hepatitis C.

E. Screen for *H. pylori* once diagnosed with ITP.

F. Referral for bone marrow aspiration if not responding to therapy. Bone marrow may appear normal or have increased megakaryocytes (early form of platelets) with thrombocytopenia.

DIFFERENTIAL DIAGNOSES

A. Thrombocytopenia may be produced in two ways: by abnormal bone marrow function or by peripheral destruction of platelets.

1. Abnormal bone marrow function:
- a. Aplastic anemia.
- b. Hematologic malignancies.
- c. Myelodysplasia: This can only be ruled out by examining the bone marrow.
- d. Megaloblastic anemia.
- e. Chronic alcoholism.

2. Non-bone marrow disorders:
- a. Immune disorders:
  - i. ITP.
  - ii. Drug induced from medications being taken.
  - iii. Secondary to CLL and SLE.
  - iv. Posttransfusion purpura.
- b. Hypersplenism resulting from liver disease.
- c. Disseminated intravascular coagulation (DIC).
- d. Thrombotic thrombocytopenic purpura.
- e. Sepsis.
- f. Hemangiomas.
- g. Viral infection, AIDS.
- h. Pregnancy.
- i. Hypothyroidism.

PLAN

A. General interventions:

1. Comanage the client with a physician for medication therapy.

B. Client teaching:

1. Instruct the client to avoid trauma; advise no contact sports.

2. Instruct the client to avoid salicylates or nonsteroidal anti-inflammatory drugs (NSAIDs).

3. Corticosteroid therapy benefits:
- a. Corticosteroids increase platelet count by increasing platelet production.
- b. Long-term therapy may decrease antibody production.
- c. Bleeding often diminishes 1 day after beginning prednisone.
- d. Platelet count usually begins to rise within a week; responses are almost always seen within 3 weeks.
- e. About 80% of clients respond, and the platelet count usually returns to normal.

C. Medical or surgical management:

1. Splenectomy is considered at least 1 year after initial diagnosis and when medical management has failed.

2. Two to 10 weeks prior to the splenectomy, clients need to be immunized with pneumococcal conjugate vaccine (PCV13), *Haemophilus influenzae* type B, meningococcal conjugate, and meningococcal B vaccines. Clients will receive pneumococcal polysaccharide vaccine (PPSV23) 8 weeks after the splenectomy and a second dose 5 years later. Annual influenza immunization is recommended.

D. Pharmaceutical therapy:

1. Initial treatment:
- a. For clients with a platelet count >30,000 platelets/fiL and are either asymptomatic or have minor mucocutaneous bleeding, observation is recommended.
- b. For clients with a platelet count <30,000 platelets/fiL, prednisone 0.5 to 2.0 mg/kg/d up to 6 weeks or dexamethasone 40 mg/kg × 4 days.
- c. For clients with a platelet count <20,000 platelets/fiL, consider hospital admission rather than outpatient management.
- d. For clients with active bleeding, intravenous immunoglobulin (IVIG) can be used with corticosteroids to achieve a rapid increase in platelet count.
  - i. IVIG dose is initially 1g/kg as a one-time dose; this dose can be repeated if necessary.
- e. For clients with secondary ITP due to hepatitis C virus infection, start antiviral therapy and closely monitor platelet count.
- f. For clients with HIV-associated ITP, begin antiretroviral therapy.
  - i. If actively bleeding, initial treatment with corticosteroids and IVIG.
  - ii. If symptomatic and fail corticosteroids, consider splenectomy.

2. Second-line drug therapy:
- a. Persistent ITP and dependent on or unresponsive to corticosteroids.
- b. Thrombopoietin receptor agonist (TPO-RA) if client places a high value on achieving a durable response.
- c. Rituximab recommended if the client places a high value on avoiding long-term medication therapy.
  - i. Screen for hepatitis B virus prior to initiating rituximab.

3. Chronic ITP:
- a. TPO-RA for a client who places a high value at avoiding surgery or places a high value on achieving a durable response.
- b. Rituximab for a client who places a high value on avoiding surgery or places a high value on avoiding long-term medication therapy.
- c. Splenectomy for a client who places a high value on avoiding long-term medication therapy or places a high value on achieving a durable response.

4. Platelet transfusions are an option rarely used.

5. Avoid aspirin, NSAIDs, and warfarin, which interfere with platelet function and blood clotting. Be aware if the client is also taking any new selective inhibitors of factor Xa such as Xarelto, Pradaxa, or Eliquis, and so forth.

6. Exogenous platelets survive no better than the client's own platelets. In many cases, platelets survive less than a few hours. This therapy is reserved for cases of life-threatening bleeding in which enhanced hemostasis for even an hour may be of benefit.

FOLLOW-UP

A. The client must be monitored very closely. Platelet counts should be drawn daily to weekly; frequency depends on the severity and course.

B. Clients receiving corticosteroids require monitoring for hypertension, hyperglycemia, myopathy, insomnia, depression, fatigue, gastric irritation or ulcer formation, and osteoporosis.

C. Prognosis for acute ITP: 80% respond and fully recover within 2 months; 15% to 20% progress to chronic ITP.

D. Prognosis for chronic ITP: 10% to 20% recover fully; the remainder continue to have low platelet counts and may see a remission or relapse over time.

E. The principal cause of death from ITP is intracranial hemorrhage.

CONSULTATION/REFERRAL

A. After diagnosis, refer the client to a hematologist within 24 to 72 hours.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Rule out hemolysis, elevated liver enzymes, low platelets (HELLP) syndrome, infection, and DIC as causes of thrombocytopenia.

2. There is an increased incidence of spontaneous abortions and hemorrhage at the time of delivery from genital tract injury.

3. Antepartum management:

a. Conduct fetal blood sampling and testing when the mother has a known history of ITP.

4. Intrapartum management:

a. Avoid fetal hypoxia, which can decrease the fetal platelet count.

b. Avoid prolonged labor.

c. Conduct continuous fetal monitoring.

d. Epidural anesthesia may be used if the platelet count is at least 100,000 cells/mm^3.

5. Postpartum management:

a. Breastfeeding is not recommended due to the possible transmission of antiplatelet antibodies through breast milk.

B. Geriatrics:

1. ITP is uncommon in this population; there is usually another cause for low platelets.

2. Avoid medications that impede platelet function.

3. Research posits that geriatrics with ITP respond well to low-dose prednisone as first-line treatment.

4. Studies suggest that once geriatrics are diagnosed with ITP, they experience approximately two bleeding-related events a year. These statistics support the importance of examining bleeding and use of ITP therapy in the elderly population suffering with ITP.

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IRON-DEFICIENCY ANEMIA (MICROCYTIC, HYPOCHROMIC)

DEFINITION

A. Microcytic anemia is characterized by small, pale red blood cells (RBCs) and depletion of iron (Fe) stores. The hematocrit (Hct) is less than 41% in males, with a hemoglobin (Hgb) less than 13 g/dL. In females, the Hct is less than 37%, with a Hgb less than 12 g/dL. Usually mild, it can become moderate or severe.

INCIDENCE

A. Iron deficiency is the most common cause of anemia worldwide; it is particularly prevalent in females of childbearing age. It is estimated to occur in 29% of adult females, 20% of individuals 80 years of age and older, 38% of those who are pregnant, and 3% of adult males in the United States. Iron deficiency is estimated to affect 37% to 61% of individuals with chronic heart failure, 24% to 85% of those with chronic kidney disease, and 13% to 90% of those with inflammatory bowel disease. Prevalence rates are highest among African American and Hispanic populations.

PATHOGENESIS

A. Anemia is acquired; it develops slowly and in stages. Iron loss exceeds intake so that stored iron is progressively depleted. As stored iron is depleted, a compensatory increase in absorption of dietary iron and in the concentration of transferrin occurs. Iron stores can no longer meet the needs of the erythroid marrow; the plasma transferrin level increases and the serum iron concentration declines, resulting in a decrease in iron available for RBC formation.

PREDISPOSING FACTORS

A. Female with history of heavy menses.

B. Chronic blood loss, gastrointestinal (GI) blood loss.

C. Family history or personal history of anemia.

D. Poor diet, including strict vegetarian diets.

E. Closely spaced pregnancies.

F. Pica.

G. Chronic hemoglobinuria due to an abnormally functioning cardiac valve.

H. Chronic aspirin use, anticoagulant, or nonsteroidal anti-inflammatory drug (NSAID) use.

I. Repeated blood donation.

J. Decreased iron absorption due to gastric surgery.

K. Taking too many antacids that contain calcium.

L. Surgeries including gastric bypass surgery, orthopedic surgeries, abdominal surgeries, and cardiac surgeries.

M. GI problems including inflammatory bowel disease, *Helicobacter pylori*, autoimmune gastritis, and celiac disease.

N. Chronic kidney disease.

O. Hookworms, whipworms.

P. Chronic use of proton pump inhibitors (PPIs) or histamine 2 antagonist.

Q. Endurance athletes.

R. Heart failure.

COMMON COMPLAINTS

A. Fatigued all of the time.

B. Heart feels like it is racing, beating hard.

C. Out of breath on exertion.

D. Loss of appetite, nausea, and vomiting.

E. Headaches throughout day.

F. Weak and dizzy.

G. Cold hands and feet.

OTHER SIGNS AND SYMPTOMS

A. Clinical presentation depends on severity, the client's age, and the ability of the cardiovascular and pulmonary systems to compensate for the decreasing oxygen-carrying capacity of the blood.

B. Initial: Exercise-induced dyspnea and mild fatigue symptoms may be minimal until the client has significant anemia.

C. As Hct falls, dyspnea and fatigue increase:

1. Malaise.

2. Drowsiness.

3. Sore tongue and mouth.

4. Skin pallor.

5. Pale mucous membranes and conjunctiva.

▶

6. Pale fingernail beds.
7. Tachycardia.
8. Palpitations.
9. Tinnitus.
10. Restless legs syndrome.

D. Severe anemia:
1. Atrophic glossitis, cheilitis (lesions at the corner of the mouth).
2. Koilonychia (thin concave fingernails with raised edges).

SUBJECTIVE DATA

A. Inquire about the onset, course, and duration of symptoms.
B. Ask the client about history of GI bleeding.
C. Take careful history of GI complaints that might suggest gastritis, peptic ulcer disease, or other conditions that might produce GI bleeding.
D. Ask if there has been a change in stool color or bleeding from hemorrhoids.
E. In menstruating clients, ask about blood loss during menses.
F. Ask about dietary intake of iron-rich foods and pica.
G. Obtain medication history, especially use of aspirin, anticoagulants, and other NSAIDs.
H. Rule out history of anemia, blood-clotting problems, sickle cell disease, glucose-6-phosphate dehydrogenase (G6PD) deficiency, or other hereditary hemolytic diseases.
I. Review occupation and activities with exposure to lead or lead paint.
J. Has the client experienced palpitations, chest pain, dizziness, or shortness of breath, especially on exertion or activity?
K. Ask about symptoms of severe anemia, including chest pain, dyspnea at rest, and palpitations.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure, and weight. Check for postural hypotension.
B. Inspect:
1. Inspect general appearance; the client may be pale, lethargic, or without overt signs if anemia is mild.
2. Conduct eye exam; check conjunctivae for paleness.
3. Examine oral mucosa and corners of the mouth (for cheilitis); note appearance of the tongue (atrophy of the papillae; smooth, shiny, beefy red appearance), angular stomatitis, or pale gums.
4. Examine the skin for dryness and perfusion.
5. Examine the nails for brittleness, flattening, ridges, and concave or spoon shape.

C. Palpate:
1. Palpate the abdomen for tenderness and enlargement of the liver and spleen.

D. Percuss:
1. Percuss the abdomen.

E. Auscultate:
1. Auscultate the heart for systolic flow murmurs, tachycardia.

F. Rectal exam:
1. Assess for masses and obtain stool for occult blood.

DIAGNOSTIC TESTS

A. Initial screening:
1. Complete blood count (CBC) with differential and peripheral smear.
2. Serum ferritin level.
3. Serum iron concentration: absent iron stores equal ferritin value less than 30 ng/mL.
4. Colonoscopy and endoscopy for postmenopausal females, premenopausal females with overt blood loss, and males.

B. Follow-up testing:
1. Serum iron, total iron-binding capacity (TIBC), retic count, and RBC indices.
2. TIBC elevated; serum ferritin decreased.
3. Urinalysis.
4. Consider screening for celiac disease.
5. Hct 3 to 4 weeks after treatment. Treatment should continue for at least 4 to 6 months after the Hct returns to normal.
6. Ultrasonography or CT scan, if indicated.

DIFFERENTIAL DIAGNOSES

A. Iron-deficiency anemia.
B. Inadequate intake of iron.
C. Any condition that causes acute or chronic blood loss.
D. Hemolytic diseases such as sickle cell and G6PD deficiency.
E. Anemia of chronic disease, thalassemia, and sideroblastic anemias.
F. Lead poisoning.
G. Neoplasm.

PLAN

A. General interventions:
1. Identify source of anemia. Discuss dietary sources of iron-rich foods. Discuss foods to avoid because they impair absorption.

B. Client teaching: *See Client Teaching Guide for this chapter, "Iron-Deficiency Anemia":*
1. Stress the importance of dietary intake of foods high in iron, which is absorbed better than most vitamins. Foods rich in vitamin C or ascorbic acid 500 mg daily increases absorption of iron. Tea and milk reduce iron absorption.
2. Provide iron supplements. Dose according to iron lab values.
3. Encourage the client to stop smoking.

C. Pharmaceutical therapy:
1. Oral iron replacement:
   a. Adults:
      i. Begin 6-month trial of ferrous sulfate tablets 300 to 325 mg daily or every other day before meals or with orange juice 1 hour before meals. Food decreases iron delivery by 50%. Recent research shows daily dosing divided into multiple doses results in poorer absorption of iron.
      ii. Iron supplement (Slow Fe) one time-release capsule daily.
      iii. Niferex 150 Forte one daily or more as required by follow-up iron levels.
2. Alternative drug therapy: Parenteral iron is indicated if the client cannot tolerate or absorb oral iron or if iron loss exceeds oral replacement:
   a. Iron dextran injection (Imferon).
   b. Iron sorbitex injection (Jectofer):
      i. Parenteral iron therapy is expensive.
      ii. It is associated with significant side effects: anaphylaxis, phlebitis, regional adenopathy, serum sickness-type reaction and staining of intramuscular (IM) injection sites. Test dose given prior to the first injection.
      iii. Dose is based on the client's weight.
      iv. Do not administer parenteral iron along with oral iron.

v. Use z-track method when administering injection. Iron stains the skin.

FOLLOW-UP

A. Follow-up of adults is variable, depending on source of blood loss. Manage signs of anemia.

B. It is advisable to see the client after 3 to 4 weeks, both to monitor the hematologic response and to answer questions about the medication, which may result in improved compliance. Check CBC and reticulocyte count.

C. If the client's Hgb level has increased by 1.0 g/dL in 3 to 4 weeks, continue iron supplementation for an additional 3 to 6 months. The Hct should return to normal after 2 months of iron therapy. However, keep taking iron supplements for another 6 to 12 months to replace the body's iron stores in the bone marrow.

D. Determine the effectiveness of iron replacement therapy by checking ferritin every 3 months for a year. Target normal ferritin is greater than 100 mcg/L.

CONSULTATION/REFERRAL

A. Refer the client to a specialist for the following:

1. Hgb is not increased by 1.0 g/dL after 1 month of treatment.

2. The therapeutic trial should not be continued beyond 1 month because the Hgb concentration has not increased and client compliance with recommended regimen is not a factor.

3. There is a steady downward trend in Hct despite treatment.

4. There is a significant drop in Hct over previous readings (rule out lab error first).

5. Lab findings show Hgb less than 9.0 g/dL or Hct less than 27%. Suspect underlying inflammatory, infectious, or malignant disease.

B. Refer the client for nutrition consultation, if indicated.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Take a multivitamins with 30 mg of iron daily.

2. Check Hct at initial prenatal visit, 28 weeks, and 4 weeks after initiating therapy.

3. Lab findings of Hgb greater than 13 g/dL and Hct greater than 40% may indicate hypovolemia. Be alert for signs of dehydration and preeclampsia.

4. Counsel the client on proper diet and refer to a dietitian.

5. Recommend one to two iron tablets a day in addition to prenatal vitamins containing iron.

6. If unable to tolerate vitamins, recommend children's Chewable Flintstones vitamins with iron, two orally, daily.

B. Adults:

1. Bleeding is the usual cause of anemia in adults. In adult males and postmenopausal females, bleeding is usually from the GI tract.

2. In premenopausal females, menstrual loss may be the underlying cause of anemia.

3. Smokers may have higher Hgb levels; therefore, anemia may be masked if standard Hgb levels are used.

C. Geriatrics:

1. Serum ferritin below 50 mcg/L should be investigated.

2. The highest prevalence of anemia is found in elderly clients and long-term care residents. Geriatrics taking iron supplements to treat anemia often complain that it has made their constipation challenges worse.

3. Studies indicate that elderly clients with low Hgb/iron levels will benefit from a diet rich in iron-dense foods and in some cases correct iron deficiencies. The highest bioavailability of iron is found in meats or meat products such as giblets, sausage, and liver. Good sources of plant-based dense iron foods are oat bran, black strap molasses, and legumes (chickpeas, lentils, dried peas). Encourage alternative measures of iron-dense foods for anemic geriatric clients and evaluate iron and Hgb levels after 4 to 6 weeks. If the Hgb level has not increased by 1.0 g/dL in 4 to 6 weeks, then begin a low-dose iron replacement synergistically with the iron-dense foods and continue to monitor lab levels per protocol. Some elderly clients have remained well-controlled with diet and iron supplement 3 days a week, which decreased the constipation side effect.

4. Research indicated that geriatrics with anemia related to pathophysiologic abnormalities or chronic disease (and *not* iron-deficiency anemia) correlated with symptoms of depression, reduced sense of well-being, anxiety, energy limitations, social withdrawal, and reduction of quality of life. Those most affected with these secondary symptoms were geriatrics with advanced congestive heart failure (CHF) and those receiving palliative care. Be sure to implement a full psychosocial assessment with this specific population and refer as needed.

5. Undifferentiated anemia of the elderly (UAE): This unexplained phenomenon presents no evidence of B_{12}/iron deficiency, normal iron studies, CR clearance greater than 30 mL/min, inflammatory markers (C-reactive protein [CRP]) that are not elevated, and yet erythropoietin levels are low for the degree of anemia. This occurs in almost one-third of all anemic clients ≥65 years old. It may be due to age-related decline of hematopoietic reserve, and at least 25% will evolve to myelodysplastic syndrome (MDS). Geriatric clients with UAE must be monitored for MDS due to the high risk for anemic complications, infections, and bleeding. Refer to a hematologist if you suspect an MDS diagnosis.

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LYMPHADENOPATHY

DEFINITION

A. Lymphadenopathy is enlargement of a lymph node, manifested in benign, self-limiting diseases and in those that are incurable and fatal. Only small lymph nodes in the neck, axilla, and groin are palpable in normal individuals. Palpable nodes in other regions, or any node exceeding 0.5 cm in size, are potentially abnormal. The body has approximately 600 lymph nodes. There are different categories of lymphadenopathy: ▶

1. Localized adenopathy.
2. Hilar adenopathy.
3. Generalized lymphadenopathy.
4. Other lymphatic abnormalities that present in other ways, such as lymphangitis, lymphadenitis, and lymphedema.

INCIDENCE

A. Lymphadenopathy is a very common presenting symptom. Age is an important diagnostic factor: In clients younger than age 30, the cause proves to be benign in 80% of cases; in clients older than age 50, the rate of benign disease falls to 40%. In primary care clients with unexplained lymphadenopathy, approximately three-fourths of clients will present with localized lymphadenopathy and one-fourth with generalized lymphadenopathy.

PATHOGENESIS

A. Inflammation and infiltration are responsible for pathologic enlargement. Localized lymphadenopathy may represent the spread of disease from an area of drainage. The left supraclavicular node is referred to as the "sentinel" node, which is in contact with the thoracic duct and drains much of the abdominal cavity. The right supraclavicular node drains the mediastinum, lungs, and esophagus. Generalized lymphadenopathy often results from infection, malignancy, hypersensitivity, and metabolic disease. Ninety percent of palpable supraclavicular nodes are a malignancy in clients 40 years of age or older.

PREDISPOSING FACTORS

A. High risk of exposure to HIV.
B. Occupational exposure.
C. History of pharyngitis, upper body infections (head and neck), or intraoral infection.
D. Exposure to animals: cats, sheep, cattle, rodents, deer ticks.
E. Recent travel, including foreign travel.
F. Exposure to bird droppings.
G. Lacerations sustained from gardening.
H. Exposure to tuberculosis (TB).
I. History of sexual exposure resulting in sexually transmitted infections (STIs).
J. History of tobacco abuse, intravenous (IV) substance abuse.
K. Cancer.
L. Anticonvulsant drugs that cause skin rash, fever, hepatosplenomegaly, and eosinophilia; for example, phenytoin (Dilantin) and carbamazepine.
M. Antibiotics including cephalosporins and penicillins.
N. Other medications:
1. Hydralazine.
2. Para-aminosalicylic acid.
3. Allopurinol.
4. Captopril.
5. Atenolol.

COMMON COMPLAINTS

A. Sore throat.
B. Fever.
C. Fatigue and malaise.
D. Loss of appetite.
E. Unintentional weight loss.
F. Swollen, painless lumps in neck.

OTHER SIGNS AND SYMPTOMS

A. May feel "good" except for finding enlarged lymph node.
B. Node location(s). For inguinal enlargement, rule out conditions that may resemble inguinal or femoral lymphadenopathy: hernias; ectopic testicular, endometrial, or splenic tissue; lipomas; varices; and aneurysms. If inguinal area is painful and tender, it is most frequently caused by STIs.
C. Skin rash.
D. Bruising or petechiae.
E. Pruritus.
F. Erythema of skin or scalp.
G. Skin eruption.
H. Night sweats.
I. Enlarged and tender abdomen, abdominal pain.
J. Joint pain.
K. Significant fever, night sweats, and unexplained weight loss are known as "B symptoms" and suggest lymphoproliferative disorders.
L. Painful nodes after drinking alcohol suggests Hodgkin lymphoma.

SUBJECTIVE DATA

A. A comprehensive and detailed history is necessary for diagnosis (see the "Predisposing Factors" section). Review the onset, course, and duration of symptoms.
B. What does the client note with respect to location, tenderness or painfulness, softness or hardness, and mobility of lymph nodes? Has the client noticed more than one enlarged lymph node?
C. Review the client's history of IV drug use. Review risk factors for HIV.
D. Review history for hobbies, specifically gardening, eating undercooked meat, and camping, and occupation (see "Toxoplasmosis" and "Lyme Disease" sections of Chapter 19, "Infectious Disease Guidelines").
E. Review medications: prescription, over-the-counter (OTC), and herbal remedies.
F. Determine whether the client has a fever or a known valvular heart disease.
G. Review any other associated symptoms or signs.
H. Review any recent exposure to family and friends with infections. Assess current living conditions. Has the client had recent immunizations?
I. Review recent dental problems or abscessed teeth.
J. Note whether the client is a smoker. If so, how much, for how long, and when did the client quit smoking?
K. Review the client's history for recent cat scratches.
L. Review the client's history for recent travel.
M. Review the client's history for new sexual partners to rule out STIs.
N. Review usual weight and any recent weight loss, noting how much over what period of time.
O. Elicit information about similar symptoms in the past, when they occurred, how they were treated (antibiotics, biopsy), and the success of the treatment.
P. Elicit information about alcohol intake, noting how much, how long, and, if the client has quit, how long ago.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure, and weight.
B. Inspect:
1. Conduct a funduscopic exam.
2. Examine the eyes, ears, nose, and throat.
3. Conduct a dermal exam and check mucous membranes for a primary inoculation site; this may be a clue to a diagnosis of cat-scratch disease (CSD). A red lymphangitic streak may indicate a localized infection.

▶

C. Palpate:
1. Palpate the abdomen. Splenomegaly frequently presents with mononucleosis, lymphomas, and sarcoidosis.
2. Conduct a clinical breast exam. Palpate mass to determine if it is a lymph node, if applicable.
3. Palpate all nodal areas for localized and generalized lymphadenopathy:
a. Hard, fixed nodes suggest metastasis and a biopsy should be taken promptly. Size alone is not itself diagnostic; any node larger than 3 cm suggests neoplastic disease.
4. Palpate the scalp in the elderly for the tender arteries of cranial arteritis.
5. Palpate the neck for thyroid gland tenderness.
D. Percuss:
1. Percuss sinuses for tenderness and transilluminate for evidence of sinusitis.
E. Auscultate:
1. Auscultate the heart and lungs.
F. Musculoskeletal system exam:
1. Assess for bone or joint swelling, tenderness, and increased warmth.
2. Examine lower extremities for evidence of phlebitis: asymmetric swelling, calf tenderness, and palpable cord.
G. Genitorectal exam:
1. Conduct careful external evaluation for herpetic lesions, masses, discharge, erythema, chancroid, scabies, and pediculosis.
2. "Milk" urethra for discharge.
3. Note any folliculitis if the client regularly shaves genital area.
4. Female pelvic exam: Look for cervical discharge, cervical motion tenderness, adnexal tenderness, and mass or "heat" in the pelvis.
5. Males: Examine the prostate and testicles for tenderness and masses, as well as the penis for discharge and rash.
6. Examine the rectum for discharge, tenderness, masses, and fistulas.

DIAGNOSTIC TESTS

A. Initial:
1. Complete blood count (CBC) with differential.
2. Peripheral blood smear: the most useful laboratory test; may be helpful in the diagnosis of chronic leukemia, infectious mononucleosis, Epstein–Barr virus (EBV), and other viral illnesses.
3. Comprehensive metabolic profile.
4. Liver function tests, especially alkaline phosphatase.
5. Angiotensin-converting enzyme (ACE) test.
6. Antinuclear antibodies (ANA) and rheumatoid factor.
7. Fungal series.
8. Interferon-gamma release assays (QuantiFERON) assessment for TB.
9. Rapid plasma reagin (RPR) and microhemagglutination assay for antibody to *Treponema pallidum* (MHA-TP) to rule out syphilis.
10. Screen for EBV.
11. Enzyme-linked immunosorbent assay (ELISA) and western blot to rule out HIV.
12. Uric acid: Elevations may reflect lymphoma or other hematologic malignancies.
13. Cultures: blood, throat, urethral, and/or cervical cultures/smears.
14. Chest x-ray.
15. Mammogram with ultrasonography of suspicious breast area, if indicated.
B. After consultation:
1. Abdominal ultrasonography or CT scan, if indicated.
2. Biopsy or fine-needle aspiration: Fine-needle aspiration is used to obtain a cytologic diagnosis of a suspected cancer. False-negative results may occasionally occur.
3. Lymph node biopsy: Lymph node biopsy is the definitive test to confirm or rule out a suspected neoplastic process.
4. Mediastinoscopy.
5. Tuberculin skin testing (purified protein derivative) and ACE determination can facilitate assessment:
a. If the client tests negative for ACE and PPD and is Caucasian, then bronchoscopy and mediastinoscopy may be necessary to rule out lymphoma. If the client tests positive for ACE but negative for PPD, then the probability is very high that sarcoidosis is the cause.
b. If the client tests negative for ACE but positive for PPD, then primary TB is likely.

DIFFERENTIAL DIAGNOSES

A. There are four general categories for lymphadenopathy:
1. Infections:
a. Mononucleosis.
b. AIDS or AIDS-related complex (ARC): Generalized adenopathy in an asymptomatic HIV-infected client indicates a high risk of progression to AIDS. The lymphadenopathy represents follicular hyperplasia in response to HIV infection.
c. Toxoplasmosis.
d. Secondary syphilis.
e. Cytomegalovirus.
f. EBV.
g. *Bartonella* (CSD).
2. Hypersensitivity reactions:
a. Serum sickness.
b. Phenytoin and other drugs.
c. Vasculitis: lupus and rheumatoid arthritis (RA).
3. Metabolic diseases:
a. Hyperthyroidism.
b. Lipidoses.
4. Neoplasia:
a. Leukemia.
b. Hodgkin disease, advanced stages.
c. Non-Hodgkin lymphoma.
d. Gastric malignancies.
e. Metastatic breast cancer.
B. Causes can be isolated by site of the enlarged nodes (Table 20.1).

PLAN

A. General interventions:
1. Pay careful attention to nodal history and characteristics on physical examination.
2. Make a careful assessment to establish that the palpable mass is a lymph node. Chronicity alone is not always serious.
B. Client teaching: *See Client Teaching Guide for this chapter, "Lymphedema."*
1. As indicated by the particular disease process causing the lymphadenopathy. See the relevant guidelines.
C. Pharmaceutical therapy:
1. Dependent on the diagnosis.

TABLE 20.1 CAUSES OF LYMPHADENOPATHY BY SITE OF ENLARGED NODES

Site of Enlarged Nodes	Cause(s)
Anterior auricular	Viral conjunctivitis Trachoma
Posterior auricular	Rubella scalp infection
Axillary	Breast malignancy Breast infection Upper extremity infection
Cervical (bilateral)	Mononucleosis sarcoidosis Toxoplasmosis pharyngitis
Epitrochlear	Syphilis (bilateral) Hand infection (unilateral)
Hilar adenopathy	Sarcoidosis (unilateral or bilateral) TB Malignancies including lung cancer and lymphoma Fungal infection (histoplasmosis, coccidioidomycosis)
Lymphoma (unilateral or bilateral)	Bronchogenic carcinoma (unilateral or bilateral) TB (unilateral or bilateral)
Submandibular or cervical (unilateral)	Buccal cavity infection Pharyngitis (can be bilateral) TB Malignancies including head and neck and thyroid
Supraclavicular (left)	Intra-abdominal malignancy Renal malignancy Testicular or ovarian malignancy
Supraclavicular (right)	Pulmonary malignancy Mediastinal malignancy Esophageal malignancy
Inguinal	Infection of lower extremity Pelvic malignancy or lymphoma STIs
Any region	Cat-scratch disease Hodgkin disease Leukemia Metastatic cancer Sarcoidosis Granulomatous infections

STIs, sexually transmitted infections; TB, tuberculosis.

FOLLOW-UP

A. Follow the client closely to evaluate resolution of lymphadenopathy and disease process.

B. Follow-up depends on the diagnosis.

CONSULTATION/REFERRAL

A. Consultation with a physician may be useful if a period of observation is needed.

B. Refer the client to a specialist after initial workup, if indicated.

C. Refer the client to an oncologist or oncologic surgeon if the client is suspected of having a malignancy, to consider the need for biopsy or best approach to obtaining a tissue diagnosis. Risk factors for malignancy include duration of lymphadenopathy greater than 4 to 6 weeks, systemic signs such as fever and night sweats, and a node that has not returned to its baseline after 8 to 12 weeks.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Regional lymphadenopathy occurs often when carcinomas metastasize to lymph nodes in the elderly.

2. Incidence of head and neck lymphadenopathy is common with geriatrics and some might be related to cutaneous malignancies. It is important to thoroughly examine the cervical lymph nodes: submental and submandibular (oral/dental infections), preauricular (viral, lymphoma, CSD), occipital (HIV), anterior (upper respiratory infection), posterior (HIV), scalp infections, seborrheic dermatitis.

3. Chronic cervical lymphadenopathy most often indicates lymphoma, metastatic cancer, or TB with elderly population.

4. A periumbilical node may suggest advanced gastric or pelvic malignancy and needs further workup.

5. For malignant cases, radiation therapy and/or chemotherapy are considered first-line treatment for geriatrics. However, surgery is first-line treatment for cutaneous malignancies.

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MALNUTRITION

DEFINITION

A. Malnutrition refers to deficiencies, excesses, or imbalances in a person's intake of energy and/or nutrients. The term *malnutrition* covers two broad groups of conditions (WHO, 2020).

1. The term *undernutrition* includes stunting (low height for age), wasting (low weight for height), underweight (low weight for age), and micronutrient deficiencies or insufficiencies (lack of important vitamins and minerals). Identification and treatment of adult undernutrition are a major concern in acute, chronic, and transition care settings.

B. A malnourished state is defined as any of the following:

1. Body mass index (BMI) less than 18.5 kg/m^2.

2. Unintentional weight loss greater than 10% within the past 3 to 6 months.

3. BMI less than 20 kg/m^2 and unintentional weight loss greater than 5% within the past 3 to 6 months.

C. Obesity:

1. BMI 25 to 30 kg/m^2 is considered overweight.

2. BMI greater than 30 kg/m^2 is considered obese.

INCIDENCE

A. Depending on the client population and criteria used to identify malnutrition, estimates of adult malnutrition range from 15% to 60%. Of geriatrics living in the community, 3% to 30% suffer from malnutrition. The prevalence rises to as high as 60% when an elderly person lives in a long-term care facility. For elderly clients in acute care, malnutrition rates reach 12% to 50%. ▶

B. Sarcopenia prevalence ranges from 2% to 27% in the community-dwelling elderly and 14% to 33% for persons who reside in a long-term care facility. Chronic illnesses and disabilities increase the risk.

PATHOGENESIS

A. Primary protein-energy malnutrition arises from insufficient dietary intake, or intake of protein of poor enough quality to make availability of essential amino acids a limiting factor in the support of metabolism.

B. Secondary protein-energy malnutrition arises when illness or other factors impair the uptake or utilization of nutrients, increase protein or energy requirements, or increase metabolic losses beyond nutrient availability.

PREDISPOSING FACTORS

A. Individuals 65 years of age or older.
B. Individuals living in a nursing or care home.
C. Individuals admitted to the hospital.
D. Individuals who abuse drugs or alcohol.
E. Individuals with malabsorption syndromes.
F. Poverty.
G. Polypharmacy.
H. Comorbidities:
  1. Chronic obstructive pulmonary disease (COPD).
  2. Coronary artery heart disease.
  3. Cancer.
  4. Depression.
  5. Neurologic disorders: cerebrovascular accident Parkinson disease, dementia.
  6. Arthritis.
  7. Chronic or end-stage renal disease.
  8. Diabetes mellitus.

COMMON COMPLAINTS

A. Losing weight without trying.
B. Decreased appetite.
C. Difficulty eating.
D. Fatigue.
E. Difficulty concentrating.
F. Feeling cold.
G. Longer healing time for wounds.
H. Depressed mood.
I. Activity intolerance.
J. Difficulty with food procurement and preparation.

OTHER SIGNS AND SYMPTOMS

A. Decreased libido.
B. Dry skin; dry, sparse, brittle hair.
C. Breathing difficulties.
D. Ankle swelling.

SUBJECTIVE DATA

A. Weight loss or gain in the past 6 months. Was weight loss intentional? (Unintentional loss of more than 10% of normal body weight in the past 3–6 months is of concern.)
B. Changes in dietary intake.
C. Gastrointestinal symptoms including nausea, diarrhea, constipation, flatulence, pain, dysphagia, xerostomia, and appetite changes.
D. Dental problems including dentures.
E. Changes in vision, smell, or taste.
F. Medications taken, including prescribed and over-the-counter (OTC):
  1. Medications that can lead to anorexia in older clients include the following:
    a. Amiodarone.
    b. Furosemide.
    c. Digoxin.
    d. Spironolactone.
    e. Levodopa.
    f. Fluoxetine.
    g. H2 antagonists.
    h. proton pump inhibitors (PPIs).
    i. Nonsteroidal anti-inflammatory drugs (NSAIDs).
G. Changes to skin, hair, conjunctiva, or buccal mucosa; delayed wound healing.
H. Place of residence:
  1. Exposure to food contamination.
  2. Toxins.
  3. Readily access to groceries.
I. Fatigue, shortness of breath, muscle cramps.
J. Comorbidities (see list under the "Predisposing Factors").
K. Functional capacity:
  1. Does the client have trouble carrying out activities of daily living (ADLs)?
  2. Can they shop and prepare food?
L. Disease and its relation to nutrition requirements.
M. Females of childbearing age: menstrual cycle irregularities.
N. History of eating disorders.
O. Cultural and religious practices.
P. Dietary history:
  1. Daily dietary intake and preferences.
  2. Dietary history.
  3. Alcohol consumption.
  4. Food allergies and intolerances.
  5. Need for assistance purchasing and preparing food.
Q. Financial resources.

PHYSICAL EXAMINATION

A. Measurement of weight and height, and proportions; compare with standards of age and sex.
B. Calculate BMI.
C. Body measurements:
  1. Measurement of waist circumference.
  2. Measure muscle mass by measuring calf and midarm circumferences.
  3. Evaluate muscle strength using a dynamometer to determine handgrip strength.
D. Inspect:
  1. Body fat distribution.
  2. Inspect oral cavity including teeth, and if client has dentures determine their fit. Note changes to the integrity of the tongue, gingiva, lips, and oral mucosa can indicate a vitamin deficiency; oral candidiasis.
  3. Inspect skin and hair, looking for dryness, fragility, flakiness, or discoloration of skin.
  4. Look for hair that is brittle, thin, and easy to pull out, as well as premature whitening of hair.
  5. Inspect nails for spooning.
E. Palpate:
  1. Ankle and sacral edema, which may indicate protein imbalances.
  2. Thyroid gland for enlargement of nodules.
F. Neurologic exam:
  1. Central and peripheral visual acuity.
  2. Assess cognitive function using a validated tool. ▶

3. Examine vibratory sense with tuning fork if B_{12} deficiency suspected.
4. Screen for visual acuity and hearing annually.

DIAGNOSTIC TESTS

A. Complete metabolic panel, complete blood count (CBC), thyroid-stimulating hormone (TSH).
B. Vitamins A, B_2, B_6, B_{12}, niacin, C, D, E, K, thiamine.
C. Minerals including zinc, iron (iron level, total iron-binding capacity [TIBC], ferritin), iodine.
D. C-reactive protein (CRP) if inflammatory process suspected.
E. Dual-energy x-ray absorptiometry (DEXA) scan.
F. Swallow evaluation if indicated.
G. Celiac serology if indicated.
H. Stool for ova and parasites if indicated.
I. Screen with valid nutrition screening tool, such as Mini Nutritional Assessment (MNA) or Malnutrition Universal Screening Tool (MUST), the Nutritional Form for the Elderly (NUFFE) for the rehabilitation setting, and the Short Nutritional Assessment Questionnaire-Residential Care ($SNAQ^{RC}$) for residential care.
J. Body composition analysis using bioelectrical impedance analysis.

Recent analysis indicates albumin and prealbumin are no longer considered reliable markers of nutrition.

DIFFERENTIAL DIAGNOSES

A. Starvation-related malnutrition:
1. Anorexia nervosa and bulimia.
2. Chronic starvation.

B. Chronic disease-related malnutrition:
1. End-stage renal disease.
2. Chronic pancreatitis.
3. Cancers.
4. Cardiovascular disease (CVD).
5. Sarcopenia
6. Rheumatoid arthritis.
7. Obesity.
8. Malabsorption syndromes including celiac disease.
9. HIV/AIDS.
10. Chronic respiratory disease including COPD and cystic fibrosis.

C. Acute disease or injury-related malnutrition:
1. Major infections.
2. Burns.
3. Acute pancreatitis.

PLAN

A. General interventions:
1. Address underlying etiology.
2. Interdisciplinary approach to problem, including primary care provider (PCP), dietitian, social worker, occupational and/or physical therapist, and nursing.
3. Social support and referrals to food pantries and food delivery services (e.g., Meals on Wheels) and coordination of nutritional services.
4. Physical therapy and/or occupational therapy to evaluate and address functional status deficiencies:
   a. Progressive resistance training two to three times a week helps to increase muscle strength and improve function especially with sarcopenia.
5. Nursing care for dietary and weight monitoring.

B. Client teaching:
1. Dietary education, including food preparation:
   a. Food first approach:
      i. Three small meals with snacks in between every day (add foods such as oil, butter, cheese, cream, sauces to meals, and snacks to boost energy intake).
      ii. Choose nourishing fluids such as milky drinks, soups, and fruit juice.
      iii. Food fortification: Add foods dense in protein and calories such as powdered milk without increasing volume.
      iv. Reassess weight in 4 weeks.
      v. Try oral nutritional supplementation for a month. If no change in nutritional parameters, then discontinue. Usually accounts for 300 to 400 kcal/d.
2. Vitamin and/or mineral supplementation as indicated per lab results.

C. Pharmaceutical therapy:
1. Dronabinol (Marinol).
2. Megestrol (Megace).
3. Testosterone.
4. Herbal products:
   a. Dandelion.
   b. Chamomile.

FOLLOW-UP

A. Check weight in 4 weeks. If no improvement, refer for enteral or parenteral nutrition support.

CONSULTATION/REFERRAL

A. Refer to psych/mental health services for eating disorders or clinical depression.
B. Refer to gastroenterology for endoscopy.
C. Refer to gerontologist if failure to improve.
D. Refer to registered dietitian, physical therapy, or occupational therapy.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. After any acute event or illness, rescreen client for malnutrition.
2. Assess for malnutrition annually in clients who reside in a community-dwelling.
3. Assess every 3 months in clients who reside in an institutional facility.
4. Routine nutritional assessment with individualized nutrition support for clients in the community.
5. Ensure client is taking 0.8 g/kg of protein daily; consider increasing to 1.2 kg/d if person has sarcopenia.
6. May need a surrogate person to verify information.

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MEGALOBLASTIC ANEMIA

DEFINITION

A. Megaloblastic anemia is characterized by abnormally large nucleated red blood cell (RBC) precursors. Ninety-five percent of cases are due to vitamin B_{12} or folic acid, which occurs when there is a failure of DNA synthesis but RNA synthesis remains unimpaired. The inability to synthesize DNA leads to abnormally large erythroid precursors and RBCs.

B. Pernicious anemia is a type of vitamin B_{12} deficiency anemia. It is a megaloblastic, macrocytic, normochromic anemia caused by a deficiency of intrinsic factor in the gastric juices produced by the stomach, which results in malabsorption of vitamin B_{12} necessary for DNA synthesis and maturation of RBCs. There is production of abnormally large and oval red cells with a mean corpuscular volume (MCV) in excess of 100 fL (femtoliters). The anemia can be severe, with hematocrit (Hct) as low as 10% to 15%.

INCIDENCE

A. The prevalence of vitamin B_{12} deficiency anemia varies with age: less than 3% in adults between the ages of 20 to 39; 4% in those aged 40 to 59; and 12% in adults over 70 years old. Twenty percent of adults age 60 or greater have suboptimal vitamin B_{12} levels, with the risk increasing with age. Of the sick or institutionalized elderly, 30% to 40% are vitamin B_{12}-deficient. It is identified in 30% of diabetic clients taking metformin.

B. Pernicious anemia is common in people of northern European descent. Both sexes are equally affected. It is most prevalent in Scandinavian and English-speaking populations. It usually occurs in the fifth and sixth decades of life; it is rarely seen in persons younger than 35 years, but it can occur in individuals in their 20s. There is an increased incidence in those with other immunologic diseases. The average age of diagnosis is 60. Rarely is pernicious anemia passed down through families. Pernicious anemia accounts for 20% to 50% of vitamin B_{12} deficiency anemia.

C. Less than 1% of the U.S. population are diagnosed with folic acid deficiency-related anemia. Approximately 3,000 pregnancies are affected by neural tube defects annually in the United States. Estimates show 11% of chronic alcoholics are folic acid-deficient.

PATHOGENESIS

A. Megaloblastic anemia is caused by deficiency or impairment of utilization of vitamin B_{12} or folate. Nonmegaloblastic anemia may be a result of liver dysfunction, alcoholism, myelodysplastic syndrome (MDS), or hypothyroidism.

B. Pernicious anemia is possibly due to an autoimmune reaction involving the gastric parietal cell that results in nonproduction of intrinsic factor and atrophy of gastric mucosa. Vitamin B_{12} deficiency can result from inadequate intake, impaired absorption, increased requirements as in pregnancy, or faulty utilization. Poor intake is rare, occurring most often in strict vegetarians. This vitamin can be obtained from eating foods such as meat, poultry, shellfish, eggs, and dairy products.

PREDISPOSING FACTORS

A. Vitamin B_{12} deficiency anemia:

1. People of northern European descent identified as a risk factor for pernicious anemia.

2. Persons over 50 years of age and older.

3. Vegetarian or vegan diet.

4. History of bariatric surgery.

5. Fish tapeworms.

6. Ileostomy, ileal resection, or abnormalities.

7. Atrophic gastritis and achlorhydria mostly in the elderly.

8. Exposure to nitrous oxide anesthesia.

9. Certain medications including acyclovir, zidovudine, fluorouracil, metformin, proton pump inhibitors (PPIs) oral estrogen contraceptives.

10. Congenital enzyme deficiencies.

11. Autoimmune disorder causing a lack of intrinsic factor leads to pernicious anemia. Medical conditions include Addison disease, Graves disease, type 1 diabetes mellitus, or myasthenia gravis.

B. Folate deficiency anemia:

1. Dietary deficiency especially during pregnancy and lactation.

2. Improper food preparation; especially common in the elderly.

3. Alcoholism.

4. Renal dialysis.

5. Psoriasis.

6. Certain medications including phenytoin, methotrexate, and valproic acid.

C. Combination of vitamin B_{12} and folate deficiency anemia:

1. Crohn disease.

2. Hypothyroidism.

COMMON COMPLAINTS

A. Weakness and dizziness.

B. Tongue is sore, red, and shiny.

C. Numbness, burning, and tingling sensation in the arms or legs.

D. Feel heart "jumping out of skin."

E. Edema of lower extremities.

F. Anorexia.

G. Diarrhea.

H. Low-grade fevers.

I. Complaint of looking pale.

OTHER SIGNS AND SYMPTOMS

A. Mild anemia:

1. Dyspnea on exertion.

2. Pallor.

3. Weakness.

4. Fatigue.

B. Severe anemia:

1. Pallor.

2. Fatigue.

3. Tachycardia.

4. Exercise intolerance.

5. Angina.

6. Glossitis.

7. Mucositis.

8. Peripheral paresthesia.

9. Palpitations.

10. Abdominal tenderness, organomegaly.

11. Advanced stages: dementia and depression; spinal cord degeneration.

C. Classic presentation involves sore t ongue and numbness and tingling in the extremities, hands, or feet.

SUBJECTIVE DATA

A. Inquire about onset, duration, and course of presenting symptoms. ▶

B. Ask the client to describe usual bowel habits. Has there been any blood in stools?
C. If gastrointestinal (GI) complaints are present; inquire about presence of red, burning tongue, abdominal complaints, diarrhea or constipation, abdominal pain, pale-colored stool, or stool that floats.
D. If neurologic complaints are present, inquire about the presence of pins-and-needles paresthesia and weakness, unsteadiness due to proprioceptive difficulties, lethargy, and fatigue.
E. Evaluation of diet: Is the client a vegetarian or vegan or anorexic? Does the client follow a fad diet?
F. Ask about alcohol consumption: How much? How long? Any history of pancreatitis?
G. Obtain medication history: over-the-counter (OTC) and prescription drugs.
H. Obtain medical history, specifically if the client has a history of gastrectomy, gastric bypass/banding, resection of ileum, or other GI disorders.
I. Review usual weight and recent loss.
J. Inquire about low-grade fevers.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure, weight. One-third of clients experience low-grade fever.
B. Inspect:
1. Observe general, overall appearance; observe walking.
2. Conduct oral exam for characteristic red, shiny tongue.
3. Conduct dermal and eye exams for color: Affected clients are slightly icteric.
4. Evaluate the look of the person related to age: Affected clients show premature aging or graying.
C. Palpate:
1. Palpate the abdomen for masses. Splenic tip palpable in 20% of clients.
2. Evaluate pedal edema.
D. Percuss:
1. Percuss the abdomen.
E. Auscultate:
1. Auscultate heart sounds and lungs. Systolic murmurs may be present.
2. Auscultate the abdomen for bowel sounds.
F. Neurologic exam:
1. Assess deep tendon reflexes (DTRs), proprioception, plantar reflex, Romberg test, vibratory sense, and mental status:
2. Look for paresthesia involving the hands and feet, gait disturbances, or memory loss (mild forgetfulness to dementia or altered thought processes). Poor finger–nose coordination may be seen; positive Romberg and Babinski signs may be present.
3. Severe B_{12} deficiency can result in ataxia, impaired proprioception, and vibratory sensation.

DIAGNOSTIC TESTS

A. Complete blood count (CBC) with differential and peripheral smear: Macroovalocytes and hypersegmented neutrophils may be present on peripheral blood smear. (They are absent in the setting of concurrent iron deficiency.)
B. Reticulocyte count.
C. Serum vitamin B_{12} level: less than 100 to 200 pg/mL; suboptimal vitamin B_{12} levels 200 to 350 pg/mL.
D. Serum folic acid levels, serum iron, serum ferritin, and total iron-binding capacity (TIBC).
E. Serum anti-intrinsic factor antibody (anti-IFAB) to rule out pernicious anemia.
F. Lactate dehydrogenase (LDH).
G. Urinalysis.
H. Stool for occult blood.
I. Indirect bilirubin.
J. GI radiographic studies.
K. Gastric analysis: Achlorhydria is found on stimulation testing.
L. Bone marrow aspiration.
M. A female with low B_{12} levels may have a false-positive Pap smear due to vitamin B_{12} effects on the epithelial cells.
N. Serum vitamin B_{12} levels may be artificially elevated in clients with alcoholism, liver disease, or cancer due to decreased hepatic clearance of transport proteins with resultant higher circulating levels of vitamin B_{12}.
O. Confirmatory test includes methylmalonic acid and homocysteine levels.
1. Methylmalonic acid and homocysteine levels are elevated with vitamin B_{12} deficiency.
2. Methylmalonic acid level is normal and homocysteine level elevated with folate deficiency.
3. Holotranscobalamin if B_{12} values are indeterminate or borderline despite clinical symptoms.

DIFFERENTIAL DIAGNOSES

A. Differential diagnosis of anemia by red cell morphology can be undertaken (MCV, mean corpuscular hemoglobin concentration [MCHC]). Common causes of each type of anemia are as follows:
1. Normochromic, normocytic: normal MCV = 80 to 100; MCHC = 32% to 36%:
a. Aplastic anemia.
b. Chronic disease including cancer, heart failure, and HIV.
c. Early iron deficiency.
d. Hemolysis.
e. Hemorrhage.
2. Microcytic: MCV = 50 to 82; MCHC = 24 to 32:
a. Chronic disease.
b. Iron deficiency.
c. Thalassemia.
3. Macrocytic: MCV greater than 100; MCHC greater than 36:
a. Antimetabolites.
b. Folic acid deficiencies.
c. Vitamin B_{12} deficiencies including pernicious anemia.
d. Chronic alcoholism.
B. Note: Iron-deficiency anemia can mask megaloblastic anemia.

PLAN

A. General interventions:
1. Most common method of determining vitamin B_{12} deficiency is by serum vitamin B_{12} assay.
2. Most common method of demonstrating folate deficiency is by measurement of serum folic acid levels.
3. Red cell indices and peripheral smear should be done to determine classification of anemia to facilitate workup.
4. Red cell distribution width (RDW) determination can assist in detecting red cell heterogeneity previously available only by exam of the peripheral smear. The RDW determination overcomes the problems of detecting coexisting microcytic and macrocytic anemias.
B. Client teaching: *See Client Teaching Guide for this chapter, "Vitamin B_{12} Including Pernicious Anemia."*

1. Neurologic symptoms usually improve with treatment; however, some neurologic deficits may not be reversible.

C. Pharmaceutical therapy:

1. Bariatric clients should receive vitamin B_{12} supplement, 1 mg orally per day for life as prevention. If anemic, then follow protocol for pernicious anemia.

2. Pernicious anemia or clients with symptomatic anemia including neurologic findings require daily injections of 1,000 mcg of vitamin B_{12} for 1 week, followed by 1,000 mcg every week for 4 week. Clients with pernicious anemia will require intramuscular (IM) injections of 1,000 mcg monthly for life.

3. If anemic client is able to ingest and absorb vitamin B_{12}, then may prescribe 1,000 to 2,000 mcg/d orally. Continue until RBC indices and vitamin B_{12} are in normal range. If anemic client has neurologic findings, then consider parenteral administration.

4. Taking 1,200 mg of calcium daily can help prevent metformin-related vitamin B_{12} deficiency.

5. Consider B_{12} supplementation in clients over 50 years of age if not meeting their recommended dietary allowance of B_{12} through fortified food:

a. Recommended daily allowance for vitamin B_{12}:
   i. Adults: 2.4 mcg/d.
   ii. Pregnant: 2.6 mcg/d.
   iii. Breastfeeding: 2.8 mcg/d.

b. Concomitant iron supplementation during the first month of therapy. Rapid blood cell regeneration increases iron requirements and can lead to iron deficiency.

FOLLOW-UP

A. The client must be seen in 2 weeks to determine response to treatment: increased reticulocyte count and increased Hct; and diminution in neurologic signs and symptoms. Neurologic symptoms should improve within 6 weeks to 3 months.

B. Evaluate the client monthly when giving vitamin B_{12} injections.

C. Clients with pernicious anemia require endoscopy every 5 years to rule out gastric carcinoma. Those with pernicious anemia may have gastric polyps and are more likely to develop gastric cancer and gastric carcinoid tumors.

D. Check the client every 6 months for Hct and check their stool for occult blood.

E. Hct value rises 4% to 5% per week in uncomplicated cases.

CONSULTATION/REFERRAL

A. Refer the client to a dietitian.

B. Rapid reticulocytosis should be seen following treatment; it peaks in 7 to 10 days.

C. Consult a physician if no change is seen.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Lactovegetarians and ovolactovegetarians do well in pregnancy.

2. Strict vegetarians or vegans who eat neither eggs nor milk products should take vitamin B_{12} supplements during pregnancy and lactation.

B. Adults:

1. Disorder is rarely seen in clients younger than 35 years of age.

2. Screen clients who have taken PPIs or H2 blockers for more than 12 months.

3. Screen clients who have taken metformin for more than 4 months.

C. Geriatrics:

1. B_{12} anemia is most commonly seen in the geriatric population. Risk of vitamin B_{12} anemia increases with age.

2. Follow up elderly clients with assessment of cardiovascular symptoms 48 hours after initiating therapy.

3. Rapid blood cell regeneration increases iron requirements and can lead to iron deficiency.

4. Studies have shown that oral high dose of cyanocobalamin for the geriatric population is safe, pain-free, and costs less than parenteral injections. A daily supplement of cobalamin of 1,000 mg (or more) or sublingual daily dose 500 μ to 1 mg of cobalamin is just as effective in correcting megaloblastic anemia as injections.

5. Common causes of megaloblastic anemia in geriatric clients are malnutrition, high alcohol intake, and overuse of PPIs or recent hospitalization requiring intravenous (IV) PPI. These causes compounded with age-related parietal cell decline lead to significant B_{12} malabsorption.

6. B_{12} deficiency often goes unrecognized in the elderly population because they rarely present with symptoms. Annual screening is recommended (or more often if clients are at risk for B_{12} deficiency). Lab tests are not always forthright; however, it is recommended that a low serum vitamin B_{12} level and an elevated serum homocysteine are most sensitive in making a definitive diagnosis for this population.

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MYALGIC ENCEPHALOMYELITIS/CHRONIC FATIGUE SYNDROME

DEFINITION

A. Fatigue is one of the most common symptoms confronting the practitioner in an office practice. A client with chronic fatigue is characterized as having fatigue with multiple associated symptoms for longer than 6 months in which these symptoms have a profound impact on daily activities.

INCIDENCE

A. Fatigue accounts for 1% to 3% of visits to generalists as an isolated symptom or diagnosis. Psychiatric disorders are involved in less than 50% of cases. Myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS), formerly known as chronic fatigue, has a reported frequency in excess of 20%. At least 25% of individuals with ME/CFS are bedbound or homebound and up to 75% are unable to work or attend school. Fatigue is seen predominantly in females, four times more than in males, and the highest prevalence is among people aged 40 to 60; however, it can affect people of all ages, including teens and children.

PATHOGENESIS

A. The Institute of Medicine (IOM) indicates chronic fatigue is caused by changes in the immune system's response to stress/infection. Possible systemic dysfunctions include chronic production of cytokines, low-functioning natural killer cells, differences in markers of T-cell activation, and disruptions within hypothalamic–pituitary–adrenal axis. The long-term effects of CFS can lead to a serious illness affecting multiple body systems, which the IOM reports as ME/CFS. Clients suffering ▶

from ME/CFS experience three primary symptoms: a sizable drop in activity level for ≥6 months, postexertional malaise that includes decrease in mental acuity, and relentless sleep difficulties. An overview of the Centers for Disease Control and Prevention (CDC) and IOM's management education can be found at www.cdc.gov/me-cfs/index.html.

PREDISPOSING FACTORS

A. Hyperthyroidism.
B. Hypothyroidism.
C. Cardiac disease: congestive heart failure.
D. Neurally mediated hypotension.
E. Infections: endocarditis, hepatitis, Epstein–Barr virus (EBV), and *Coxiella burnetii.*
F. Respiratory disorders: chronic obstructive pulmonary disease (COPD) and sleep apnea.
G. Anemia.
H. Arthritis and related disorders.
I. Cancer.
J. Alcoholism.
K. Side effects from drugs such as sedatives and beta-blockers.
L. Psychological conditions such as insomnia, depression, anxiety, and somatization disorder.
M. Female.
N. Race/ethnicity: Whites over other races and ethnicities.

COMMON COMPLAINTS

A. Sudden onset of profound fatigue with other symptoms including flulike symptoms. Fatigue is greater than 6 months and interferes with participation in family, work, or leisure activities. Does not improve with rest.
B. Unrefreshing sleep.
C. Extreme exhaustion lasting more than 24 hours after physical or mental exercises.
D. Loss of memory and concentration.
E. Joint pain that moves around without swelling or redness.
F. Muscle fatigue.
G. Swollen lymph nodes in neck/armpits.

OTHER SIGNS AND SYMPTOMS

A. Weakness.
B. Postexertional malaise with physical or mental activities.
C. Poor physical conditioning.
D. Undernutrition and poor appetite.
E. Stress.
F. Obesity.
G. Emotional problems: depression, anxiety, and somatization disorder.
H. Myalgia and arthralgia.
I. Problems with thinking and memory.
J. Orthostatic intolerance including light-headedness, dizziness, weakness, or visual changes.

SUBJECTIVE DATA

A. Review history for onset, duration, and course description of the fatigue.
B. Perform thorough mental status exam. Ask the client about significant losses, low self-esteem, and occurrence of crying spells and suicidal thoughts. Utilize a depression screening tool. There is high prevalence of depression and suicide in this client population.
C. Ask the client about history of any abuse of hypnotic drugs, alcohol, or tranquilizers.
D. Review medications, both over-the-counter (OTC) and prescription drugs.
E. Review the client's medical history for cardiac, thyroid, and other medical conditions.
F. Review sleep and insomnia history.
G. Review history for family illness, new baby, or postpartum.
H. Establish last menses to rule out pregnancy.
I. Review exercise patterns.
J. Review diet with 24-hour recall.
K. Elicit history of fever, night sweats, weight loss, and enlarged lymph node(s).
L. Inquire about recent major life changes, such as moving or a change in job.
M. Establish usual weight, and review recent weight gain or loss, over what period.
N. Review any recent infections.
O. Review high-risk sexual practices and intravenous (IV) drug use to rule out HIV exposure.
P. Review recent history for transfusion of blood products to rule out hepatitis or HIV exposure.
Q. Obtain history of the client's daily living and working habits.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure, and weight; check for postural hypotension.
B. Inspect:
  1. Observe general overall appearance.
  2. Skin: Conduct dermal exam for changes in pigmentation, purpura, dryness, rashes, jaundice, pallor, splinter hemorrhages, or petechiae.
  3. Eyes: Conduct a funduscopic exam to rule out Roth spots and tuberculoma.
  4. Check sclerae for icterus.
  5. Throat: Inspect pharynx for petechiae at the junction of the hard and soft palate to rule out mononucleosis.
  6. Extremities: Inspect joints for inflammation.
C. Palpate:
  1. Palpate the neck to rule out goiter.
  2. Palpate all lymph nodes (neck, axilla, and groin) for size, degree of tenderness, and distribution.
  3. Complete clinical breast exam for masses.
  4. Palpate the abdomen for organomegaly, masses, and ascites.
  5. Palpate the joints for tenderness.
  6. Assess the genitalia for masses and tenderness to rule out infection.
D. Percuss:
  1. Percuss the abdomen for organomegaly, masses, ascites, and hepatic tenderness.
E. Auscultate:
  1. Auscultate the heart and lungs.
F. Neurologic exam:
  1. Complete mental status assessment.
G. Rectal exam:
  1. Assess for masses, prostatic pathology, and occult blood.

DIAGNOSTIC TESTS

A. Complete blood count (CBC) with differential and peripheral smear.
B. Erythrocyte sedimentation rate (ESR).
C. Calcium, phosphate, albumin, sodium, potassium, chloride, and bicarbonate.
D. Fasting glucose.
E. Renal function: blood urea nitrogen (BUN), creatinine, and glomerular filtration rate (GFR). ▶

F. Liver function: bilirubin, alkaline phosphatase (ALP), gamma glutamyl transaminase (GGT), alanine transaminase (ALT), and aspartate transaminase (AST).
G. Thyroid panel to rule out hyperthyroidism or hypothyroidism.
H. Iron studies: serum iron, iron-binding capacity, and ferritin.
I. Vitamin B_{12}, serum folate, and 25-hydroxy-cholecalciferol (vitamin D).
J. Sleep study.
K. Creatine kinase (CK).
L. Urinalysis.
M. Additional tests as clinically indicated: rheumatoid factor, antinuclear antibodies (ANA), EBV, HIV serum test, and creatine phosphokinase (CPK) if ther is muscle weakness.

DIFFERENTIAL DIAGNOSES

A. ME/CFS:
1. The CDC and IOM include the following three criteria for ME/CFS syndrome diagnosis:
a. A substantial reduction or impairment in the ability to engage in preillness levels of occupational, educational, social, or personal activities that persists for more than 6 months and is accompanied by fatigue, which is often profound, is of new or definite onset (not lifelong), is not the result of ongoing excessive exertion, and is not substantially alleviated by rest.
b. Postexertional malaise.
c. Unrefreshing sleep.
2. At least one of the two following manifestations is also required for the diagnosis:
a. Cognitive impairment.
b. Orthostatic intolerance.
B. Hypercalcemia.
C. Early diabetes mellitus.
D. Hypothyroid or hyperthyroidism.
E. Cardiac disease.
F. Anemia.
G. Liver disease.
H. Connective tissue diseases.
I. Infectious diseases including Lyme disease, hepatitis B/C, EBV, coccidioidomycosis, syphilis, and parvovirus 19.
J. Disturbed sleep or obstructive sleep apnea.
K. Occult neoplasm.
L. Infection: history of fever, sweats, weight loss, and diffuse adenopathy. These symptoms also suggest HIV, especially with high-risk behaviors (see "Human Immunodeficiency Virus" section in this chapter).
M. Mononucleosis.
N. Psychiatric disorder.
O. Vitamin deficiency (vitamin D and B_{12}).
P. Neurologic disorders including multiple sclerosis, Parkinson disease, myasthenia gravis, spinal stenosis, or craniocervical instability.
Q. Gastrointestinal (GI) disorders including celiac disease, food allergies or intolerance, and inflammatory bowel diseases.
R. Environmental exposures including lead, mercury, mold, and Gulf War illness.

PLAN

A. General interventions:
1. Treatment goal: reduce symptoms and increase quality of life.
2. Determine client's view of their illness before proceeding with client education.
3. Manage underlying disease (refer to specific chapters).
4. Treat the client with nutritional or vitamin supplementation, as indicated.
5. Provide activity management guidance and dietary recommendations.
6. Assist with obtaining accommodations for school and work or when applying for disability.
7. Strong client–provider alliance is essential.
8. Evaluate the client for the possibility of confusing focal neuromuscular disease with generalized lassitude.
9. Consider a sleep study to determine if obstructive sleep apnea is a contributor to fatigue.
B. Client teaching:
1. Discuss and review the evidence for the diagnosis and offer a careful explanation of symptoms. Provide encouragement and review symptom management plan.
2. Review the diagnostic criteria for depression (see Chapter 25, "Psychiatric Guidelines"), and describe the neurochemical mechanisms by which depression relates to fatigue. Refer for individual counseling or group therapy.
3. Review the idiopathic nature of ME/CFS and its nonprogressive nature. The clinical course includes gradual improvement with the hope of full recovery (10%–15% of adults fully recover). Illness severity varies from client to client. Over time, clients may improve to the point that the diagnosis is removed. Relapses are common.
4. Encourage mild exercise not to exceed client's energy level. Refer to physical therapy for exercise plan development. Pace activities.
5. Provide nutritional education.
6. Discuss and promote sleep hygiene.
C. Pharmaceutical therapy:
1. For orthostatic intolerance:
a. Increase dietary sodium.
b. Antihypotensive agent: fludrocortisone 0.1 mg daily.
2. Low-dose antidepressant therapy for disordered sleep:
a. Amitriptyline HCl 25 mg at bedtime.
b. Doxepin HCl 10 to 20 mg every bedtime.
c. Short-term hypnotic: zolpidem (Ambien) 5 to 10 mg at bedtime (use cautiously with elderly).
d. Trazodone 50 mg at bedtime.
3. Nonsteroidal anti-inflammatory drugs (NSAIDs), serotonin-norepinephrine reuptake inhibitors, antiepileptics, and medical marijuana for symptomatic relief of myalgia, arthralgia, or headache.
4. Correct anemia with iron supplements, if applicable.
5. Muscle relaxant, such as cyclobenzaprine 5 to10 mg at bedtime as needed may also be used.
6. Methylphenidate or modafinil for cognitive dysfunction and fatigue.
D. Nonpharmacologic interventions:
1. Massage therapy, hot or cold packs, or warm water exercises for muscle aches and pains.
2. Tai chi or yoga to improve balance.
3. Encourage relaxation interventions such as meditation or mindfulness.
4. Recommend calendar, reminder systems, and memory book.
5. Compression stockings for orthostatic problems.
6. Pacing of physical and cognitive activity.
7. Assistive devices, such as motorized scooters, handicap parking stickers, and shower chairs.
8. Home health aides for those who are most severely ill. ▶

9. School or work accommodations, such as flexible hours and shortened days.
10. To minimize GI symptoms, eliminating certain foods including caffeine, alcohol, spicy foods, aspartame, sugar, and possibly dairy or gluten is suggested.

FOLLOW-UP

A. Follow up in 2 weeks to reevaluate status and then monthly, depending on signs and symptoms.
B. Follow closely if the client is depressed.

CONSULTATION/REFERRAL

A. Consult a physician if no improvement is seen with therapies. Emphasize the legitimacy of the client's symptoms and summarize the workup, its rationale, and its findings.
B. Refer the client to a mental health professional as indicated by depression and/or suicidality.
C. Refer to occupational therapy and/or physical therapy.

INDIVIDUAL CONSIDERATIONS

A. Adults:
1. Fatigue is most often explained by common factors such as overexertion, poor physical conditioning, inadequate quantity or quality of sleep, obesity, malnutrition, stress, obstructive sleep apnea, and emotional problems.

B. Geriatrics:
1. Prescribing medications should be tailored for the client, using low-dose medications.
2. Give precautions about falls if the client is using medications to aid in sleep.
3. Review importance of maintaining hydration and rising slowly to avoid falls related to orthostatic hypotension.
4. Use nonpharmacologic measures to help promote rest and increase energy in geriatrics suffering with chronic fatigue: Modify the environment (place iPad, phone, and access to food/drinks at bedside); adjust temperature to client preferences; modify activities of daily living (ADLs) such as sitting during shower rather than standing; and develop patterns of resting between meals and activities.
5. Studies indicate that occupational therapy and physical therapy decreased symptoms of chronic fatigue and weakness in geriatrics and increased benefits for quality of life.

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ME/CFS Initiative (formerly the Chronic Fatigue Syndrome):. http://solvecfs.org

CLIENT TEACHING GUIDE

IRON-DEFICIENCY ANEMIA

PROBLEM You have been diagnosed with iron-deficiency anemia. Iron is an important building block for red cells. Red blood cells carry oxygen to the body.

CAUSES Iron-deficiency anemia is the most common form of anemia. Your body may not be making enough iron, which means it is making fewer red blood cells that are too small.

Or you may have anemia from a loss of blood somewhere in your body.

Your body normally gets iron through your diet and by your body reusing old red blood cells. However, when these needs are not met, the body becomes anemic.

The following are the common causes of iron loss:

A. History of heavy, long, or frequent menstrual periods or abnormal postmenopausal bleeding.
B. Cancer of the esophagus, stomach, or colon.
C. Esophageal varices.
D. Use of aspirin, or arthritis medication, for a long period of time, which may cause gastrointestinal bleeding.
E. Peptic ulcer disease.
F. Celiac disease.
G. Crohn disease.
H. Gastric bypass surgery.
I. Taking too many antacids that contain calcium.
J. Strict vegetarian diet.

PREVENTION

A. Everyone's diet should include iron. Red meat, liver, and egg yolks are important sources of iron.
B. Increase your intake of iron-fortified flour, bread, and cereals.
C. Eat more foods that are higher in iron during periods of time when you are at risk for anemia, such as pregnancy and menstrual periods.
D. Elderly individuals who have a poor appetite or do not eat three to four meals a day should also increase their dietary intake of iron.

TREATMENT PLAN

A. Initial treatment starts with finding the cause of your anemia. Blood work may be performed to measure your iron levels before starting treatment. Most of the time, iron-deficiency anemia can be treated with iron supplementation.
B. Iron supplementation is needed to build up the iron stores in your body as directed by your healthcare provider. Ways to increase your iron stores include the following:
    1. Take iron supplements on an empty stomach.
    2. If you take antacids, you should take your iron supplement 2 hours before or 4 hours after your antacid to improve iron absorption.
    3. Take iron supplements with vitamin C, which improves absorption of iron.
    4. Take in the morning or earlier in the day.
C. Intravenous iron may be necessary if the iron is not well-absorbed in the gastrointestinal tract.

Activity:

A. Iron-deficiency anemia does not impair your activity unless you are experiencing extreme fatigue, weakness, shortness of breath, or dizziness. Fall precautions are essential if you are experiencing any of these symptoms.

Diet:

A. It is recommended to increase your diet with iron-rich foods. Some suggestions of iron-rich foods include the following:
    1. Meat: beef, pork, or lamb, especially liver.
    2. Poultry: chicken, turkey, and duck, especially dark meats.
    3. Fish: especially shellfish, sardines, and anchovies.

(*continued*)

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CLIENT TEACHING GUIDE

IRON-DEFICIENCY ANEMIA (*CONTINUED*)

4. Leafy green cabbage family members such as broccoli, kale, and turnip greens.
5. Legumes including lima beans, peas, pinto beans, and black-eyed peas.
6. Peanut butter.
7. Whole-grain bread, iron-enriched pastas, grains, rice, and cereals.

B. Foods to avoid:

1. Avoid high oxalate foods including tea, coffee, spinach, kale, strawberries, chocolate, and broccoli. They can decrease absorption of iron.
2. Foods that can impair iron absorption include eggs and dairy products. Wait several hours between taking iron and eating dairy products or eggs.

You Have Been Prescribed the Following Medication: _____

Iron supplements should be taken on an empty stomach.

Take iron supplements with a glass of orange juice or with a vitamin C tablet.

If you take antacids, take the iron supplement 2 hours before or 4 hours after the antacid to improve absorption.

Iron supplements may turn your stools black, which is a harmless side effect.

You Need to Notify the Office If You Have:

A. Shortness of breath, rapid heartbeat, or chest pain with activity.
B. Constipation unresolved using over-the-counter preparations, or if you have the need for daily use of preparation for constipation.
C. Upcoming surgery.
D. Other: _____

Phone: _____

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CLIENT TEACHING GUIDE

LYMPHEDEMA

PROBLEM Lymphedema is the backup of fluid in the lymphatic system into an arm or leg. The fluid causes severe swelling, which restricts movement and can lead to infection and, in rare cases, a form of cancer called lymphangiosarcoma.

CAUSES Many factors can cause lymphedema. These include a diagnosis of cancer treated with radiation, surgical removal of lymph nodes, infection, and disorders from birth, which affect the structure of the lymph system.

PREVENTION

A. Protect your arm or leg while recovering from cancer treatment.
  1. Avoid heavy lifting, if it involves an arm.
  2. Avoid strenuous exercise.
  3. Avoid heat on your arm or leg.
  4. Avoid tight clothing.
  5. Wear a glove when there is a risk of injury to the hand.

TREATMENT PLAN

A. Raise the affected arm or leg whenever lying down to improve pain and swelling.
B. Wear compression stockings. Put them on before getting out of bed in the morning. Take them off just before going to bed at night.
C. Apply lotion every day to the affected area.
D. Check the affected area every day and report any skin changes to your healthcare provider, especially any cracks or cuts.
E. Follow up with your primary healthcare provider and other specialty providers on a regularly scheduled basis.

Activity:

A. Get regular exercise. Discuss with your healthcare provider what type and frequency of exercise is safe for you.
B. Exercise leg muscles by pumping your ankles when sitting. Rocking in a rocking chair is another option.

Diet:

A. Discuss with your healthcare provider the type of diet that best suits your needs: diabetic diet, low-fat diet, low-cholesterol diet, and/or low-sodium diet.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Any new symptom(s) not present at your last office visit.
B. Any of these symptoms:
  1. Fever over 101°F.
  2. Increased redness, pain, tenderness to touch, and/or warmth in the affected arm or leg.
  3. New pain, swelling, or warmth in an arm or leg.
C. Other: _____

Phone: _____

RESOURCE Vascular Cures–Lymphedema: http://vascularcures.org/lymphedema/

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CLIENT TEACHING GUIDE

REFERENCE RESOURCES REGARDING HIV/AIDS

A. The Body, The Complete HIV/AIDS Resource is the most visited internet site about HIV: www.thebody.com.
B. The "Well" Project is designed for HIV-positive females, children, and family: www.thewellproject.org/.
C. The AIDS InfoNet provides reliable and up-to-date treatment information: www.aidsinfonet.org.
D. AIDS Healthcare Foundation: www.aidshealth.org.
E. Centers for Disease Control and Prevention (CDC) provides general information on HIV: www.cdc.gov/hiv.
F. POZ Health Services Directory is a comprehensive guide to HIV care and services: directory.poz.com/napwa.
G. Information regarding preexposure prophylaxis: www.hiv.gov/federal-response/ending-the-hiv-epidemic/prep-program-resources.

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CLIENT TEACHING GUIDE

VITAMIN B_{12} INCLUDING PERNICIOUS ANEMIA

PROBLEM You have been diagnosed with a condition called vitamin B_{12} anemia. You may have a type of B_{12} anemia called pernicious anemia. It is a condition in which vitamin B_{12} is not well-absorbed. Vitamin B_{12} is necessary for red blood cell function.

CAUSE Vitamin B_{12} anemia is a common problem in pregnancy, with vegetarian diets and with previous stomach problems, and as you get older. If you have been told you have pernicious anemia, your condition may be caused by the lack of a special factor in your stomach juices whereby your body cannot absorb the vitamin, or it may be from an autoimmune reaction.

PREVENTION Vitamin B_{12} anemia is treatable; however, pernicious anemia requires lifelong treatment.

TREATMENT PLAN

A. If you have pernicious anemia, you will need vitamin B_{12} injections for the rest of your life. This treatment cannot be given in pill form.

1. After you have been on the shots for a while, the nurses can teach you or a family member to give the shot. Please ask your healthcare provider about this.
2. Common side effects of vitamin B_{12} shots include:
   a. Pain and burning at the place the shot is given. This does not last very long.
   b. Some people experience diarrhea after taking the shot.

B. You may need to take iron tablets, too.

C. You may be sent to see a nutritionist to help you review your diet and how you prepare foods.

Activity: Vitamin B_{12} anemia may cause the loss of some senses and give you numbness and tingling, memory loss, loss of coordination, and some depression or irritability.

A. It is important to avoid extremely hot foods and drinks.

B. It is important to use caution in your home, such as the following:

1. Do not use loose "scatter" rugs, which can cause slips.
2. Install shower or tub rails to help get in and out, as well as toilet rails to get up and down more easily.
3. Use handrails going up and down stairs.
4. Do not use extremely hot water for bathing, showers, and doing dishes.
5. Use nonslip surfaces in the tub and shower.
6. Do not use a heating pad if you do not have all your sensations, in order to avoid burns.

A home safety evaluation should be done to reduce the risk of falls. A checklist can be obtained from smartcarema.com/wp-content/uploads/2015/05/Home-Safety-Assessment-Checklist.pdf.

Diet: A balanced, healthy diet is important. Increase your liquids to 8 to 10 glasses a day; iron supplements tend to cause constipation. Increase the fiber in your diet. If you are over 50 years of age, you need to increase your intake of low-fat dairy products such as yogurt and milk, and B_{12} fortified foods. If you follow a vegan diet, include foods fortified with vitamin B_{12}.

Medication: _____

You Have Been Prescribed the Following Iron Supplement: _____

You Need to Take It: _____

Your Next Vitamin B_{12} Shot Is Due: _____

You Need to Notify the Office If: _____

A. You feel worse the first week after the shot or have symptoms such as chest pain and shortness of breath.

B. You have worsening symptoms, such as problems with balance and walking.

C. You have leg pain, especially when you put your weight on it.

D. You would like to make arrangements for home injections.

E. Other: _____

Phone: _____

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CHAPTER 21

MUSCULOSKELETAL GUIDELINES

Carsen Cash and Jill C. Cash

AGING KNEE/KNEE PAIN

DEFINITION

A. The knee is the largest joint of the body. The knee is not protected by layers of fat or muscle; thus, exposure of the knee may lead to a high incidence of trauma. The articular cartilage is a firm, rubbery protein material covering the end of a bone. It acts as a cushion between the bones so that the bones do not grind together. When this articular cartilage breaks down, symptoms such as knee pain, swelling, bone spur formation, and decreased motion develop. Over a period of years, the joint slowly changes and normal activity becomes painful and difficult.

B. There is a loose relationship between knee pain and osteoarthritis of the knee. Many older people are pain-free with radiologic arthritis of the knee, whereas many people with knee pain have little evidence of osteoarthritis.

INCIDENCE

A. One-fourth of people aged 50 and older report chronic knee pain; for two-thirds of these, the pain is severe and disabling. Osteoarthritis of the knee is one of the five leading causes of disability among elderly males and females.

PATHOGENESIS

A. Clients with knee pain who do not have inflammatory arthritis may have crepitus, bony tenderness, bony enlargement, effusion, or muscle wasting. Depending on the cause of the problem, knee pain occurs due to problems such as bone rubbing bone, extra fluid in the joint space which causes pressure, irregularities of the bone, and torn cartilage.

PREDISPOSING FACTORS

A. Heredity.

B. Weight gain: increases pressure on the knee joints. A pound of weight gain equals approximately 4 lbs of weight pressure on the knee joint.

C. Age: The ability of the cartilage to heal itself decreases with age.

D. Sex: Occurs more frequently in females than in males.

E. Trauma and history of injury to the knee.

F. Repetitive injuries in sports or other trauma.

G. Activity requiring long periods of standing and poor posture/gait.

H. Chronic conditions: metabolic disorders, muscle weakness, poor fitness, vitamin C deficiency, and vitamin D deficiency.

COMMON COMPLAINTS

A. Knee pain at rest and with activity.

B. Inability or decreased ability to perform certain daily functions.

C. Knee gives way while walking, standing, or moving.

D. Difficulty with getting up out of a chair.

E. Need for assistive devices to ambulate.

OTHER SIGNS AND SYMPTOMS

A. Inability to stand, walk, or sit for long periods of time.

B. Pain may increase with exposure to cold or heat.

C. Pain at rest.

POTENTIAL COMPLICATIONS

A. Falls.

B. Fractures.

C. Inability to complete activities of daily living (ADLs) and/or other activities.

SUBJECTIVE DATA

A. Inquire regarding onset, location, duration, and frequency of knee pain.

B. Did trauma precipitate the pain, either recently or long ago?

C. Elicit a clear description of the pain and factors that aggravate or relieve it.

D. Does knee swell, feel warm to touch, or turn red at any time?

E. Does pain increase or worsen with rest/activity?

F. Ask the client to report a pain scale with rest and with activity.

G. How does pain interfere with daily living activities? Is the client unable to work? Does knee pain waken the client at night? Do they have difficulty with walking, climbing steps, or getting into the car?

H. What has the client used for pain? Over-the-counter (OTC) products and prescription products? Heat/ice/brace and so forth?

I. Has the client experienced any type of pain like this in the past?

J. Does the client have any medical and surgical history regarding the knees?

K. Symptoms and signs of common knee pathologies:

1. Meniscus tear: locking of knee with flexion and giving way of knee.

2. Collateral ligament tear or strain: pain at lateral or medial sides; instability.

3. Anterior cruciate tear: popping sound at injury site and immediate swelling. ▶

The contributions of Julie Adkins to this chapter in prior editions are acknowledged here.

4. Posterior cruciate tear or strain: pain in interior knee.
5. Patellofemoral syndrome: popping or snapping, pain under the patella with motion, and pain on stairs or hills; pain in the anterior knee after sitting for a long period of time (movie theater sign).
6. Tendinitis: pain over the patellar tendon.
7. Prepatellar bursitis: swelling over the patella with inability to kneel due to swelling.
8. Nonspecific effusion: effusion worse with exercise.

PHYSICAL EXAMINATION

A. Inspect:
1. Gait: Observe for any abnormal movement used to compensate for the pain in the knee.
2. Note symmetry of muscles above the knee for atrophy.
3. Assess the knee for swelling, redness, or gross deformity. Swelling is classified as either localized (bursa) or generalized (intraarticular). A bursal swelling is most frequently found over the patella or over the tibial tubercle. Occasionally, a bursal swelling may present in the popliteal area due to a cyst. Intraarticular swelling is usually due to an irritation of the synovium and can cause a generalized swelling, or an effusion, of the entire knee.
B. Palpate:
1. Palpate the knee for points of tenderness, effusion, or changes in skin temperature. The knee is easier to palpate when it is flexed, making the skeletal landmarks more distinct. Palpate both the medial and lateral aspect of the knee and joint line area.
2. Test for joint stability involving the collateral ligaments and cruciate ligaments.
3. Assess range of motion (ROM) actively and passively: flexion, extension, and internal and external rotation (internal and external rotation occurs at the level of the hip, so look for causes of hip pain if this elicits pain at the hip or knee).
4. Perform McMurray test to evaluate for torn meniscus.

DIAGNOSTIC TESTS

A. X-ray of affected knee or both knees for comparison. Perform knee x-rays while the client is standing.
B. Possible MRI or CT as necessary. Consider if mechanical symptoms and effusion is present.
C. Bone scan may be considered if the client has failed to respond to therapy after 6 to 12 weeks of therapy.
D. Laboratory values to consider: complete blood count (CBC), erythrocyte sedimentation rate, uric acid, rheumatoid factor.

DIFFERENTIAL DIAGNOSES

A. Knee trauma (meniscal tears, medial or lateral collateral ligament tear, anterior cruciate ligament tear, patella dislocation/subluxation, fracture, osteochondral defect).
B. Septic arthritis (must not miss).
C. Arthritis (rheumatoid arthritis, psoriatic arthritis, reactive arthritis, osteoarthritis).
D. Crystal arthropathies (e.g., gout, pseudogout, nontraumatic knee pain that is not associated with activity, pain with rest).
E. Bursitis.
F. Referred pain from the hip or back.
G. Baker cyst (posterior knee pain not related to trauma).
H. Malignancy.
I. Avascular necrosis.
J. Fibromyalgia.
K. Nontrauma acute knee pain that increases acutely with activity, including cartilage injury and osteoarthritis.

PLAN

A. General interventions:
1. Knee exercises and bracing, if necessary.
2. Activity modifications should be discussed with the client to reduce pain. Exercise is good for chronic knee conditions like osteoarthritis and can likely help with weight loss.
3. Nonsurgical management includes appropriate footwear and assistive devices.
4. Complementary and alternative modalities include glucosamine chondroitin, acupuncture, osteopathy, and chiropractic interventions.
5. Joint aspiration, if indicated.
B. Client teaching:
1. Weight loss education should be discussed and encouraged. Dietary management along with exercise routine should be considered. Approximately 4 to 5 lbs of pressure on the knee are relieved with every 1 lb of weight loss.
C. Pharmacologic therapy:
1. Acetaminophen.
2. Nonsteroidal anti-inflammatory drugs (NSAIDs; e.g., naproxen, ibuprofen, Mobic).
3. Topical medication such as capsaicin, topical NSAIDs, or lidocaine patches may be considered.
4. Topical medication, diclofenac (Voltaren gel), may be used on large joints such as the knee QID as needed for joint pain.
5. Intraarticular treatments include the following:
a. Steroid injections (Depo-Medrol), which may provide short-term relief (1–4 weeks).
b. Hyaluronan injections (viscosupplementation) may be considered but are not routinely recommended due to lack of quality evidence showing benefit over intraarticular placebo.
c. Platelet-rich plasma injections have a growing population of research surrounding its use, but due to current lack of evidence many do not recommend its use.

FOLLOW-UP

A. Follow up in 1 to 2 weeks for evaluation of knee pain and prescribed modality effectiveness.
B. Evaluate for side effects of pharmacologic treatment.

CONSULTATION/REFERRAL

A. Refer to physical therapy for exercise regimens or physiotherapy. May include knee taping and water therapy.
B. Nonoperative sports medicine referral may be appropriate depending on the practitioner's level of confidence with the presenting complaint.
C. Surgical consultation if there is no relief from pharmacologic or physical therapy methods. May include arthroscopy versus partial or total knee replacement, dependent on the objective findings.

INDIVIDUAL CONSIDERATIONS

A. Adults:
1. Evaluate client individually for renal, gastrointestinal, or hepatic disorders in regard to pharmacologic preparations. ▶

B. Geriatrics:

1. Ensure access to proper eyeglasses, hearing aids, and assistive devices such as canes, walkers, elevated toilet seats, grab bars, and shower chairs.

2. Genetic mutations and aging decrease the cartilage's ability to heal itself; females over 50 years old will develop knee osteoarthritis more than males; and previous injury to the knee will lead to challenges (possibly knee replacement) after the age of 60 years.

3. Differential diagnoses specific for geriatrics with knee pain include the following:

a. Chondromalacia patellae: tenderness over the kneecap.

b. Iliotibial band syndrome: lateral knee tenderness at the fibular head and lateral femoral condyle.

c. Anserine bursitis: pain distal to the knee radiating over the medial tibia.

4. Physical therapy effectively reduces chronic knee pain and is the first-line nonpharmacologic pain management strategy of choice.

5. Black cumin oil to alleviate pain in geriatrics suffering with chronic knee pain. The oil is to be applied and generously massaged into the knees three times a week for 1 month to experience effective results. Afterward, once a day or three times a week for maintenance pain management (client-specific).

6. Systematic reviews of clinical trials compared young adults (<65 years of age) and geriatric population (≥65 years of age) who had undergone knee arthroplasty and observed there was neither evidence of pulmonary embolism incidences nor pain/functional status differences between the two groups. However, risks and benefits must still be thoroughly discussed with geriatric clients for them to make an informed decision with their surgeons.

7. Geriatric clients are at higher risk of falling due to functional decline. Falls increase morbidity and efforts should be made to prevent them.

8. Evaluate polypharmacy and other comorbidities and possible drug interactions.

9. Evaluate for medication compliance and level of alertness before starting drugs with potentially hazardous side effects.

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BUNION

DEFINITION

A. A bunion is a bony enlargement at the base of the great toe. The metatarsophalangeal (MTP) joint of the first (great) toe moves into a valgus position, hence the alternative name hallux valgus.

INCIDENCE

A. Approximately 9 out of 10 bunions occur in females. 9 out of 10 females wear shoes that are too small and/or too tight, and/or are too narrow.

PATHOGENESIS

A. The first metatarsal is angled medially and dorsally by increased pressure, which causes a bony prominence at the MTP joint. Muscles work to stabilize this deformity and will shift the phalanx laterally, causing the abnormal angle at the base of the great toe. The great toe may angle laterally toward the second toe or move all the way under it, causing the skin under the foot to become thicker and more painful. This may force the second toe out of alignment and overlap the third toe. The pain may become chronic.

PREDISPOSING FACTORS

A. Likely multifactorial, including abnormal foot mechanics, abnormal first MTP joint anatomy, genetics.

B. Wearing poorly fitting shoes or shoes that cause abnormal foot mechanics.

COMMON COMPLAINTS

A. Pain in the great toe at rest and with walking/movement.

B. Difficulty wearing shoes.

C. Great toe looks deformed due to hallux valgus deformity.

OTHER SIGNS AND SYMPTOMS

A. MTP joint is tender and enlarged.

B. Client cannot walk normally due to pain.

C. It hurts to bend the toe.

POTENTIAL COMPLICATIONS

A. Acute arthritic pain.

B. Bursitis.

C. Hammertoe deformity of second toe.

D. Nerve entrapment.

SUBJECTIVE DATA

A. Ask the client what activity brought about or preceded the episode of pain.

B. Ask the client to describe the pain; note duration of pain and impairment of activity.

C. Inquire regarding any limited range of motion (ROM) of toe/toes.

D. Ask the client if there is any evidence of sores, calluses, or open areas of skin.

E. Ask the client what treatments have been used and what makes the pain better or worse.

F. Ask the client to list all medications currently being taken, including over-the-counter (OTC) preparations.

PHYSICAL EXAMINATION

A. Inspect:

1. Inspect the MTP joint for deformity and enlargement.

2. Inspect skin for erythema, skin irritations, open sores, drainage, and calluses. ▶

3. Evaluate active ROM of MTP joint, plantar flexion, and dorsiflexion; compare with contralateral side.
4. Evaluate for other toe involvement.

B. Palpate:
1. Palpate the MTP joint for tenderness.
2. Check passive ROM for rigidity and crepitation and degree of pain on movement.
3. Palpate peripheral pulses in legs.

DIAGNOSTIC TESTS

A. None. Advanced cases are easily diagnosed by inspection and exam.

B. X-rays may be used to confirm the diagnosis and to assess for inflammation and joint changes.

DIFFERENTIAL DIAGNOSES

A. Bunion.
B. Morton neuroma.
C. Bursitis.
D. Gout.
E. Infection.

PLAN

A. General interventions:
1. Recommend the use of a bunion shield/pad. Low-heeled shoes with a wide toe box and splinting may be beneficial.
2. Advise using ice over the side and top of the toe for comfort and elevation.
3. Encourage performing stretching exercises of extensor and flexor tendons.
4. **Subacute cases (4–6 weeks)** may require a nonsteroidal anti-inflammatory drug (NSAID) for pain relief, dependent on renal function of the client. They may require a local intraarticular injection for pain/inflammation relief.
5. **Chronic cases (8–10 weeks)** may require a referral to an orthopedist or podiatrist.
6. Cases involving severe pain and/or difficulty walking warrant x-rays and surgical referral.

B. Client teaching:
1. Educate the client on the most common cause (tight-fitting shoes). Encourage the client to wear wide-toed shoes.
2. Educate the client on use of a cotton or rubber spacer to be worn between first and second toe.

C. Pharmaceutical therapy:
1. NSAIDs may be used to reduce pain and inflammation, unless the client has intolerance to NSAIDs. Acetaminophen may also be useful.
2. Further therapy may be considered for chronic conditions/severe pain.

FOLLOW-UP

A. Follow-up is determined by the client's needs and by whether complications are present. All treatments, including surgery, are nonurgent.

CONSULTATION/REFERRAL

A. Orthopedist or podiatrist referral for chronic cases; surgical intervention may be helpful.

B. Physical therapy for stretching exercises of the extensor and flexor tendons before deformity becomes permanent.

C. All treatments, including surgery, are nonurgent.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. Use precautionary measures when using NSAID therapy in the geriatric population due to possible gastrointestinal and renal adverse effects.
2. Taping is an effective therapy for improving bunions (hallux valgus) in adults; however, its use in geriatrics may be associated with impaired balance and decreased ability to walk up and down stairs.
3. Hallux valgus increases fall risk in the geriatric population. Research indicates that moderate-severe deformity and bunion-related pain impair physical function and balance independent of knee and hip arthritis. Consider discussing surgical interventions for these clients and review fall safety measures in the home.
4. Corrective surgery for the geriatric population has potential risks: deep vein thrombosis, pulmonary embolism, and infection. Bunion surgery requires cast immobilization, which increases risks for clotting. Preop education must include therapeutic interventions such as anticoagulant therapy (long-/short-term), modifications for nonweight-bearing activities of daily living (ADLs) and exercises, and professionally monitored physical therapy (after recovery) when returning to consistent weight-bearing activities. Surgery is usually not an option for clients who are ≥80 years old, or have uncontrolled diabetes, a history of systemic cancers, or uncontrolled gout.

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DEGENERATIVE DISC DISEASE (NECK/LUMBAR)

DEFINITION

A. Degenerative disc disease is a general term for the gradual deterioration of the discs between the vertebrae of the spine that can occur naturally or through injury. Injuries causing degenerative disc disease often involve twisting motions, where portions of the disc may tear, resulting in thinner and more fragile cartilage. Any level of the spine can be affected. The disc itself has very few nerve endings and no blood supply. The disc has no way for self-repair mechanism, and the pain created by the damaged disc can either be a chronic condition or have periodic flare-ups.

INCIDENCE

A. In the United States, back pain is the second leading symptom that prompts visits to a healthcare provider. As many as 80% of adults experience at least one episode of back pain during their lifetime and 5% experience chronic problems with their back. Cervical degenerative disc disease is much less common than disc degeneration of the lumbar spine. After a person reaches the age of 60, some level of disc degeneration is a normal finding rather than an exception.

PATHOGENESIS

A. The degenerative changes of the spine can be subdivided into three stages:

1. Dysfunction: may involve outer annular tears, separation of the endplate, cartilage destruction, and facet synovial reaction. Inflammation can cause muscle tension and spasms, leading to pain and local tenderness.

2. Instability: disc resorption and loss of disc space height, possibly leading to subluxation.

3. Restabilization: progressive changes leading to osteophyte formation or bone spurs and stenosis.

PREDISPOSING FACTORS

A. Injury/inflammation.
B. Age-related, occurs more commonly as one ages.
C. Genetic component.
D. Smoking and obesity have been linked to increased incidence of degenerative disc disease.

COMMON COMPLAINTS

A. Pain that is usually related to activity; flare-ups at times, but then returns to a low-grade pain level or pain will go away entirely. Cervical disc degeneration is most frequently felt as a stiff neck. Lumbar symptoms may include local tenderness, hypomobility, and painful extension of the lumbar back.
B. Muscle spasms of the neck, arms, or shoulders due to cervical nerve inflammation or impingement. Lumbar symptoms may include a "giving away or catch in the back" and pain with standing and/or flexion.
C. Headache, muscle tenderness, stiffness, and reduced movement; pain in buttocks or upper thighs.

OTHER SIGNS AND SYMPTOMS

A. Paresthesia of upper extremities and hands with decreased strength and hand grip.
B. Referred pain to hips, knees, ankles, and feet.
C. Progression to chronic pain (pain lasting over 3 months).

POTENTIAL COMPLICATIONS

A. Presence of bowel and bladder symptoms.
B. Cauda equina syndrome.
C. Disability.

SUBJECTIVE DATA

A. When did the pain begin?
B. What activity brought about or preceded the episode? Was there an injury that could be related to the pain? Activity modification?
C. Rate the pain on a scale of 0 to 10, with 0 being no pain, or on the visual analogue scale, a series of faces of varying intensity.
D. Does the pain radiate to any other point of the body?
E. What factors make the pain better or worse?
F. Are there any problems with bladder or bowels, or has there been paresthesias in the saddle region?
G. Have there been fevers, chills, night sweats, or weight loss?
H. What medications are currently being taken or have been tried, including over-the-counter (OTC), topicals, and herbal?
I. Is there any family history of osteoporosis or degenerative disc disease?

PHYSICAL EXAMINATION

A. Vital signs as appropriate. Notice red flags such as weight loss, fever, elevated pulse.
B. Start with a general physical examination to determine the source of the problem, dependent on where there is pain: neck, lower back, arms, legs, and so forth.
C. Inspect:
1. Inspect general appearance, skin rashes, or deformities.
2. Observe gait and assistive device use, if any.
D. Palpate:
1. Assess the motion of spine and neck. Assess for pain with twisting, bending, or moving.
2. Assess muscle strength or weakness.
3. Palpate areas of tenderness or muscle spasms.
4. Sensory changes: Assess increased or decreased sensation associated with point of tenderness.
5. Check deep tendon reflexes (DTRs).
6. Assess motor skills such as toe or heel walk.
E. Auscultate:
1. Auscultate carotid arteries.
2. Auscultate abdomen.
3. Conduct a complete heart exam.
4. Conduct a complete lung exam.

DIAGNOSTIC TESTS

A. X-ray of affected area may reveal a decrease in the height space between disc, bone spurs, sclerosis, facet hypertrophy (enlargement), and instability during flexion or extension of limbs. X-ray will show fracture, infections, or tumors if suspected. Flexion and extension radiographs have limited value in degenerative disease, but they are still appropriate to obtain if worried about other causes of neck or back pain.
B. MRI or CT scan is used for more severe causes, such as loss of water in a disc, stenosis, or herniated disc, as well as to rule out differential diagnoses.

DIFFERENTIAL DIAGNOSES

A. Degenerative disc disease.
B. Muscle strain.
C. Disc herniation.
D. Infection.
E. Fracture.
F. Inflammatory cause, such as ankylosing spondylitis.
G. Tumor.
H. Cauda equina or conus medullaris syndromes.
I. Fibromyalgia.

PLAN

A. General interventions:
1. Initial decisions are made according to the seriousness of the findings. Some problems need immediate attention, possibly even surgery. The vast majority of problems, however, do not require immediate surgery.
B. Client teaching: *See Client Teaching Guide for this chapter, "Neck Exercises."*
1. Educate the client about avoiding actions that aggravate the condition:
a. Modify activities to avoid lifting heavy objects and playing sports or other activities that require rotating the back or neck.
b. Teach correct ergonomics, such as how to lift heavy objects, how to sit in a chair, and how to correct sleep postures.
2. Nonsurgical interventions:
a. Rest.
b. An exercise program or physical therapy that may strengthen the back and avoid further inflammation or injury.
c. Alternating heat and ice to the affected site. ▶

C. Pharmaceutical therapy:

1. Mild pain medications can reduce inflammation and pain. These medications will not stop degeneration, but they will help with pain control.

a. Nonsteroidal anti-inflammatory drugs (NSAIDs) block the inflammatory response in joints. Use can decrease renal function and excessive use can lead to kidney problems.

b. Nonnarcotic pain relievers: The most commonly used are acetaminophen and aspirin, but they must be avoided in clients with gastrointestinal disorders or liver problems.

c. Narcotic pain medications: Narcotics relieve pain by acting as a numbing anesthetic to the central nervous system. Their intended use should be for treatment of severe pain that is not expected to last more than a few days. Narcotics can cause many side effects, such as nausea, vomiting, constipation, sedation, or drowsiness. Narcotics may be addictive. Precautions about falls are paramount with these types of medications.

d. Muscle relaxants can help relieve pain but have been shown to be only marginally effective. There is a significant risk for drowsiness and falls. Fall precautions and driving restrictions must be advised while using narcotics and muscle relaxants.

FOLLOW-UP

A. Follow-up is determined by the client's response to conservative measures for pain relief, impact on activities of daily living (ADLs), ability to carry out work duties, or worsening of symptoms.

CONSULTATION/REFERRAL

A. Physical therapy: may include modalities of heat and ice, massage, ultrasound, and electric stimulation. Other treatments may include bracing, flexibility and strength training, pool therapy, and/or posture training.

B. Referral for epidural steroid injections: a combination of steroid and a local anesthetic given through the back into the epidural space. Injections are used when conservative treatments options have failed. Referral should be made to a pain control specialist for epidural steroid injections.

C. Referral to a neurosurgeon if immediate, emergent findings are found on evaluation or if symptoms last over 6 months without any relief.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Assess living conditions, support systems, transportation issues, and impact on ADL. Educate the client regarding the disease process and review possible treatment options.

2. Assess all medications the client is currently taking for interactions and review precautions with the client regarding pain control medications used.

3. Review assistance that is available to avoid aggravating activities. Implement education for back posture, ergonomics with table computers and laptops/electronic tablets, lifting precautions, and abdominal muscle strengthening.

4. When prescribing pain medications or muscle relaxants, discuss ways to avoid falls and injuries, noting side effects of medications, such as drowsiness that may occur when performing ADLs. Acupuncture and/or corticosteroid injections might be beneficial. Radiofrequency denervation most often does *not* alleviate pain from intervertebral discs.

5. Special considerations are needed if surgery is necessary: family support, rehabilitation, transportation, and home healthcare.

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HAMMER TOE

DEFINITION

A. A hammer toe is a deformity of the second, third, or fourth toe. The toe is bent at the proximal interphalangeal (PIP) joint, resembling a hammer.

PATHOGENESIS

A. Hammer toe results from shoes that do not fit properly or a muscle imbalance. Muscles work to straighten and bend the toes. If the toe is bent for long periods, the muscles tighten, leading to the deformity. Trauma is also a common mechanism for the development of hammer toe.

PREDISPOSING FACTORS

A. Shoes that narrow at the toe push the toes into a bent position, making the toes rub against the shoe. Eventually, the toe muscles are unable to straighten the toe with any shoe or barefoot.

COMMON COMPLAINTS

A. "My toe is bent."

B. "I can't find shoes to fit due to my bent toe."

C. "I have a sore or callus on my toe because it won't straighten out."

OTHER SIGNS AND SYMPTOMS

A. Formation of corns and calluses on the middle joint of the toe or on the tip of the toe.

B. Pain in the toes and feet.

C. Difficulty finding shoes to fit feet.

POTENTIAL COMPLICATIONS

A. Ulcers of the middle joint of the toe may develop, possibly leading to infection or osteomyelitis.

B. The client may have chronic pain of the toe. Abscesses of corns and calluses may develop.

SUBJECTIVE DATA

A. Is there pain over the ball of the foot?

B. Is the toe rubbing on shoes abnormally?

C. Is skin over the top of toe starting to thicken?

D. Do the toes appear crooked?

PHYSICAL EXAMINATION

A. Inspect:

1. Take notice of any deformity of the toe and surrounding toes.

2. Examine the metatarsophalangeal (MTP) joints for irritation and thickening.

3. Note corns and calluses on top of the toe if present.

4. Examine the extensor tendons of the toes for flexibility. It should be tight when the ankle is placed in plantar-flexion position.

B. Palpate:

1. Palpate over MTP joints. Note tenderness over the MTP joint and a positive MTP joint squeeze.

2. Note active and passive range of motion (ROM) of all toes and ankle.

DIAGNOSTIC TESTS

A. X-rays:

1. X-rays are not routinely recommended. They rarely provide any additional information not found on physical examination.

2. X-rays should be performed if severe swelling, unusual color, or unequal movement/ROM of toes is present.

DIFFERENTIAL DIAGNOSES

A. Hammer toe.

B. Rheumatoid arthritis.

C. Osteoarthritis.

D. Reflex sympathetic dystrophy: Consider if there is extensive swelling and discoloration.

PLAN

A. General interventions:

1. Conservative treatment consists of new shoes with soft, roomy toe areas. Shoes should be 1/2 in. longer than the longest toe. Sandals may help as long as they do not pinch or rub the affected area of the toe. Toe exercises can stretch and strengthen the muscles, and toes can be gently stretched manually. Advise using corn pads or cushions to cover calluses or thickened skin. Consider paring the large corns and calluses with sharp dissection. Toe spacers may also be suggested to use for comfort.

B. Pharmaceutical therapy:

1. Local steroid injection at the most painful MTP head may be considered, with reemphasis on passive stretching exercises.

FOLLOW-UP

A. Consider injection of the joint if the case is severe. Aftercare treatment involves resting the foot for 3 days, wearing loose-fitting shoes, using a toe spacer, and applying ice for approximately 15 minutes every 4 to 6 hours.

B. Restart passive stretching of the foot after 3 to 4 weeks.

C. If pain recurs or persists, repeat injection at 6 weeks.

CONSULTATION/REFERRAL

A. Physical therapy for active assessment and treatment. Increasing the flexibility of the MTP joints and toes will prevent hammer toe.

B. Consider referral to a surgeon/podiatrist for evaluation for surgical intervention, such as tendon lengthening, tendon transfer, or arthrodesis.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. Evaluate the client's ability to perform passive stretching exercises due to other comorbidities.

2. Assess clients with diabetes for corns and calluses. Monitor for infection, poor circulation, and/or decreased sensation in the feet.

3. Fall precautions should be discussed with clients at risk for falls who have pain/numbness of the foot or who wear adaptive footwear.

B. Geriatrics:

1. Orthoses may be inserted inside the shoe or custom-made to fit the foot to accommodate the toe's deformity. Instruct client and family to assess the foot's pressure points, and to alert the medical provider of any skin changes or signs of skin tears/ulcers.

2. For the elderly population, no surgery is recommended in the absence of pain.

3. Surgical correction of hammer toes gives significant improvement in older and younger adults and is not associated with an increase in complications even in the geriatric population.

4. Educate and encourage proper foot care for geriatrics. Avoid tight shoes and high heels; monitor feet for discoloration, calluses, and fungal infections; keep feet and toes dry and clean; trim nails straight across; and regularly see a podiatrist to assess for toenail fungus, ingrown toenails, and preventive measures of feet complications.

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MORTON NEUROMA

DEFINITION

A. A neuroma is a benign tumor of a nerve. Morton neuroma is not actually a tumor, but a chronic irritation/inflammation and thickening of the tissue that surrounds the digital nerve leading to the toes. This neuroma occurs as the nerve passes under the ligament of the metatarsals.

INCIDENCE

A. The neuroma usually occurs between the third and fourth toes.

B. It occurs in females 5 to 10 times more frequently than males.

PATHOGENESIS

A. Morton neuroma occurs when pressure is exerted from the bottom of the foot from walking on hard surfaces with poorly padded shoes or barefoot, and/or pressure from the sides of ill-fitting tight shoes. The third interspace is more narrow than the other interspaces; thus, the digital nerve at this location is more prone to compression and trauma. Chronic irritation and/or trauma have been linked to the development of Morton neuroma.

PREDISPOSING FACTORS

A. Poor padded shoes or walking barefoot.

B. Tight shoes or high heels.

C. Trauma.

COMMON COMPLAINTS

A. Burning pain in the ball of the foot radiating to the toes. Pain intensifies with activity and wearing shoes and is relieved with rest and removing shoes. Night pain is rare. Some say it feels as though they are walking on a stone or marble.
B. Possible numbness or unpleasant feeling in toes.
C. Sharp pain between toes, dead-feeling of toes.
D. Radiation of the pain to the hindfoot or leg, causing cramping, can occur.

OTHER SIGNS AND SYMPTOMS

A. Symptoms may worsen with tighter shoes than usual.
B. Selective shoes are worn for comfort.

SUBJECTIVE DATA

A. Describe the pain in the foot. Is it a burning, stabbing sensation in the toes?
B. Assess pain intensity. Ask regarding dull or sharp pain between toes.
C. Where does pain occur in the foot?
D. Does the client have difficulty walking due to pain or numbness?
E. Does the client take certain measures to improve pain, such as wearing specific shoes, avoiding shoes, or not going barefoot?

PHYSICAL EXAMINATION

A. Inspect:
  1. Inspect foot for redness, swelling, or pain.
  2. Inspect for color change, bruising, rash, or other changes in skin.
B. Palpate:
  1. Palpate affected foot for palpable mass or a "click" between toes.
  2. Apply pressure to area to reproduce pain for local tenderness.
  3. Assess active and passive range of motion (ROM) of toes and foot.

DIAGNOSTIC TESTS

A. X-ray of the foot to rule out other pathology, such as fracture.

DIFFERENTIAL DIAGNOSIS

A. Morton neuroma.
B. Fracture or stress fracture.
C. Arthritis.
D. Metatarsal stress fracture.
E. Ganglion cyst.

PLAN

A. General interventions:
  1. Recommend the use of orthoses to help relieve irritation by lifting and separating the bones to eliminate pressure on the nerve.
B. Client teaching:
  1. Make a change in footwear. Avoid high heels or tight shoes. Use of toe spacers may improve symptoms.
  2. Ice for soreness: Apply ice to site for 15 minutes every 4 to 6 hours. Acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs) may be used for pain.
  3. Encourage taking extra time in walking because of irritation.
  4. Encourage the use of assistive walking devices to reduce pressure on foot.
C. Pharmaceutical therapy:
  1. Injection: Local digital nerve block may be trialed.
  2. NSAIDs may have lower effectiveness due to poor penetration to these tissues.
  3. Neuropathic medications, such as tricyclic antidepressants (e.g., amitriptyline) and antiepileptic drugs (e.g., gabapentin), may be administered to lessen the severity of nerve pain.

FOLLOW-UP

A. One month or sooner to evaluate pain relief and/or exacerbating symptoms such as numbness or gait instability.
B. Steroid injection to the point of tenderness if there is no relief from conservative treatments. Steroid injection may be repeated in 6 weeks if pain recurs or persists.

CONSULTATION/REFERRAL

A. Referral to an orthopedist or podiatrist for possible neurectomy if other treatment modalities are unsuccessful or if pain continues after two steroid injections.

INDIVIDUAL CONSIDERATIONS

A. Adults:
  1. Injection: Aftercare to include rest for several days and avoidance of all unnecessary weight-bearing.
  2. Surgery may cause secondary problems, such as numbness in adjacent toes.
B. Geriatrics:
  1. Encourage client to wear properly fitting shoes that are comfortable and that do not place weight on the balls of the feet.
  2. Avoid or decrease activities of jogging, running, and jumping that place great amounts of impact on the toes or balls of the feet. Encourage geriatric clients to participate in brisk walking for cardiovascular aerobic exercise.
  3. Arch support devices and foot pads will help prevent neuropathy from developing further. Short-term steroid treatment may help with inflammation; however, proper footwear is the key to maintaining relief.

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NECK AND UPPER BACK DISORDERS

DEFINITION

A. Nonspecific disorders: self-limited, usually benign disorders with unclear etiology, such as regional upper back and neck pain and shoulder pain adjacent to the neck.
B. Degenerative disorders: consequences of aging or repetitive use, or a combination thereof, such as degenerative disc disease and osteoarthritis.
C. Potentially serious neck or upper back disorders: fractures, dislocation, infection, tumor, progressive neurologic deficit, or cord compression.

PREVALENCE

A. Neck pain in the adult population has a prevalence between 10% and 20%, similar to that of low back pain. In combination with upper back pain, the prevalence is higher.

PATHOGENESIS

A. Cervical strain is irritation and spasm of the upper back and cervical muscles. The upper portion of the trapezius and the levator scapulae muscles, rhomboid major and minor muscles, and the long cervical muscles are most often affected.

PREDISPOSING FACTORS

A. Whiplash-like injuries.
B. Cervical strain.
C. Cervical arthritis.
D. Holding your head in a forward posture or odd position. Frequently seen in clients who regularly use computers, cell phones/texting, laptops, and tablets.
E. Sleeping on a pillow too high or too flat.
F. Stress/tension.

COMMON COMPLAINTS

A. Aching neck.
B. Tightness and tenderness in neck area.
C. Stiffness and tightness in shoulders.
D. Stiff neck and a headache upon awakening.

OTHER SIGNS AND SYMPTOMS

A. Limited range of motion (ROM).
B. Back pain: guarding with cervical motion.
C. Numbness in upper extremities.
D. Muscle weakness.

SUBJECTIVE DATA

A. What are the presenting symptoms? Note pain, numbness, weakness, or stiffness.
B. Was there any type of injury, either recently or in the past?
C. Is the pain located primarily in the neck, upper back, or shoulder? Is there any radiation noted?
D. How do these symptoms limit the client's activity?
E. How long can the client sit, stand, walk, or do overhead work?
F. Is the client able to lift? If so, how much weight is bearable? Compare with normal weight.
G. How long has the client had these symptoms?
H. How have the symptoms evolved, from the beginning of discomfort until now?
I. If the client has a history of similar or the same pain, what therapy was used in the past and what were the results?
J. Does the client have any medical problems?

PHYSICAL EXAMINATION

A. Infection may include severe cervical spasms (nuchal rigidity), elevated temperature, chills, hypotension, and tachycardia.
B. Check temperature, blood pressure, and pulse.
C. Inspect:
  1. Observe stance and gait. Note the client's coordination and use of extremities.
D. Palpate:
  1. Palpate trigger points in upper back, paracervical, and rhomboid muscles.
  2. Palpate for any bony tenderness in neck, shoulders, and upper back.
  3. Perform ROM tests.
  4. Assess the client for reduced ipsilateral and contralateral bending of the neck.
  5. Check for fracture, or inability to move the neck due to pain, and severe cervical midline vertebral pain. Note tenderness, the client holding head for stability; look for possible neurologic deficits.
E. Assess:
  1. Assess deep tendon reflexes (DTRs), bilaterally:
    a. Biceps reflex tests fifth and sixth cervical nerve root.
    b. Brachioradialis reflex tests fifth and sixth cervical nerve root.
    c. Triceps reflex tests seventh and eighth cervical nerve root.
  2. Test muscle strength in shoulders.
  3. Abduction, elbow flexion, or supination tests fourth and fifth cervical discs.
  4. Check for weakness of radial wrist extension, indicating fifth and sixth cervical disc problems. Check for weakness of elbow extension and ulnar wrist flexion, indicating seventh cervical nerve impairment. Check weak finger abduction and adduction, indicating seventh and eighth cervical nerve impairment. Measure circumference at the forearm and upper arm for muscle atrophy. Dominant arm is 1/4 in. greater than nondominant arm.
F. Sensory:
  1. Test light touch, pinprick, pressure sensations in forearm and hand. Possible cervical spinal cord compromise is indicated by paresthesia of upper extremities, weakness of upper or lower extremities, and difficulty walking.
G. Percuss:
  1. Percuss back, spine, and neck areas. Tumor is indicated by tenderness to vertebral percussion, cachexia.
H. Auscultate:
  1. Auscultate heart and lungs.

DIAGNOSTIC TESTS

A. Radiography of cervical spine.

DIFFERENTIAL DIAGNOSES

A. Cervical strain.
B. Myofascial pain syndrome.
C. Cervical arthritis.
D. Cervical nerve root compression with radiculopathy.
E. Rotator cuff tendinitis.
F. Rotator cuff tendon tear.
G. Postlaminectomy syndrome.
H. Spinal stenosis.
I. Torticollis (which may be present at birth or caused by injury or disease).

PLAN

A. General interventions:
  1. Correct posture and lifestyle modifications (e.g., exercise, strengthening) are imperative for the client to remain free of pain. Client education and therapy depend on individual diagnosis.
B. Client teaching: *See Client Teaching Guide for this chapter, "RICE Therapy and Exercise Therapy."*
  1. Teach the client to use local applications of cold packs during the first 3 days of acute complaints and hot pack applications thereafter.
  2. Encourage the following changes in lifestyle:
    a. Sitting straight with shoulders held high.
    b. Sleeping with the head and neck aligned with the body and a small pillow under the neck.
    c. Driving with arms slightly shrugged, using arm rests.
    d. Avoiding carrying objects with a strap over shoulders.

e. Making ergonomic changes in computer, cell phone, tablet use, and so forth.

3. Suggest adjustments in tasks at work and at home.

4. Encourage daily stretching exercises, including shoulder roll, scapular pinch, and neck stretches.

5. Have the client perform ROM exercises daily.

6. Tell the client to avoid extremes of ROM, prolonged periods in one position, and any other aggravating activity.

7. Explain relaxation techniques and stress reduction.

C. Pharmaceutical therapy:

1. Nonprescription medications: acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs).

2. Prescription medications: NSAIDs for nighttime use.

3. The evidence for muscle relaxants (specifically cyclobenzaprine and tizanidine) in *acute* neck pain has a fair grade at best.

FOLLOW-UP

A. Evaluate the client after 2 weeks of conservative treatment. If pain continues after 2 to 3 weeks despite adequate therapy, order radiography, and physical therapy, including ultrasonography, massage, and gentle cervical traction, beginning at 5 lbs for 5 to 10 minutes once a day. Soft cervical collar may be worn while doing physical work.

CONSULTATION/REFERRAL

A. Consult or refer the client to a physician if there is still no improvement after adequate time for healing and no relief is noted with physical therapy and medications.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Geriatric neck and upper back pain are most often caused by the natural aging process of degenerative discs, falls (60%), forced hyperextension/hyperflexion injuries, poor body mechanics with reading/computer or sleeping (posture and improper pillows), or pathologic fractures.

2. Geriatric treatment management options include physical therapy, acetaminophen, short-term NSAIDs/steroids, rigid collar immobilization, halo cast with reduction, or surgical disc fusion for severe cases (uncontrolled pain). There is no difference in morbidity, complications, or mortality between younger and older adults with the latter treatment choices.

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PLANTAR FASCIITIS AND BONE SPURS

DEFINITION

A. Plantar fasciitis is an inflammatory condition in the plantar fascia that causes pain in the arch of the foot and radiates to the heel. The plantar fascia is a long, thin ligament lying directly beneath the skin on the bottom of the foot. It connects the heel to the front of the foot and supports the arch. Heel spurs refer to a thin strip of bone seen on radiographs that extend anteriorly from the posterior part of the calcaneus. Spurs were previously thought to cause pain; however, because many with a spur do not have plantar fasciitis, and many with plantar fasciitis do not have a heel spur, it is agreed upon that it is likely not the case that they cause plantar fasciitis pain.

INCIDENCE

A. Plantar fasciitis is among the most common causes of heel pain in the United States. Plantar fasciitis is seen in both males and females and most often affects active males ages 40 to 70 years. Approximately two million people are treated for this condition every year. One out of 10 have heel spurs, but only 1 out of 20 people with heel spurs have foot pain.

PATHOGENESIS

A. Repetitive small tears in the plantar fascia causing collagen breakdown at the medial tubercle of the calcaneus.

PREDISPOSING FACTORS

A. Athletes: overuse injury from running.

B. Tight or weak muscles/tendons (Achilles tendon, heel cord, gastrocnemius, soleus muscle).

C. Poor arch support/improper footwear (poor support in shoes).

D. Anatomic abnormalities (low arch support, flat foot, high arch, tibial torsion, overpronated foot, leg-length discrepancy, forefoot varus, thinning of fat pad).

E. Obesity.

COMMON COMPLAINTS

A. Severe foot pain in the bottom of the foot, especially first thing in the morning.

B. Burning pain when walking.

C. Stiffness in foot/heel.

D. May be worse in the morning, improve during day, and then get painful at the end of day.

OTHER SIGNS AND SYMPTOMS

A. Both heels may be affected.

B. Pain is located in the medial tubercle of the calcaneus, medial of the longitudinal arch.

C. Heel spurs may or may not be present.

D. Pain worsens with standing for long periods of time.

SUBJECTIVE DATA

A. Ask the client when pain began and how long it lasts. Is the pain constant or intermittent?

B. Does pain occur with walking, running, and standing? Is there nighttime pain?

C. Does pain begin on the first few steps after getting out of bed or after long periods of sitting or resting?

D. What is the quality or characteristics of the pain? Rate pain on a scale of 0 to 10.

E. Locate pain site; does pain radiate into toes or leg?

F. Was there a behavior change during/around the time of the pain, such as a new workout routine, new footwear, and so forth?

G. Does anything relieve the pain?

PHYSICAL EXAMINATION

A. Inspect:

1. Examine feet bilaterally. Assess for high arch.

2. Note swelling, discoloration, or rash.

B. Palpate:

1. Palpate both feet, noting tenderness at point tenderness. Point tenderness will be noted over insertion on medial heel (calcaneus medial tubercle). ▶

2. Perform passive dorsiflexion of toes and ankle. Have the client stand on tips of toes to see if this elicits pain.

C. Auscultate:

1. Auscultate the heart and lungs.

DIAGNOSTIC TESTS

A. X-ray may be performed but is often normal and not needed. Perform if tumor, spur, or fracture is suspected.

B. MRI may be ordered if thickening of proximal plantar fascia is noted or rupture of proximal fascia is suspected.

DIFFERENTIAL DIAGNOSES

A. Plantar fasciitis.

B. Heel pain.

C. Heel spur.

D. Fracture, including calcaneal or talar stress fractures.

PLAN

A. General interventions:

1. Conservative treatment includes no long periods of standing for the next 6 to 8 weeks.

B. Client education:

1. Arch support: Encourage the client to get a pair of shoes with good arch supports. New shoes with supportive arches or adding arch supports into the shoes is suggested.

2. Shoe inserts are suggested. Suggest getting proper shoe fitting for running if the client is an athlete.

3. The client should avoid walking on hard surfaces and never go barefoot. The client should also avoid wearing sandals and flip-flops.

4. Ice therapy may help with pain control and swelling. One can freeze a smooth plastic water bottle nearly full of water and carefully roll this underneath the sole of the foot for an ice and massage therapy combination.

5. Exercises:

a. Roll foot arch with a tennis ball for 20 to 30 minutes each evening to help stretch plantar fascia.

b. Perform calf stretches against a wall, leaning forward against the wall, extending one leg behind you, one leg in front of you, stretch the leg, and reverse.

C. Pharmaceutical therapy:

1. Nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen (Motrin) 800 mg TID or naproxen (Naprosyn) 500 mg PO BID for comfort.

2. Cortisone injections may be attempted for severe cases. A corticosteroid lidocaine 1 to 1.5 mL injected directly into the most tender area on the sole of the foot may be helpful.

FOLLOW-UP

A. Recommend follow-up in 4 weeks following treatment. Pain should slowly improve with aggressive treatment management. The client must be compliant with instructions given for improvement. May take 6 to 12 months for complete resolution.

B. If pain worsens, consider diagnostic workup (x-ray or MRI).

C. Use caution with steroid injections. Multiple injections can cause the plantar fascia to rupture, resulting in flat foot and chronic pain.

CONSULTATION/REFERRAL

A. Refer to a podiatrist if conservative treatment therapy fails after 6 weeks.

B. Recommend physical therapy to stretch calf muscles and plantar fascia, using specialized ice treatments and/or massage.

C. Surgical treatment: Only after 12 months of aggressive nonsurgical treatments should surgical treatment be considered. Possible surgery may be recommended to lengthen the calf muscle (gastrocnemius) or to have a plantar fascia release procedure whereby the ligament is partially cut to relieve tension in the tissue. Large bone spurs may be surgically removed with open incision if indicated.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Caution when using NSAIDs in the geriatric population due to gastrointestinal or kidney adverse effects.

2. Fall precautions should be discussed when having severe pain and difficulty walking.

3. Evaluate support systems for transportation to physical therapy if client is unable to drive.

4. Foot health directly affects the ability to retain ambulation as we age. Plantar fasciitis is one of the most common orthopedic complaints. Eighty percent of plantar fasciitis pain and inflammation will resolve spontaneously in the older adults.

5. Common causes of plantar fasciitis in elderly clients include flat feet, overuse or weight-bearing on toes/balls of feet, rheumatic disease, sedentary lifestyle, obesity, and bone spurs on toes/feet.

6. Exercises to alleviate fasciitis pain: calf-plantar stretch, foot–ankle circles, and toe curls.

7. Suggest proper-fitting arch support. Encourage wearing new shoes with arch support or inserting silicone heel inserts into the shoe.

8. Encourage preventive measures of avoiding walking barefoot or wearing house shoes/slippers (slip-on or clog type).

9. Lidocaine patches or short-term steroids/NSAIDs may be effective in controlling plantar fasciitis and bone spur pain. Surgery is not recommended until nonoperative measures have been implemented for a minimum of 6 months and outcomes were unsuccessful.

10. Aim for interventions that maximize independence and quality of life.

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SCIATICA

DEFINITION

A. Sciatica is a sharp or burning pain, usually associated with numbness, that radiates down the posterior or lateral leg that can result in neurosensory and/or motor deficits. Sciatica indicates abnormal function of the lumbosacral nerve roots or one of the nerves in the lumbosacral plexus.

INCIDENCE

A. The prevalence of sciatica in the general population is 40%, although only 1% have any neurosensory or motor deficits. Around 85% of cases of sciatica are caused by herniated discs, the vast majority of which occur at the L4 to L5 or L5 to S1 level.

PATHOGENESIS

A. Pressure on the nerve from a herniated disc, bony osteophytes, a compression fracture, or any other extrinsic pressure (e.g., pelvic mass or epidural process, "wallet sciatica") causes progressive sensory, sensorimotor, or sensorimotor visceral loss. Disturbances along anywhere on the length of the sciatic nerve can give rise to sciatica, but the nerve roots close to the diseased discs and spinal joints are the most common sites. Typically, sciatica affects only one side of the body.

PREDISPOSING FACTORS

A. Inflexibility.
B. Obesity.
C. Trauma.
D. Bony osteophytes.

COMMON COMPLAINTS

A. Pain around the buttock area.
B. Pain often associated with numbness traveling down the lateral or posterior leg.
C. Numbness.
D. Paresthesia.

OTHER SIGNS AND SYMPTOMS

A. Difficulty walking with affected leg.
B. Positive straight leg raise test, positive Patrick sign (flexion, abduction, and external rotation of the hip, or FABER).
C. Decreased sensation.

SUBJECTIVE DATA

A. Elicit onset of symptoms, duration, and what makes pain better or worse.
B. Inquire about previous episodes of pain or trauma.
C. Have the client point to the area of pain, numbness, or tingling.
D. Are symptoms unilateral or bilateral?
E. Question the client about loss of bowel or bladder control or other deficits and/or changes.
F. Does the client notice leg weakness or difficulty walking?

PHYSICAL EXAMINATION

A. Inspect:
  1. Inspect gait and movement of back and extremities.
B. Palpate:
  1. Palpate spinous processes. Percuss for tenderness over the spinous processes.
  2. Examine flexion and extension of spine.
C. Neurologic exam:
  1. Examine neurologic function of back and lower extremity.
    a. Straight leg raising sign is positive.
    b. Dorsiflexion of ankle is positive.
    c. Check for loss of sensation in radicular pattern. Light-touch pinprick and two-point discrimination are not present.
    d. Look for decrease or loss of deep tendon reflexes (DTRs).
    e. Check muscle strength of lower extremities.
    f. Assess DTRs.

Cauda equina syndrome is a surgical emergency characterized by bowel and bladder dysfunction; saddle anesthesia at the anus, perineum, or genitals; and widespread or progressive loss of strength in the legs or gait disturbances.

DIAGNOSTIC TESTS

A. Radiography, when red flags for fracture, cancer, or infection are present.
B. CT scan or MRI, when cauda equina, tumor, infection, or fracture is suspected; MRI is test of choice for clients with prior back surgery.

DIFFERENTIAL DIAGNOSES

A. Sciatica of unknown etiology.
B. Lumbosacral strain.
C. Herniated disc.
D. Bony osteophytes, spinal stenosis.
E. Compression fracture.
F. Neoplasm of spine.
G. Pelvic mass.
H. Epidural process causing progressive sensory, sensorimotor, or sensorimotor visceral loss.
I. Meralgia paresthetica.

PLAN

A. General interventions: Care for these clients should evolve over a three-step process:
  1. Step 1 (2–4 days):
    a. Activity is limited by degree of pain, and although rest is recommended it is not superior to activity in clients who are able to remain active.
    b. Recommend application of heat or cold packs to site as needed.
  2. Step 2 (7–14 days):
    a. Reevaluate neurologic and back exam; tell the client "Let pain be your guide" when resuming normal daily activities.
    b. Have the client perform gentle stretching exercises.
    c. Encourage walking on flat surfaces.
    d. Educate the client regarding proper care of the back, with regard to exercises, posture, and so forth.
    e. Provide handouts on back exercises/stretches for the client. *See Client Teaching Guide for this chapter, "Back Stretches."*
    f. Physical therapy may be implemented at this time if no significant improvement is noted.
  3. Step 3 (2–3 weeks):
    a. Reevaluate the client, noting degree of improvement with exam.
    b. Continue muscle toning and reconditioning exercises.
    c. If improvement is noted, gradually increase physical activities.
    d. Reinforce healthy care of the back. ▶

e. Continue physical therapy until the client can perform exercises without assistance or until released by the physical therapist.

B. Pharmaceutical therapy:

1. Nonsteroidal anti-inflammatory drugs (NSAIDs) as needed: naproxen (Naprosyn) 500 mg initially, followed by 250 mg every 6 to 8 hours.

2. Acetaminophen may also be used as needed, especially if the client is not able to tolerate NSAIDs.

3. For more severe pain not relieved by NSAIDs, consider acetaminophen (Tylenol) with codeine for short duration. Narcotics should not be used for more than 2 weeks.

4. Muscle relaxants should not be used for more than 2 weeks:

a. Cyclobenzaprine (Flexeril) 10 mg one to three times daily.

b. Muscle relaxants place clients at risk for drowsiness. Warn the client not to mix medications with alcohol because it may potentiate the medication.

FOLLOW-UP

A. Initial follow-up is needed in 1 to 2 weeks.

CONSULTATION/REFERRAL

A. If cauda equina syndrome is suspected, prompt referral to a physician and/or ED is necessary.

B. If bilateral sciatica is associated with vertebral collapse, osteoporosis, neoplasia, and/or vascular disease, consult with a physician.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Sciatica pain is common due to physiologic changes of the pelvis as pregnancy progresses to term. Avoid use of NSAIDs. Physical therapy may be used as indicated.

B. Adults:

1. For adults older than 50 years presenting with no history of backache, consider differential diagnosis of neoplasm. The most common metastasis is secondary to the primary site of breast or prostate, or to multiple myeloma. Pain is most prominent in recumbent position and rarely radiates into buttock or leg.

C. Geriatrics:

1. Bilateral sciatica is associated with vertebral collapse, osteoporosis, neoplasia, and/or vascular disease. Refer the client to a physician immediately.

2. Use caution when prescribing medication to the elderly due to the risk for drowsiness and potential falls.

3. Implement education of proper body mechanics during activities of daily living (ADLs) and exercise. Avoid heavy lifting, twisting, or prolonged sitting/standing.

4. Exercises to prevent recurrences and spinal health (sciatica prevention) include back flexion stretches, lower back stretch, calf stretch, and low-impact exercise. See www.livestrong.com/article/333512-spine-stretch-and-sciatica-exercises-for-the-elderly/ for a full description of these age-friendly exercises.

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SHOULDER PAIN

DEFINITION

A. The shoulder is the most mobile joint in the body. The shoulder can be moved in all three planes via abduction/adduction, internal/external rotation, and flexion/extension, and can be moved through a full 360° plane. The shoulder has a large degree of range of motion (ROM), but it comes with the cost as the shoulder is more prone to injury than any other joint.

B. Anatomy of the shoulder:

1. The shoulder joint is made up of bones held in place by tendons, ligaments, and muscles.

2. The shoulder joint has three joints:

a. Glenohumeral joint (the main joint of the shoulder).

b. Acromioclavicular joint.

c. Sternoclavicular joint.

3. Unlike other joints, all joints work together to permit a universal motion of the shoulder.

4. Types of cartilage in the shoulder:

a. White cartilage is present on the ends of the bones and allows the bones to glide and move on each other (articular cartilage).

b. Labrum cartilage is fibrous cartilage found only around the shoulder socket and acts as a static stabilizer of the shoulder.

5. Multiple muscles are responsible for movement in the shoulder. These muscles attach to the scapula, humerus, and clavicle, forming the shoulder cap and underarm. These muscles include the following:

a. Serratus anterior.

b. Subclavius.

c. Pectoralis minor.

d. Sternocleidomastoid.

e. Levator scapulae.

f. Rhomboid major and minor.

g. Trapezius.

h. Deltoid.

INCIDENCE

A. According to the Centers for Disease Control and Prevention, in 2010 nearly 1.2 million people in the United States visited an ED for shoulder problems.

B. A community survey of 644 elderly people over the age of 70 (318 males and 326 females) revealed the prevalence of shoulder disorders as being approximately 21%.

C. Shoulder disorders were more common in females (25% vs. 17%).

D. 70% of the cases involved the rotator cuff.

E. Fewer than 40% of the subjects sought medical attention for their symptoms.

F. Studies confirm that shoulder pain is a chronic problem that interferes with daily care and activities for the elderly population.

PATHOGENESIS

A. The shoulder is an unstable joint due to the joint's ROM, which increases the likelihood of joint injury and degenerative changes. Major injuries to the shoulder include rotator cuff tears and bone fractures involving the clavicle, scapula, and proximal humerus. ▶

B. The most common shoulder problems are dislocation, separation, rotator cuff disease, rotator cuff tear, frozen shoulder, fracture, and arthritis.

PREDISPOSING FACTORS

A. Overuse of the shoulder and/or frequent overhead reaching.
B. Manual labor.
C. Sports.
D. Injury.
E. Arthritis.
F. Tendon inflammation or tear.
G. Fracture.
H. Instability.

COMMON COMPLAINTS

A. Pain on movement of the shoulder or arm.
B. Limitation of movement of the shoulder.
C. Stiffness of the shoulder.

OTHER SIGNS AND SYMPTOMS

A. Swelling of the shoulder.
B. Difficulty lifting the arm and getting dressed.
C. Difficulty with routine activities of daily living (ADLs).

POTENTIAL COMPLICATIONS

A. Fracture.
B. Gallbladder disease.
C. Heart disease/myocardial infarction (MI).
D. Disease of the cervical spine.
E. Tumors.
F. Infection.
G. Nerve-related disorders.

SUBJECTIVE DATA

A. Ask client what activity brought about or preceded the episode. Any falls? Any direct blow injuries?
B. Inquire about the duration of pain, what makes it worse, and what makes it better.
C. Ask client to describe the pain, for example, dull, aching, or burning. Any numbness or tingling?
D. Does pain radiate to other areas of the back, arm, or neck?
E. Have client rate pain on a scale of 0 to 10, with 0 being no pain.
F. Note a list of all medications currently being taken, particularly any over-the-counter (OTC) preparations, rubs/creams, splints/sling, or electrical stimulation devices.

PHYSICAL EXAMINATION

A. Inspect:
1. Always compare each area bilaterally for comparison and contour of anatomy.
2. Inspect general appearance and gait, noting evenness and symmetry of motion, as well as swing arm in tandem with opposite lower extremity.
3. Observe shoulder movement as the client disrobes: Normal is smooth, natural, and bilateral. Abnormal findings are jerkiness and client's attempt to move to reduce pain.
4. Inspect skin for discoloration, abrasions, scars, or evidence of previous pathology.
5. Inspect the clavicle for distortion or abnormality, possibly indicating a fractured clavicle.
6. Inspect the deltoid portion of the shoulder, which should be rounded as a result of the draping of the deltoid muscle over the greater tuberosity of the humerus. Normally, the area is full and round and the two sides are symmetrical. Inspect for atrophy or asymmetry, possibly representing a dislocation.
7. Inspect the posterior shoulder, the scapula. In the resting position, the scapula is positioned over ribs 2 to 7 and medial border is approximately 2 in. from the spinous processes. The triangular area of the scapula is opposite T3. Inspect for winged effect, which may indicate weakness or atrophy of the serratus anterior muscle. Scapular asymmetry also occurs when the scapula has only partially descended from the neck to the thorax, causing a webbing or shortening of the neck (Sprengel deformity).
B. Auscultate:
1. Auscultate carotid arteries.
2. Auscultate abdomen.
3. Conduct a complete heart exam and lung exam.
C. Palpate: (The provider should position self behind the seated client with cupped hands on the deltoid and acromion.)
1. Palpate the suprasternal notch and the joint that holds the clavicle in place.
2. Palpate in a sliding motion the anterior superior surface of the clavicle.
3. Palpate the acromioclavicular joint by pushing in a medial direction against the distal end of the clavicle. Then ask the client to flex the shoulder until the arm is straight up, then extend the arm back down. Movement of the joint will be palpated under the fingers. Note any crepitation, which may indicate osteoarthritis or dislocation of the lateral end of the clavicle.
4. Palpate the acromion and the greater tuberosity of the humerus.
5. Palpate the bicipital groove, which is located anterior and medial to the greater tuberosity. It is more easily palpable if the arm is externally rotated, providing for a more exposed position for palpation. This requires gentle palpation because the long head of the biceps lies within it, and too much pressure may elicit pain, causing difficulty with further evaluation.
6. Palpate the scapula. It is clinically important to palpate this area, for it is frequently a site of referred pain from the cervical spine.
7. Palpate the soft tissues of the shoulder, which is divided into four clinical zones:
a. Rotator cuff (the supraspinatus, infraspinatus, subscapularis, and the teres minor muscles). Note tenderness during palpation. This may be due to defects, tears, or tendon detachment. The supraspinatus muscle is the most commonly ruptured muscle, especially near its insertion. Passive extension of the shoulder moves the rotator cuff into a palpable position. Hold the client's arm and lift the elbow posteriorly.
b. Subacromial and subdeltoid bursa can be very tender if bursitis is present. Palpate for thickening, masses, specific tenderness, or crepitation.
c. Axilla: Abduct the arm with one hand as you gently insert your index and middle fingers into the axilla. Then return the client's arm to the side to relax the skin; your fingers will be able to penetrate higher into the axilla. Palpate for lymph node enlargement or tenderness. Palpate the anterior wall, medial wall, and posterior walls of the axilla.
d. Prominent muscles of the shoulder girdle: Palpate the muscles bilaterally for size, shape, consistency, ▶

and tone. Palpate the sternocleidomastoid, pectoralis major, bicep, deltoid, trapezius, rhomboid minor and major, latissimus dorsi, and serratus anterior.

8. Evaluate the ROM, both active and passive. The shoulder girdle involves six motions: abduction, adduction, extension, flexion, internal rotation, and external rotation.

9. Among many tests described to diagnose shoulder pain, two useful tests to help to diagnose a rotator cuff tear are the following:

a. The empty can (Jobe) test is sensitive and specific in detecting a supraspinatus tear. Elevate both arms to 90° and angled forward 30° to place the humerus in line with the scapula. The shoulder is internally rotated by pointing the thumb down to the ground. The clinician applies a downward force as the client attempts to resist. Pain and/or weakness are deemed a positive test.

b. Drop arm test. The arm is abducted to at least 90°. Lower the arm slowly and control the descent. The inability to perform this task slowly and with control is a positive test. The client may still have a tear even if this test is negative.

D. Mental status:

1. Assess for confusion.

DIAGNOSTIC TESTS

A. Plain x-rays of the shoulder will show any injuries or fractures to the bones.

B. MRI and/or ultrasound create a better picture of soft tissues, identifying injuries to the ligaments and tendons.

C. Arthrogram: A dye is injected into the shoulder to visualize the joint, muscles, and tendons.

D. Arthroscopy is a surgical procedure that uses a fiber-optic camera to inspect the joint. This procedure may show injuries that may not be apparent on exam or other tests. Sometimes arthroscopy may be used to correct the problem.

E. Electromyography (EMG) can be used to evaluate nerve function, if indicated.

DIFFERENTIAL DIAGNOSES

A. Shoulder pain.

B. Disease of the cervical spine.

C. Heart disease.

D. Gallbladder disease.

E. Liver disease.

PLAN

A. General interventions:

1. Educate the client regarding proper treatment and care for the injured shoulder.

B. Client teaching:

1. Educate the client on rest, and altering activities to avoid overexertion or overdoing activities to prevent shoulder pain.

2. Educate the client on sling use, if indicated.

3. Educate the client on RICE: Rest, Ice, Compression, and Elevation.

4. Educate the client on ROM and strengthening exercises.

C. Pharmaceutical therapy:

1. Nonsteroidal anti-inflammatory drugs (NSAIDs) or aspirin as indicated. Consider kidney function and other chronic conditions.

2. Corticosteroid injection may be considered if shoulder condition does not improve.

3. Oral prednisone therapy to reduce inflammation, short term only.

4. Cautionary use of opioid analgesics for pain.

FOLLOW-UP

A. Approximately 23% of all new episodes of shoulder pain completely resolve within 1 month and 44% within 3 months of onset.

B. Approximately 50% of clients with an acute shoulder who are treated conservatively recover within 5 months, and approximately 60% recover within 12 months.

C. The client's personality traits, coping styles, and occupational factors may determine otherwise.

D. Follow-up should be in 1 to 2 weeks to reevaluate shoulder function, pain status, and impact of daily life abilities.

CONSULTATION/REFERRAL

A. Acute fracture or dislocation requires immediate referral to orthopedic surgeons. Urgent referral as needed for any red flag diagnoses.

B. Referral to orthopedic surgeon if there are positive imaging findings or if surgical interventions are needed.

C. Referral to physical therapy (PT) or physiotherapist for exercise, strengthening, strapping, immobility devices, or ultrasound therapy, as well as prevention of further injuries.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Soft tissue lesions are a prevalent cause of shoulder pain within the geriatric population. These include rotator cuff tendinitis/rupture, frozen shoulder, acromioclavicular joint osteoarthritis, and subacromial impingement. Studies indicate that geriatric clients suffering with shoulder-related pain experience an impact on their functional level and quality of life.

2. Consider acute versus chronic shoulder interventions. Current recommendations are home exercises with a PT and a focus on assisted "wall walking" and ROM.

3. NSAID use is dependent on comorbid diseases such as gastrointestinal, cardiac, or renal disease.

4. Cautionary use of opioid analgesics due to fall risk. If using opioid analgesics, educate the client regarding fall risk prevention.

5. Need for ADL support to avoid injury. Special attention is given to clients using mobility devices requiring use of the affected shoulder.

6. Consider fluoroquinolone-associated tendinopathy, including tendon rupture, in clients who complain of shoulder pain after the use of fluoroquinolone antibiotics (ofloxacin, ciprofloxacin, levofloxacin). Perform an MRI for assessment/evaluation. Refer to orthopedics as indicated.

7. In cases of frozen shoulder with geriatrics, avoid rest and immobilization and begin gentle home exercises that include stretching the arm in external/internal rotation, flexion, and horizontal adduction. Transition to PT once mobility improves. Steroid injections are recommended to reduce pain and allow aggressive PT. However, if no response after 6 to 12 months, consider surgical correction.

RESOURCE

Exercise: A Guide from the National Institute on Aging. https://go4life.nia.nih.gov/exercise/shoulder

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SPINAL STENOSIS

DEFINITION

A. Spinal stenosis is a narrowing of the spinal canal that results in pressure on the spinal cord and/or the nerve roots. The narrowing may involve a small or large area of the spine. In the lumbar area, pressure may elicit pain or numbness in the legs. In the cervical area, it may produce pain or numbness in the shoulders and arms/hands. The stenosis occurs in up to three areas:

1. The canal in the center of the column where the nerve roots run.

2. The canals at the base of the nerves branching out from the spine.

3. The openings of the vertebrae through which the nerves pass and go to other areas of the body.

INCIDENCE

A. Spinal stenosis is most common in people older than the age of 50.

B. The incidence of lumbar spinal stenosis in the United States has been estimated at 8% to 11% of the population, and the rate at which people are undergoing lumbar stenosis surgery is increasing. Lumbar stenosis accounts for 75% of the cases.

PATHOGENESIS

A. Spine:

1. The spine consists of 26 bones that extend in a line from the base of the skull to the pelvis. Twenty-four of those bones are called vertebrae:

a. Seven cervical vertebrae.

b. Twelve thoracic vertebrae.

c. Five lumbar vertebrae.

2. The sacrum consists of five fused vertebrae between the hip bones.

3. The coccyx has three to five bones at the lower tip of the vertebral column, which are linked together and cushioned by shock-absorbing discs.

B. Narrowing of the spinal canal may be inherited or acquired. Spinal stenosis most often results from a gradual, degenerative aging process where the ligaments of the spine may thicken and calcify. The bones and joints enlarge where surfaces of the bone begin to project from the body (called osteophytes or bone spurs), thereby decreasing the space (neural foramen) for nerve roots leaving the spine. Disc degeneration can cause similar narrowing of the vertebral space as the soft material from the inside of the disc protrudes and presses on the spinal cord and/or nerve roots.

PREDISPOSING FACTORS

A. Aging with secondary calcification changes (narrowing of the spinal canal).

B. Osteoarthritis (the most common cause of spinal stenosis).

C. Herniated disc (places pressure on the spinal cord and nerve root).

D. Trauma to the spine.

E. Bone disease such as Paget disease of the bone (may see structural narrowing of the spinal canal).

F. Tumors of the spine.

G. Ossification occurs when calcium deposits form, turning the fibrous tissues of the ligament into bone and in turn pressing on the nerves of the spinal canal.

H. Rheumatoid arthritis.

I. Spinal surgery.

COMMON COMPLAINTS

A. The neck or back may not be painful, but if the narrowed space is pushing on a nerve root, symptoms will associate with the nerve root in the arms or legs.

1. Numbness in arms or legs.

2. Weakness in arms or legs.

3. Cramping in arms or legs.

4. Pain radiating down the leg (sciatica), causing difficulty in walking.

OTHER SIGNS AND SYMPTOMS

A. Symptoms develop slowly over time.

B. Pain comes and goes, generally not continuous.

C. Pain worsens during certain activities such as walking or change of position.

D. Some symptom relief by rest and/or any flexed forward position (lumbar).

POTENTIAL COMPLICATIONS

A. Incontinence.

B. Paralysis.

C. Unresolved presenting symptoms or worsening of symptoms.

D. Surgical risks include infection, tear in the membrane covering the spinal cord, blood clot formation, and neurologic deterioration.

E. Lumbar spinal stenosis can dramatically affect quality of life, especially in the elderly.

SUBJECTIVE DATA

A. When were the symptoms first noticed? Ask client what brought about or preceded the episode.

B. Have the symptoms worsened over time? Describe the pain and note certain times of the day that symptoms were more prominent.

C. Describe pain, as well as radiation of pain, or effect (numbness or tingling) on other body parts: hips, thighs, knees, calves, ankles, feet, arms, hands, fingers.

D. What makes the pain worse? What makes the pain better?

E. Have the client rate pain on a scale of 0 to 10, with 0 being no pain.

F. Ask the client to list all medications currently being taken, particularly substances not prescribed and illicit drugs.

G. Are there any other medical problems or chronic conditions that may contribute to symptoms?

H. Any incontinence issues with bowel or bladder? If so, when did symptoms begin and how are they changing?

PHYSICAL EXAMINATION

A. Check vital signs: temperature, pulse, respirations, and blood pressure.

B. Inspect:

1. Inspect and observe general appearance, gait, assistive devices used, positioning of affected portion of the spine, and overall appearance of pain.

2. Inspect for gross abnormalities of weakness of arms or legs, foot drop or drag.

3. Inspect legs for edema/swelling, skin rashes or discolorations of skin of arms/hands or leg/feet; skin temperature changes or symptoms of stasis; and fatty masses (lipomas) in the area of the low back, which may be a sign of spina bifida.

C. Auscultate:

1. Heart.

2. Lungs.

D. Palpate: cervical spine and lumbar spine.

1. Neck:

a. Palpate the neck with the client in the supine position; the muscles of the neck are relaxed in this position and the bony structures are more defined.

b. Palpate the hyoid bone (situated above the thyroid cartilage), which is opposite the C3 vertebral body.

c. Palpate the "Adam's apple," which marks the C4 body, whereas the lower portion of the Adam's apple is the C5 level. The first cricoid ring, inferior to the sharp lower edge of the thyroid, is opposite the C6 body. This ring is immediately above the site for an emergency tracheostomy. Too much pressure on palpation may cause the client to gag. The carotid tubercles are at the horizontal level of C6 and located laterally about 1 in. from the first cricoid ring. The carotid tubercles should be palpated separately to avoid restriction of flow to both carotid arteries at the same time. C1 can be located as a small hard bump which lies between the angle of the jaw and the styloid process.

d. Palpate the posterior neck while standing behind the client, cupping the anterior neck to avoid tension on the muscles. The posterior aspect includes the occiput, spinous processes, mastoid process, and the facet joints. Begin at the base of the skull, palpating C2 (C1 lies deep). Palpate from C2 to T1 for normal curvature of the spine. If the vertebra is uncertain, it can be determined with the anterior structures of the anterior neck.

e. Palpate the lumbar spine while sitting on a stool behind the standing client. Place the fingers on the top of the iliac crest with the thumbs on the midline of the back (L4–L5) and palpate spinous processes downward. Pain referred from the spine may be reproduced in the back of the legs during palpation. Pain with palpation of the coccyx is usually the result of direct blow trauma. Palpate the anterior aspect of the spine, with the client lying supine with knees bent to relax the abdominal muscles. The umbilicus lies at L3 to L4. L4 to S1 are palpable below the aorta's division into the common iliacs. L5 to S1 may be palpated when palpating below the umbilicus and pushing into the abdomen with abdominal relaxation.

f. Palpate the soft tissues of the neck and lumbar spine:

i. The anterior triangle: This is made up of the two sternocleidomastoid muscles, superior by the mandible and inferior by the suprasternal notch.

ii. The posterior aspect involves the trapezius muscles and the greater occipital nerves at the base of the skull, as well as the superior nuchal ligament that extends to the C7 process.

iii. Soft tissue palpation of the lumbar spine involves the following:

1) The midline raphe (supraspinous and interspinous ligaments and the paraspinal muscles).

2) The iliac crest (gluteal muscles).

3) The posterior superior iliac spine (sacral triangle).

4) The sciatic area (sciatic nerve: the largest nerve in the body runs vertically down the midline of the posterior thigh, giving off branches to the hamstring muscles, and then divides into the tibial and peroneal areas).

5) The anterior abdominal wall and inguinal areas. Pain in the inguinal area usually indicates disorders of the hip joint.

g. Assess the range of motion (ROM) of the cervical and lumbar spine with basic movements of flexion, extension, lateral rotation to the left and right, and lateral bending to the left and right:

h. Rotation: Ask the client to shake their head from side to side.

i. Flexion and extension: Ask the client to touch their chin to their chest and to look up at the ceiling.

j. Lateral bending is assessed by having the client try to touch their ear to their shoulder without lifting the shoulder to their ear.

k. Normal lateral bending is 45° toward each shoulder.

l. Vertebral motion is greatest where the disc is the thickest and larger joint surfaces are present (L4, L5, S1), so motion taking place is greater than between L1 and L2.

m. ROM of the lumbar spine includes flexion (bend as far forward as client can with knees straight and try to touch toes—measure the distance from floor to fingertips), extension (place palm on their back and have client bend backward as far as possible—estimate the range of extension), lateral bending (lean to the left and then lean to the right as far as possible—estimate the degree and compare sides), and rotation (rotate the pelvis and compare ranges of rotation).

n. Perform a neurologic exam that includes muscle testing and examination of the entire upper extremities for weakness or disorders of sensation. There are eight nerves exiting the cervical spine. Stenosis or disorders such as a herniated disc frequently reflected to the upper extremity usually originate from the C5 to T1. The primary nerves involved in the sensory distribution of the upper extremities are the following:

i. C5: lateral arm, axillary nerve.

ii. C6: lateral forearm, thumb, index, and half of middle finger, musculoskeletal nerve.

iii. C7: middle finger.

iv. C8: ring and little finger, medial forearm, medial antebrachial-cutaneous nerve.

v. T1: medial arm, medial brachial cutaneous nerve.

The neurologic exam of the lumbar spine is completed by performing an assessment of the ROM.

o. Assess reflexes and sensation: Biceps reflex and lateral arm axillary nerve indicate primarily origination from C5 to C6:

i. Assess C6 by the brachioradialis reflex tested proximal to the wrist. C6 supplies sensation to the ▶

lateral forearm, the thumb, the index, and half of the middle finger. To assess, form the number six with your thumb, index, and middle finger by pinching your thumb and index finger and extending the middle finger.

ii. Access C7 (radial nerve) by having the client begin extension of the tricep/elbow from a position of flexion as motion is resisted. C7 also involves the median and ulnar nerves. To test wrist flexion, ask the client to make a fist and flex the wrist as you resist against the palmar aspect of the closed fist. To test the radial nerve (C7), press on the dorsum of the client's extended fingers. To test the reflex of C7, tap the triceps tendon where it crosses the olecranon fossa at the elbow. Sensation testing is supplied to the middle finger by C7.

iii. C8 has no reflex, muscle strength, or sensation test to determine integrity.

iv. To test nerves emanating from T12, L1, L2, and L3—the iliopsoas is the main flexor of the hip—have the client sit on a table, place hand over the iliac crest, and ask them to actively raise their thigh from the table.

v. Sensation testing of L1, L2, and L3 is over the area of the anterior thigh between the inguinal ligament and the knee.

vi. Muscle testing of L2, L3, and L4—femoral nerve—is performed by having the client sit on the edge of the table, stabilizing the distal end of the thigh, and instructing them to extend their knee while offering resistance to the motion.

vii. To test the obturator nerve (hip adductors), instruct the client to abduct the legs after placing a hand on the medial sides of the knee and ask to abduct knees to resistance.

viii. L4 muscle testing involves the tibialis anterior tested by resistance to dorsiflexion and inversion by pushing against the dorsal medial aspect of the first metatarsal bone.

ix. Reflex testing of L4 involves the patellar reflex and sensation testing of the medial side of the leg below the knee.

x. Neurologic muscle testing of L5 involves the deep peroneal nerve by placing your thumb on the dorsal surface of the foot so that the client must dorsiflex their great toe to reach it. Oppose this motion by pushing on the nail bed of the great toe. To test the superior gluteal nerve (L5), have the client lie on their side and instruct them to abduct the leg and then push against the lateral side of the thigh for resistance. For reflex testing of L5, test the tibialis posterior reflex.

xi. Muscle testing of S1 involves the superficial peroneal nerve and is performed by securing the client's ankle and having them plantar flex and evert the foot and oppose the motion by resistance pushing. The inferior gluteal nerve is tested by having the client flex the knees and extend the hips as resistance is applied to hip extension while palpating the gluteus maximus for tone.

xii. The Achilles tendon reflex is the tendon reflex mediated by the S1 and S2 tibial nerve. Sensation of the S2 dermatomes includes the lateral malleolus and lateral side and plantar aspect of the foot. S2, S3, and S4 are the principal nerves of the bladder and supply the intrinsic muscles of the foot. There is no deep reflex for S2, S3, and S4. The dermatomes around the anus receive innervation from S2, S3, and S4. The abdominal (lie supine and stroke each quadrant of the abdomen with the sharp edge of a reflex hammer), cremasteric (stroke the inner side of the upper thigh with the sharp end of the reflex hammer), and anal reflex (simply touch the perianal skin, causing contraction as response) are superficial and require skin stimulation.

xiii. Pathologic reflexes (the significance of their presence or absence is the reverse of the normal reflexes). These include the following:

1) Babinski test: Run a sharp instrument across the plantar surface of the foot from the calcaneus to forefoot. If negative, the toes either do not move or bunch up uniformly. If positive, the great toe extends while the other toes plantar flex and splay. Results should be negative in adults.

2) Hoffman test: Instruct the client to pronate the hand so that the palm is facing the ground. Place the client's middle finger between the clinician's index and middle fingers from the side at the level of the client's distal interphalangeal joint, so that the clinician's fingers are pointing toward the client's thumb. Using the thumb, the clinician then should flick the client's distal phalanx in a downward fashion. A positive (abnormal) test has occurred when the client's thumb adducts toward the palm, while a negative (normal) test has occurred when there is no movement of the thumb.

3) Oppenheim test: Run your fingernail along the crest of the tibia. Normally, there should be either no reaction, or the client should complain of pain; if abnormal, the great toe extends while the other toes plantar flex and splay.

p. Perform complete abdominal exam.

E. Mental status:

1. Assess for any psychopathologic component impacting symptomology and diagnosis.

DIAGNOSTIC TESTS

A. Diagnostic imaging: MRI scan or a CT scan with and without myelogram. Plain CT scans are of limited value unless they are made with very fine segmental scan slices.

B. Bone density or dual-energy x-ray absorptiometry (DEXA) scan testing is used to assess for osteopenia and/or osteoporosis.

C. Electrophysiologic studies are mainly used to rule out differential diagnosis of spinal cord lesions or disorders of the nervous system, but may be helpful when the client presents with multisegmental symptoms. These studies are often complicated with coexisting conditions such as diabetes mellitus or polyneuropathy affecting the nerves.

D. Laboratory tests can help to rule out inflammatory processes: complete blood count (CBC), C-reactive protein (CRP), renal function test, basic metabolic panel (BMP), and erythrocyte sedimentation rate.

DIFFERENTIAL DIAGNOSES

A. Spinal stenosis.

B. Conditions that may mimic cauda equina (ruptured abdominal aortic or iliac aneurysms, acute aortic dissection, acute leg ischemia, or deep vein thrombosis [DVT]).

C. Claudication (presentation of intermittent leg pain may indicate claudication: arterial, peripheral, or neurogenic).

D. Diabetic polyneuropathy.

E. Paget disease:

1. Paget disease is a chronic disorder typically resulting in enlarged and abnormal bones; the spine is one of the most common sites affected by this disease. The lumbar spine is affected in at least half of the cases. Paget disease may present symptoms of disc prolapse with nerve root entrapment and with the increased vascularity of the pagetic vertebrae, which may diminish the spinal cord or nerve root blood supply; this causes pain.

F. Amyloidosis associated with hemodialysis or amyloid tumors may cause spinal stenosis or even cauda equina syndrome.

PLAN

A. General interventions:

1. Cauda equina syndrome is a rare form of spinal stenosis. It occurs due to the compression of the cauda equina, and symptoms may include loss of bowel control, bladder or sexual dysfunction, pain, or weakness or loss of feeling in one or both legs. Cauda equina syndrome is serious and requires urgent medical attention. Clients should be transported to the hospital immediately.

2. Spinal stenosis of the cervical spine can be far more dangerous by compressing the spinal cord, leading to major body weakness or even paralysis, and requires urgent medical attention. Clients should be transported to the hospital immediately.

3. Physical therapy:

a. Build strength and endurance.

b. Maintain flexibility and stability of spine.

c. Improve balance and gait.

d. Reduce pain.

4. Alternative therapies such as acupuncture.

5. Bracing may be used for lumbar stenosis to help support clients with weak abdominal muscles or multilevel degeneration and also help regain or maintain stability.

B. Client teaching:

1. Home treatments may include hot or cold packs as necessary.

2. Assistive ambulatory devices such as canes or walkers may be used to relieve pain and provide stability and fall prevention. Instruct the client on the proper use of these devices.

3. Encourage the use of home exercises. Provide the client with proper back exercises.

C. Pharmaceutical therapy:

1. Pain control:

a. Nonsteroidal anti-inflammatory drugs (NSAIDs) if not contraindicated.

b. Muscle relaxants to treat the muscle spasms associated with spinal stenosis.

c. Antidepressants such as amitriptyline can help ease chronic pain.

d. Antiseizure medications such as gabapentin (Neurontin) or pregabalin (Lyrica) may be used to reduce pain caused by damaged nerves.

e. Opioids may be considered to control severe pain. All clients must be assessed individually for comorbid conditions, as well as medication interactions and precautions, before prescribing medications. Fall risk assessment; independence of activities, driving; and mental capabilities should also be evaluated.

2. Epidural steroid injections may reduce pain and increase function in the short term for clients with spinal stenosis with or without radiculopathy. Repeated steroid injections can weaken bones and connective tissue, so injections should be limited.

FOLLOW-UP

A. Follow-up is determined by the client's needs: complications from surgery, continued pain and inflammation, or assessment of functional status with nonsurgical therapy interventions.

CONSULTATION/REFERRAL

A. Refer to a neurosurgeon if nonsurgical modalities fail. The extent of the client's pain and the client's preferences all factor into whether to have surgery.

B. Urgent surgical consult should be obtained immediately if the client has numbness or weakness that interferes with walking, impaired bowel or bladder function, or other neurologic involvement.

C. Pain management specialists should be considered if conservative measures are not meeting client's needs.

D. Refer to physical/occupational therapy for evaluation and treatment.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. When the elderly client can no longer walk sufficiently to care for themselves and perform essential tasks, then surgery is recommended. The priority of surgery is to increase the client's activity tolerance with less pain.

2. Booklet for exercise: A guide from the National Institute on Aging is available free of charge to educate clients on exercises-motivation-safety, along with self-tests, benefits, and nutrition. This booklet can be obtained from NIA Information Center, PO Box 8057, Gaithersburg, MD 20898-8057.

3. Nonpharmacologic measures to treat spinal stenosis in the adult and elderly population are becoming more acceptable for conservative spinal pain management among clients and their medical providers. Recent studies postulated that according to a satisfaction score, acupuncture for spinal stenosis pain was significantly more effective than physical exercises and medications.

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SPRAINS: ANKLE AND KNEE

DEFINITION

A. Sprains are ligament stretching or partial tearing from forceful stress on the joint. Sprains are categorized as follows:

1. Grade 1: microscopic tears without ligament tearing or joint instability.

2. Grade 2: partial tearing of involved ligaments and laxity of joint with moderate function loss.

3. Grade 3: ligament tearing with severe function loss and joint instability.

INCIDENCE

A. *Ankle* sprains are among the most common injuries seen in primary care. Forty percent of all ankle sprains presenting to the ED in a 2016 study were in those between the ages of 18 and 35 years and less than 15% were >49 years old.

B. *Knee* injuries are among the 10 most common causes of occupational injury and worker compensation claims.

PATHOGENESIS

A. Sudden stress to a supporting ligament causes ligament stretching or tearing. Sprains are usually the result of jumping, falling, or rotating a joint.

B. Ankle sprains are most often inversion sprains, with symptoms on the lateral side of the joint.

C. Eversion injuries affect the medial side.

D. Knee sprains most often involve the patellofemoral joint.

PREDISPOSING FACTORS

A. Previous injury to ankle or knee.

B. Athletic activities.

C. Patellofemoral instability.

COMMON COMPLAINTS

A. "I twisted my ankle or knee."

B. "I stepped off of a step and came down on the side of my foot."

C. Swelling, pain, weakness of ankle or knee from a previous injury.

OTHER SIGNS AND SYMPTOMS

A. First degree: minimal pain, mild to moderate pain with stress, little swelling, minimal tenderness with palpation, little functional loss, unimpaired weight-bearing or walking, internal microdamage with full continuity.

B. Second degree: moderate pain with range of motion (ROM), swelling, marked tenderness on palpation, moderate loss of function, difficulty with weight-bearing or walking, mechanical dissociation with partial loss of continuity.

C. Third degree: severe pain, especially with passive inversion, severe swelling, marked tenderness, marked decrease in ROM, intolerant of weight-bearing or walking, joint instability, discoloration of skin, complete rupture of a ligament.

SUBJECTIVE DATA

A. Inquire about history of trauma.

B. Have the client describe the injury: time, place, activity, predisposing factors, and time symptoms developed.

C. Determine if the symptoms are acute or chronic.

D. Inquire about the type and location of the pain.

E. Have the client describe the pain and what conditions aggravate or relieve pain.

F. Ask if there are symptoms of popping, clicking, locking, recurrent swelling, or giving way of joint.

G. Ask if there is pain or other symptoms elsewhere, such as low back, hip, or leg.

H. Explore history of any previous ankle or knee injury.

I. Determine if the current injury was evaluated and treated previously.

J. Have the client describe the ability to bear weight on the extremity and to tolerate ROM.

K. Review the client's medical history for arthritis, gout, cancer, autoimmune disorders, or metabolic disease.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure.

B. Inspect:

1. Observe ambulation. Note overall appearance and facial grimaces during exam.

2. Inspect injured area for swelling, discoloration, and deformity. Compare injured side with the uninjured side.

C. Palpate:

1. Palpate the injured site for tenderness.

2. Palpate the joints above and below the injured site.

3. Perform ROM (active and passive), resisted ROM to evaluate strength.

4. Check for catching or locking of the knee on extension.

5. Assess the neurovascular status of the knee or ankle and distal extremity.

6. *Ankle*:

a. Palpate for tender sulcus in anterolateral aspect on inversion of the ankle.

b. Assess for pain with palpation over malleolar zone. Observe for bone pain with palpation over posterior edge or tip of lateral or medial malleolus.

c. Note if client is able to bear weight on extremity.

d. Assess for pain aggravated by forced ankle inversion. Perform squeeze test and external rotation stress test to assess for syndesmosis.

e. Perform isometric test of plantar flexion and eversion,

f. Perform anterior drawer test, talar tilt test.

7. *Knee*: Palpate for tenderness on the medial and lateral joint line. Perform McMurray test to detect a torn meniscus (see Section II: Procedures, "Evaluation of Sprains"). Symptoms of sprained knees:

a. Meniscus tear: locking of knee with flexion and giving way of knee.

b. Collateral ligament tear or strain: pain at lateral or medial sides.

c. Anterior cruciate tear: popping sound at injury site and immediate swelling. ▶

d. Posterior cruciate tear or strain: pain in interior knee.
e. Patellofemoral syndrome: popping or snapping, pain under patella with motion, and pain on stairs or hills.
f. Tendinitis: pain over patellar tendon.
g. Prepatellar bursitis: swelling over patella with inability to kneel due to swelling.
h. Nonspecific effusion: effusion worse with exercise.

8. See Section II: Procedures, "Evaluation of Sprains."

DIAGNOSTIC TESTS

A. The Ottawa ankle rules can assist with clinical decision-making if an x-ray is required for further assessment. These rules include ordering an x-ray series if the following exist (Stiell et al., 1994):

1. Pain in the malleolar zone and one of the following:
a. Bone tenderness at posterior edge or tip of lateral malleolus. Or
b. Bone tenderness at posterior edge or tip of medial malleolus. Or
c. Unable to bear weight immediately and in the ED for four steps.

B. Radiography of extremity, if fracture is suspected.
C. MRI, if mechanical symptoms and effusion persist.
D. Bone scans or MRI are usually reserved for those who have failed to respond after 6 to 12 weeks of therapy.

DIFFERENTIAL DIAGNOSES

A. Ankle sprain:
1. Fracture.
2. Acute dislocation.
3. Infection.
4. Ligament strain.
5. Tendinitis or tenosynovitis.
6. Nonspecific foot or ankle pain.

B. Knee sprain:
1. Fracture.
2. Dislocation.
3. Septic arthritis.
4. Infected prepatellar bursitis.
5. Inflammation.
6. Tumor.
7. Meniscus tear.
8. Collateral ligament tear.
9. Anterior cruciate ligament tear.
10. Posterior cruciate ligament tear.
11. Collateral ligament sprain.
12. Cruciate ligament sprain.
13. Patellofemoral syndrome.
14. Effusion, nonspecific.
15. Patellar tendinitis.
16. Prepatellar bursitis.
17. Nonspecific knee pain.

PLAN

A. General interventions:
1. Reinforce the degree of injury and the need to take care of extremity to prevent further damage.

B. Client teaching: *See Client Teaching Guide for this chapter, "RICE Therapy and Exercise Therapy." Give the Client Teaching Guide for this chapter, "Ankle Exercises" or "Knee Exercises."*
1. Educate the client regarding measures to improve healing. RICE (Rest, Ice, Compression, and Elevation) therapy should be stressed.
2. Teach the client techniques to protect the ankle and prevent future injuries. Techniques may include proper support (ankle brace) for the ankle when performing activities that will stress the joint (e.g., running, playing sports). Encourage the client to perform daily exercises to strengthen the joint. Exercises of choice should be directed by physical therapy.

C. Pharmaceutical therapy:
1. Drug of choice:
a. Nonsteroidal anti-inflammatory drugs (NSAIDs) to reduce pain and inflammation.
b. Consider one of the following: ibuprofen, naproxen, or diclofenac. Selective NSAIDs (celecoxib) are not superior to nonselective NSAIDs.
c. If there is increased risk for bleeding, acetaminophen with codeine may be used for pain.
2. Injectable medication:
a. Methylprednisolone acetate (Depo-Medrol) may be used if symptoms continue to be present 6 to 8 weeks after injury.
b. Repeat injection in 4 to 6 weeks if symptoms have not been reduced by 50%.

FOLLOW-UP

A. Schedule initial follow-up in 2 weeks to evaluate current therapy or sooner if problems arise.

CONSULTATION/REFERRAL

A. Refer the client to an orthopedic surgeon for the following: suspected fracture, positive squeeze test (syndesmosis injury), tendon rupture, dislocation, penetrating wound injury.
B. Refer the client to an orthopedic surgeon if therapy is unproductive and symptoms have not begun to regress within 6 weeks.
C. Refer to physical therapy for evaluation and treatment to enhance healing of injury. Physical and manual therapy both have an A rating in the treatment of an acute ankle sprain in the 2021 guidelines and in the treatment of acute knee sprain in the 2017 guidelines.
D. If no improvement with conservative treatment after 6 to 8 weeks, order an MRI to evaluate injury and refer to orthopedic surgeon.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. Medication log of geriatric clients should be available for interaction and consideration of this population for gastrointestinal, renal, cardiovascular, and endocrine disorders.
2. One in five minor injuries (such as sprains/strains) occur with adults ≥65 years old, and there is a higher acuity and longer length of stay with older adults than younger adults suffering from minor injuries.
3. Falls are the main cause of sprains/strains injuries for geriatrics. A thorough assessment of the home environment, activities of daily living (ADLs), traveling aspects, and areas the client frequently visit is recommended to help prevent fall-related accidents and potential fractures.

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CLIENT TEACHING GUIDE

ANKLE EXERCISES

A. Education: Exercising and strengthening muscles in your lower leg, foot, and ankle will improve flexibility of the joint, keep it strong and stable, and prevent future injury. This will also increase the range of motion necessary to prevent pain and injury. The following guidelines are encouraged:

1. Exercise in your bare feet or in stocking feet.
2. Count slowly ("1,001, 1,002," etc.) when you must hold a position and count.
3. Do each exercise 10 times the first day, and increase the repetitions by five each following day until you reach a maximum of 30, unless otherwise instructed.
4. Repeat prescribed exercises three times a day.
5. Exercise slowly and get the greatest stretch possible.
6. Stop any exercise that causes new, unusual, or intense pain.
7. You need to perform daily stretching exercises and toning to speed your recovery.
8. It takes months to adequately heal these types of injuries. Therefore, do not be discouraged that it takes time for your ankle to heal.
9. Sports to avoid: stop-and-go activity, basketball, running, and impact aerobics.
10. You may need to wear a Velcro ankle brace or high-top tennis shoes for support.

B. Toe and foot bend (floor):

1. Sit on the floor or bed with legs out straight.
2. Bend the injured foot back toward the head and curl toes.
3. Then point the injured foot away and bend toes back.

C. Toe rise and foot slide (chair):

1. Sit in a chair with knees bent at a right angle and feet flat on the floor.
2. Raise all toes on the injured foot and slide the foot back 3 to 4 in.
3. Relax.
4. Continue the sliding and toe raising until the heel can no longer be kept on the floor.

D. Toe and foot bends (chair):

1. Sit in a chair with knees bent at a right angle and feet flat on the floor.
2. Slide the foot on the injured side forward as far as possible while keeping the toes and heel in contact with the floor.
3. From the straight-leg position, bend the foot toward the head as far as possible.
4. Lower your foot back onto the floor.
5. Bend the foot back and forth from the straight-leg position.

E. Heel-cord stretch:

1. Stand straight and face a wall with feet together, arms straight out, and palms flat against the wall.
2. Lean toward the wall, bending the elbows, to stretch the cords above the heels.
3. Continue leaning to a count of five and then straighten up again.

F. Inner-tube stretch (1):

1. Sit with feet dangling side by side at a right angle to your legs.
2. Tie stretch bands around your feet until they are snug.
3. Keeping ankles together, move toes as far apart as possible.
4. Hold the stretch for a count of five.

G. Inner-tube stretch (2):

1. Sit with feet dangling.
2. Cross your feet at the ankles.
3. Tie bands snugly around feet.
4. Move toes as far apart as possible.
5. Hold the stretch for a count of five.

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CLIENT TEACHING GUIDE

BACK STRETCHES

You have been approved to do back-stretching exercises to help with your low back pain. Follow the instructions, starting slowly to build up your strength.

EQUIPMENT Use a mat or a towel on your floor for extra padding and comfort.

A. In the lying position:

1. Lie on your back with knees bent. Cross your arms over your chest.
2. Raise your head and shoulders and curl your trunk upward, no more than 6 in.
3. Keep the small of your back pressed against the mat.
4. Exhale during the curl up.
5. Hold _____ seconds; do _____ repetitions _____ times a day (Figure 1).

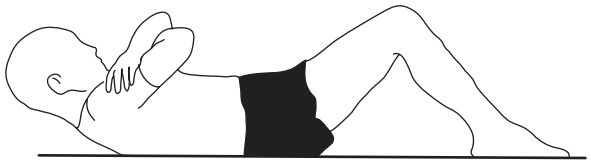


FIGURE 1 Lie on a mat or towel. Raise head and shoulders as demonstrated.

B. In the standing position:

1. Stand with your back against the wall.
2. Place your feet shoulder-width apart and 18 in. from the wall.
3. Slowly slide down the wall until you are in the "chair" position.
4. Hold for 10 seconds and relax, then slide back up the wall to a standing position.
5. Do ____ repetitions ____ times a day (Figure 2).

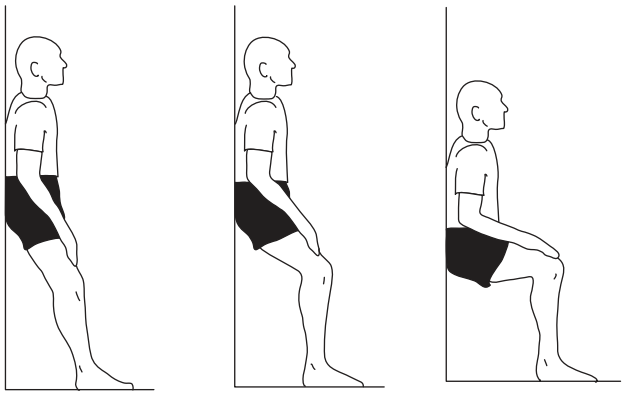


FIGURE 2 Place your feet shoulder-width apart. Slide down against the wall to the "chair" position.

C. In the lying position:

1. Bring your right knee slowly to your chest, holding it in place with your hands on your knee.
2. Relax the buttock and your back muscles.
3. Hold ____ seconds, then relax with your right knee down.
4. Repeat with your left knee.
5. Now that you have stretched both legs, pull both of your knees up, holding them in place with your hands on your knees.
6. You will be curled in the fetal position.
7. Hold ____ seconds, then relax with your knees down.
8. Do _____ repetitions _____ times per day (Figure 3).

(*continued*)

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CLIENT TEACHING GUIDE

BACK STRETCHES (*CONTINUED*)

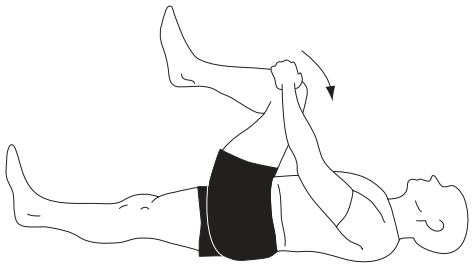


FIGURE 3 Pull your left knee toward your chest to stretch; repeat with your right knee, as demonstrated.

D. In the lying position:

1. Lie on your back with your knees bent.

2. Tighten your abdominal muscles and squeeze. As you squeeze the buttocks muscles, flatten your back toward the mat/towel (as shown in Figure III.4). Relax.

3. Tighten your buttocks muscles and lift your abdomen or "tummy" toward your knees while arching your back. Relax.

4. Hold _____ seconds; do _____ repetitions _____ times a day (Figure 21.4).

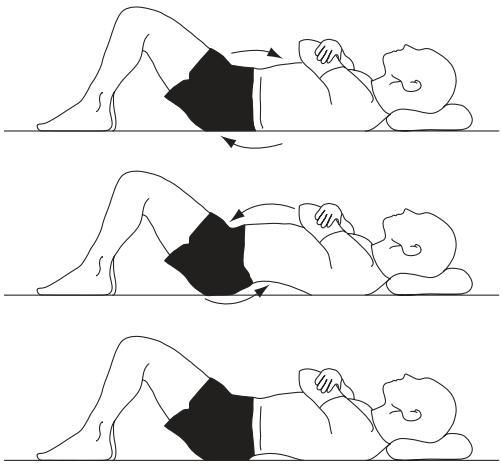


FIGURE 4 Lie with knees bent, flatten your back, then lift your tummy toward knees with back arched.

In addition to the previous exercises, yoga stretches are also very effective in adding flexibility to all muscles. Some that center on the back muscles are the following:

A. Cat stretch: Starting in tabletop position on your hands and knees (back straight), alternate between arching your back and rounding it upward as you push down on the floor with your hands and tops of your feet.
B. Cobra stretch: Lie straight out face downward (on your stomach) with full extension of the body on the ground; bring hands upward to shoulder area, bending the elbows; lift chest and torso upward as you inhale slowly; hold position for 15 to 30 seconds; lower yourself back down as you exhale.
C. Swan stretch: Lie face down on a mat or the floor with arms close to the body and hands at the shoulders; legs can be together or apart; as you inhale, lift the upper body off the mat or floor, keeping hips in contact with the mat or floor; keep the spine straight at all times; exhale and lower the body to the mat or floor. Repeat three to five times.
D. Camel pose: Begin by kneeling with the legs hip-distance apart; rest hands on your lower back and begin to lean backward until your hands can rest on your feet; hold for 30 to 60 seconds.
E. Pike stretch: Sit on floor with the back straight and legs straight out in front of you; lean forward and reach for your toes. You can do this standing up as well: standing straight, then bend over toward your toes. You do not need to touch your toes, just stretch downward.
F. Mermaid stretch: Sit straight on the floor with the right knee bent and the left knee bent underneath your body; hold your left ankle with your left hand; raise your right arm/hand over your head and reach for the ceiling as you inhale; hold for 30 seconds; exhale as you lower it. Switch sides and repeat using the left arm.

Pictures of many of the stretches can be accessed at www.wikihow.com/Get-a-More-Flexible-Back.

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CLIENT TEACHING GUIDE

KNEE EXERCISES

Knee exercises will strengthen the muscles that support the knee joint. Strong muscles protect the knee joint by keeping the knee in place and causing less strain on the knee to prevent injury. The muscles in the front of the thigh (quadriceps) and back of the thigh (hamstrings) need to be strong to support the knee to prevent injury.

CARE FOR YOUR KNEE Follow the type of exercises recommended for your knee. You may begin straight-leg raises after the pain resolves. Continue to wear your brace with activities as directed. It may take months for your knee to completely heal. You may gradually resume normal activity as directed by your provider. If you are not sure what activities you may perform, ask your provider before performing them.

A. Quadriceps sets:
1. Exercise may be done while standing, sitting, or lying down.
2. Straighten the knee with intensity.
3. Hold for 10 counts, then relax.
4. Repeat the exercise three to four times per day.
5. If a cast or splint is in place, straighten the knee until the front of the thigh and the cast pinch together.
6. If the knee is bent, keep the foot planted and use the floor to push against.

B. Cocontractions:
1. Similar to quads, but the difference is to tighten the entire thigh while straightening the knee.
2. Hold for 10 counts, then relax.
3. Repeat the exercise approximately three to four times a day.

C. Hamstring sets:
1. Pull the leg back against the other foot, floor, or cast.
2. Hold approximately 10 counts, then relax.
3. Perform these sets three to four times a day.

D. Wall squats:
1. Stand with the head, back, and hips flat against a wall.
2. Step out from the wall about 2 feet, with the back still against the wall.
3. Slowly slide your back down the wall as far as you can to almost a sitting position and hold for 10 to 15 seconds.
4. Slowly slide back up the wall. Repeat.

E. Heel lifts:
1. Lie on your back with support under the knee.
2. Lift the heel while resting the knee on support.
3. Make the knee as straight as possible.
4. Hold for 10 counts, then relax.
5. Do not use weights.
6. Perform these exercises three to four times a day.

F. Straight-leg raises:
1. Straighten the knee.
2. Lift and pause for 5 to 10 counts, then relax.
3. Lower the leg and relax, then repeat.
4. Do three to five sets, three to four times a day. Rest between each set.
5. Start with _____ lbs of ankle weight and work up to _____ lbs.
6. Gradually increase the weight used.

G. Hip flexors:
1. While sitting, lift the knee toward the chest.
2. Hold for 10 counts, then relax.
3. Lower the leg and relax. Repeat.
4. Do 10 times, three to four times a day.
5. Start with _____ lbs of weight and work up gradually to _____ lbs.
6. The weights should be on the knee or ankle.

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CLIENT TEACHING GUIDE

KNEE EXERCISES (*CONTINUED*)

H. Hamstring stretches:

1. While seated on a sturdy table with the foot of your injured leg resting on the floor, lean forward with the chin directed toward the toes.

2. Hold for at least 10 counts, then relax.

3. The knee should be straight from your hips.

4. Do not do this exercise with bouncing or violent movements.

5. Do _____ minutes, _____ times a day.

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NECK EXERCISES

Start each exercise slowly. Ease off the exercise if you start to have pain. Neck exercises are necessary for postural awareness, to prevent your muscles from becoming stiff and to stretch the neck muscles; relaxation techniques help prevent further injury of the neck. An overworked neck may cause pain in your head, upper back, shoulders, and arms. You may feel tingling or numbness of fingers.

PREVENTION: MEASURES TO PREVENT NECK PAIN

A. Avoid bending over a desk when working on a computer, reading, or writing.
B. Raise your work to eye level, keeping you from bending your head down.
C. Change your position often. Take breaks when working on something for a long period of time and do some stretching exercises.
D. Putting too many pillows under your head can cause a "kink" in your neck. Avoid sleeping on your stomach.

TREATMENT PLAN

Neck exercises: Try to do these exercises three times a day, repeating each exercise 5 to 10 times each.

A. Active range of motion:
  1. Turn the head to each side as if you were looking over your shoulder.
  2. Bend the head sideways, bringing the ear toward each shoulder.
  3. Tuck the chin in, bringing the head forward toward the chest.
  4. Shrug your shoulders and then roll them around as far as possible.

B. Stretching exercise:
  1. To stretch to the right side, place your right hand behind your head and bring your chin toward your chest.
  2. To stretch to the left side, drop the left shoulder down and tilt the right ear to the right shoulder.
  3. Lie on your back. Nod your head, bringing your chin toward your throat.
  4. Squeeze or pinch your shoulder blades together, keeping your chin tucked.

You Need to Notify the Office If You Have:

A. Increased pain in neck.
B. Numbness and tingling of fingers and hands.
C. Other physical symptoms (caused by exercise).
D. Other: _____

Phone: _____

RESOURCES

Allina Hospitals and Clinics: Home Exercise Program for Neck Pain: www.allina.com

Exercise: A Guide From the National Institute on Aging. NIH Free Publication No. 01-4258: www.nih.gov.nia

Kaiser Permanente: Neck Arthritis-Exercises: www.kp.org; enter V867 in the search box to learn more about neck exercises

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CLIENT TEACHING GUIDE

RICE THERAPY AND EXERCISE THERAPY

RICE therapies are used for muscle injuries. RICE stands for rest, ice, compression, and elevation.

REST

A. Take it easy; eliminate abuse; reduce regular exercise or activities of daily living as needed, but do not eliminate them.
B. Change activity or components of activity; for example, change running to walking.
C. Stop sports; restrict squatting, kneeling, and repetitious bending.
D. Limit weight-bearing; immobilize with crutches for partial weight-bearing.

ICE

A. Use cold in the acute phase of the injury (first 72 hours) to reduce pain and swelling.
B. Use cold in the form of ice in a plastic bag or even use frozen peas in a bag.
C. Apply cold four to eight times a day for 20 minutes at a time, with 45 to 60 minutes between applications.

COMPRESSION

A. Avoid trying to provide support with elastic bandages; a brace, overlap taping, or a cast may be needed.

ELEVATION

A. Elevate the joint above the level of the heart to reduce swelling.
B. Apply cold compress while the joint is elevated.

EXERCISE THERAPY

A. Begin exercise therapy after the initial 48-hour period if pain and swelling begin to resolve.
B. Do gentle range-of-motion exercises several times a day within the limits of pain for 7 to 10 days after knee ligament strain.
C. Exercise after 6 minutes of icing to take advantage of the cold's numbing effect.
D. Start with nonresistive, nonweight-bearing exercises.
E. Use exercises that improve range of motion and strength.
F. Maintain fitness of the extremity.

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CHAPTER 22

RHEUMATOLOGIC GUIDELINES

Jill C. Cash and Hsiao-Hui "Joyce" Ju

ANKYLOSING SPONDYLITIS

DEFINITION

A. Ankylosing spondylitis (AS) is a chronic, inflammatory joint disease that causes chronic joint pain/swelling, which primarily affects the spine and sacroiliac (SI) joints; however, larger peripheral joints can also be affected.

INCIDENCE

A. AS occurs in approximately 1% to 2% of the general population.

B. Prevalence rates differ between races. AS occurs in approximately 0.04% to 0.06% of non-Caucasians and in approximately 0.1% to 1.4% of Caucasians. North America and Europe have the highest prevalence of AS (31.9 and 23.8 per 10,000, respectively).

PATHOGENESIS

A. Inflammation occurs at the entheses (insertion site of the ligaments and tendons of the bone) throughout the body. Primary sites of involvement include the lower back, SI joint, and lower extremities. Inflammatory cells invade the joint and erode the bone and fibrocartilage of the joints. The body responds by trying to repair the tissue, in which fibrous scar tissue is formed. The structure is replaced by the ossified scar tissue, which causes fusion of the joint; in return, flexibility of the joint is lost. The eroded joint tries to repair itself by osteoblast formation in building new bone tissue. The impaired bone remodeling results in the production of new enthesis that deposits itself on top of the existing enthesis. Calcification of the spinal ligaments also occurs, and the radiograph of the fused vertebral bodies creates a unique "square" appearance often referred to as a "bamboo spine."

PREDISPOSING FACTORS

A. Sex (males > females, 3:1 ratio).

B. Race (Caucasians more prevalent, occurring more in the northern European countries).

C. Age: Onset of symptoms usually occurs during the early 20s. However, diagnosis is usually made several years after the onset of symptoms, more into the late 20- to 30-year range. AS rarely occurs after the age of 50.

D. Family history of AS.

E. Thought to have a genetic predisposition: serum human leukocyte antigen (HLA)-B27 positive genotype in approximately 90% to 95% of AS cases. However, only 5% of clients with a positive HLA-B27 develop any type of spondyloarthropathy.

COMMON COMPLAINTS

A. Gradual onset of low back pain with initial onset of symptoms occurring in the early 20s.

B. SI joint pain.

C. Joint pain occurring for more than 3 months, with morning stiffness.

D. Inflammatory low back pain/joint pain that improves with activity and worsens with rest.

E. Pain worse in the early morning and at night.

F. Dactylitis (sausage digits of the fingers/toes).

G. Enthesitis (inflammation/pain at insertion site of the tendon or ligament to the bone).

OTHER SIGNS AND SYMPTOMS

A. Peripheral joint pain and swelling.

B. Decreased range of motion (ROM) in joints/back/neck.

C. Extraarticular features such as anterior uveitis or iritis.

D. Fatigue.

E. Weight loss.

F. Shortness of breath (SOB).

G. Psoriasis.

H. Inflammatory bowel disease.

SUBJECTIVE DATA

A. Ask the client when back/joint pain began. Have symptoms been present for several months/years? At what age did pain initially begin?

B. Is pain intermittent or constant?

C. Does pain improve or worsen with activity? Inflammatory back pain improves with activity and worsens with rest.

D. Any response to nonsteroidal anti-inflammatory drugs (NSAIDs)?

E. Does client have complaints of other joint pain?

F. Has the client noted any weight gain/loss, fatigue, or fevers?

G. Any history of psoriasis or other skin rashes?

H. Any gastrointestinal (GI) changes or chronic problems such as Crohn disease, ulcerative colitis, or diarrhea?

I. Any pulmonary problems such as SOB, difficulty breathing, or SOB with activity?

J. Any cardiac changes or problems?

K. Any history of amyloidosis?

PHYSICAL EXAMINATION

A. Check vital signs, blood pressure (BP), pulse, respirations, and temperature as indicated. ▶

The contributions of Julie Barnes to this chapter in prior editions are acknowledged here.

B. Inspect:
1. Assess the cervical spine. Ask the client to stand straight and assess the degree of cervical change. Ask the client to perform flexion, extension, lateral flexion, and rotate the head.
2. Inspect the thoracic and lumbar spine for kyphosis (hunchback). Note degree of chest expansion of thoracic spine. Assess lumbar spine, noting the ROM with flexion/extension. Have the client bend to the right and left side and note any difference in flexion.
3. Assess gait. If abnormal gait, assess for hip pain/involvement.
4. Inspect hands/feet for sausage digits.
5. Inspect joints in hands/wrists/feet/knees for tender, swollen joints.
C. Auscultate:
1. Heart.
2. Lungs.
D. Palpate:
1. Examine the SI joint by palpating it while lying supine and while lying on the side. Have the client flex one knee while lying supine, and externally rotate the hip. Assess for pain at the SI joint area while pressure is applied on the knee.
2. Assess for pain at the entheses site along the spine. Assess Achilles tendon and plantar fascia areas where the tendon attaches to the calcaneus.

DIAGNOSTIC TESTS

A. Serum blood work:
1. HLA-B27 commonly positive.
2. Rheumatoid factor (RF) usually negative.
3. C-reactive protein (CRP).
4. Erythrocyte sedimentation rate (ESR).
5. Complete blood count (CBC).
6. Comprehensive metabolic panel (CMP).
7. Creatinine kinase.
8. Alkaline phosphatase.
9. Complement levels (C3, C4).
B. Radiographic studies:
1. X-ray of cervical/thoracic/lumbar spine and pelvis:
a. Radiographic changes may be subtle in the first few years.
b. As AS progresses, the spine is noted to have "squaring" of the vertebral bodies.
c. Sclerosis of the SI joint: sacroiliitis grade 2 or above (grade 2 is bilateral sacroiliitis).
2. X-ray of peripheral joints: erosions noted in the larger joints, loss of joint space, sclerosis.
3. MRI of SI joints.
4. Bone mineral density (BMD) screening: high incidence of osteoporosis noted in clients diagnosed with AS.

DIFFERENTIAL DIAGNOSES

A. Reactive arthritis.
B. Psoriatic arthritis (PsA).
C. Spondylitis associated with inflammatory bowel disease.
D. Rheumatoid arthritis (RA).

PLAN

A. General interventions:
1. Treatment for AS includes nonpharmacologic and pharmacologic interventions.
2. Osteoporosis screening and treatment are recommended for these clients.
3. Systemic steroids are not routinely prescribed; however, for severe symptoms of pain and swelling, joint injection may be considered.
B. Client teaching:
1. Encourage a healthy, active lifestyle as exercise improves symptoms. Regular physical activities are recommended as tolerated. Physical therapy, postural training, and/or regular, structured exercises may offer benefits.
2. Emphasize medication adherence and monitor for potential side effects of drug therapy.
3. Review fall prevention strategies with the client. The home should be modified to prevent falls. Suggestions include installing grab bars in the bathroom tub/shower, removing loose rugs in the house, and keeping walkways in the home clear and clutter-free.
4. Wear seat belts properly at all times.
5. Avoid contact sports and high-impact exercises/sports in clients with severe spinal involvement.
6. Emphasize the importance of regular follow-ups for disease evaluation and management of comorbidities.
C. Pharmaceutical therapy:
1. NSAIDs are first-line therapy for pain/symptoms produced from AS. NSAIDs should not be taken on an empty stomach to avoid gastric irritation. All medications should not be used longer than 4 weeks:
a. Ibuprofen 400 mg every 4 to 6 hours as tolerated.
b. Naproxen 500 mg BID as tolerated.
c. Celecoxib (Celebrex) 100 to 200 mg daily to BID as tolerated.
d. Indomethacin 75 mg one to two times per day.
e. Meloxicam 7.5 to 15 mg daily.
f. Diclofenac 50 mg Q8H.
2. Nonopioid analgesic:
a. Acetaminophen 325 to 650 mg every 4 to 6 hours or 1,000 mg TID.
3. Tumor necrosis factor alpha antagonists (anti-TNF) medications may be prescribed by a rheumatologist after failed NSAID use.
D. Nonpharmacologic interventions:
1. Refer for physical therapy.
2. Screen for depression and anxiety and encourage to join support groups.

FOLLOW-UP

A. Clients treated with NSAIDs should have routine follow-up at 2 weeks following initial diagnosis or sooner if not improving with treatment.
B. Clients treated with NSAIDs should be followed every 3 months with close monitoring of kidney and liver function.

CONSULTATION/REFERRAL

A. Clients diagnosed with AS should be referred to rheumatology for evaluation and treatment.

INDIVIDUAL CONSIDERATIONS

A. Adults:
1. The onset of symptoms in adults with AS is typically in the 20- to 30-year range. Proper diagnosis and treatment of AS is often delayed for years due to unawareness of inflammatory low back pain as a clinical feature of AS.
2. Standard precautions should be followed for NSAIDs in clients with renal insufficiency and clients taking anticoagulants, systemic glucocorticoids, and other interacting medications. ▶

B. Geriatrics:
1. Not commonly diagnosed in this group of clients over age 50.
2. As the population lives longer, we will see an increase in late life onset of AS.

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FIBROMYALGIA

DEFINITION

A. Fibromyalgia is a chronic clinical condition characterized by generalized musculoskeletal multisite pain (MSP) and is often associated with fatigue or sleep difficulties.
B. Two screening guidelines are available to classify fibromyalgia: one from the Analgesic, Anesthetic, and Addiction Clinical Trial Translations Innovations Opportunities and Networks (ACTTION)-APS Pain Taxonomy (AAPT) criteria and one from the American College of Rheumatology (ACR).
1. The 2019 AAPT diagnostic criteria are:
a. MSP at six or more defined sites out of nine (head, left/right arm, chest, abdomen, upper/lower back and spine, left/right leg).
b. Sleep difficulties or fatigue.
c. One and two above have been present for 3 months or longer.
2. The ACR criteria for diagnosis include scoring of widespread pain index from 19 defined body areas and symptom severity scale, with these symptoms lasting for at least 3 months not explained by other disorders. Diagnosis of fibromyalgia is based on client's symptoms. Tender points of 19 body areas from the previous ACR criteria are no longer required.
C. Somatic symptoms are also assessed for and present with fibromyalgia. Somatic complaints may include cognitive problems, sleeping difficulties, fatigue, headaches, and other associated symptoms.

INCIDENCE

A. Fibromyalgia affects about 2% to 3% of the population in the United States and is more common in females than males at a ratio of 9 to 10:1. Usual age of onset is between 20 and 50 years; however, it can occur from childhood to late adulthood with symptoms that may last more than 5 years before being diagnosed. The prevalence of fibromyalgia in a rheumatology practice is about 20%.

PATHOGENESIS

A. The cause is unclear. Studies of sleep physiology, neurohormonal function, muscular function, and psychological factors support a central mechanism for the disorder linked to depression. Other research suggests a pathophysiologic and psychological disorder.

PREDISPOSING FACTORS

A. Life stress.
B. Depression.
C. Female.
D. Age mid-30s and older.

COMMON COMPLAINTS

A. Common complaints are multifocal pain present longer than 3 months; moderate to extreme fatigue; morning stiffness; nonrestorative sleep; difficulty with concentration; pain worsening with stress; exposure to cold; inactivity or overactivity; sensitivity to touch, light, and sound; cognitive difficulties; and changes in barometric pressure.

OTHER SIGNS AND SYMPTOMS

A. Numbness.
B. Swelling.
C. Reactive hyperemia of skin.
D. Raynaud phenomenon.
E. Irritable bowel syndrome (IBS) and bladder symptoms.
F. Headaches.
G. Restless legs syndrome (RLS).
H. Anxiety/depression.

SUBJECTIVE DATA

A. Determine onset, duration, and course of complaints.
B. Does fatigue interfere with the client's daily activity?
C. Note sleep quality. Does the client feel rested after sleeping?
D. Do exacerbations of discomfort occur with stress, activity, and cold?
E. Has the client experienced stress and/or depression in the past?
F. Does the client have a family history of rheumatoid disease?
G. Has the client ever been diagnosed with chronic fatigue syndrome, Lyme disease, or thyroid disease?

PHYSICAL EXAMINATION

A. Check temperature if indicated, pulse, and blood pressure (BP).
B. Inspect:
1. Observe overall appearance, gait, and posture.
2. Observe the nails, skin, mucous membranes, eyes, joints, and spine. If clubbing is noted and tender points are minimal, consider hypertrophic osteoarthropathy.
3. Check joints for swelling, deformities, erythema; if clubbing is noted and tender points are minimal, consider hypertrophic osteoarthropathy.
C. Palpate:
1. Soft tissue tenderness. New ACR fibromyalgia classification criteria removed the need to palpate for tender points due to a lack of standardized tender point examination among clinicians.
2. Previous ACR criteria required the physical examination findings of pain in at least 11 of 18 specified tender point sites for a diagnosis of fibromyalgia.
D. Auscultate:
1. Heart.
2. Lungs.

DIAGNOSTIC TESTS

A. Laboratory studies are normal with fibromyalgia.
B. Testing to consider assessing for other conditions includes:
1. Complete blood count (CBC). ▶

2. Erythrocyte sedimentation rate (ESR).
3. C-reactive protein (CRP).

C. If the history and physical suggest some type of inflammatory, rheumatic condition, also order:
1. Antinuclear antibody (ANA).
2. Rheumatoid factor (RF).
3. Cyclic citrullinated peptide (CCP).

D. If there are concerns regarding a possible thyroid or muscle condition, also order:
1. Thyroid-stimulating hormone (TSH).
2. Creatinine phosphokinase.

E. Assessment for a sleep disorder should include ordering a sleep study.

DIFFERENTIAL DIAGNOSES

A. Fibromyalgia.
B. Rheumatoid arthritis (RA).
C. Osteoarthritis (OA).
D. Polymyalgia rheumatica (PMR).
E. Ankylosing spondylitis.
F. Myositis.
G. Systemic lupus erythematosus.
H. Hypothyroidism.
I. Chronic fatigue syndrome.
J. Obstructive sleep apnea.

PLAN

A. General interventions:
1. Routine follow-up is recommended. Multiple therapies may be beneficial for controlling symptoms.
2. Stress the importance of regular physical activity to reduce pain.
3. Support groups are beneficial for clients and families.

B. Client teaching: *See Client Teaching Guide for this chapter, "Fibromyalgia."*
1. Teach the client that fibromyalgia is a recognizable syndrome that is benign in nature. Clients can be assured it is not "all in their head."
2. Exercise: Encourage the client to exercise daily, including stretching programs along with walking, low-impact cardiovascular conditioning such as cycling, and low-impact aerobics. Initially, pain may increase with the first 2 weeks of exercise, then improves with a routine exercise program.
3. Pain control: Pain may improve with exercise, hot baths, heating pads, warm weather, and stress reduction.

C. Pharmaceutical therapy:
1. Amitriptyline 10 to 50 mg at bedtime for sleep.
2. Cyclobenzaprine 10 to 30 mg daily or other muscle relaxants.
3. Nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen 200 to 600 mg every 4 to 6 hours. Take with food never on empty stomach.
4. Analgesics such as acetaminophen (Tylenol) as needed.
5. Selective serotonin reuptake inhibitors (SSRIs) if depression is present.
6. Pregabalin (Lyrica) 75 mg BID to 150 mg BID; maximum dose of 450 mg/d.
7. Duloxetine HCl (Cymbalta) 30 to 60 mg once a day. Give precautions of nausea with medication. If nausea is severe, consider prescribing Zofran 8 mg 30 minutes prior to Cymbalta dose. Advise client that nausea will resolve after 3 to 4 weeks.
8. Milnacipran (Savella) titrated dose 12.5 mg on day 1, 12.5 mg BID for 2 days, 25 mg BID from days 4 to 7, then 50 mg BID (recommended dose). Maximum dose is 100 mg BID. Withdraw gradually. Caution in those with renal impairment.
9. Current guidelines suggest guarded use of opioids chronically in nonmalignant pain. Opioids have not been studied in randomized controlled trials and should be considered only after all other medicinal therapies have been exhausted. Tramadol, a centrally acting analgesic with atypical opioid and antidepressant-like activity, is moderately effective in treating fibromyalgia pain.
10. Neurontin (Gabapentin) is approved for use in treatment of neuropathic pain but not for fibromyalgia. Neurontin 300 mg orally TID should be the starting dose, with titrations upward as tolerated.

FOLLOW-UP

A. Schedule regular visits in the initial 2 to 4 weeks to evaluate how therapy is helping. Educate the client on the nature of the condition during each visit, provide positive reinforcement for exercising, and supervise treatment adherence and effectiveness. Visits may then be scheduled every 3 months to monitor progress.

CONSULTATION/REFERRAL

A. Consult with a physician if the client has abnormal laboratory results.
B. Consult or refer the client to a physician if depression is suspected and current medication therapy is unsuccessful.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Fibromyalgia pain in pregnancy is mostly low back pain and fatigue is the most common symptom.
2. Fibromyalgia may impact pregnancy, physiologically and psychologically, in a negative manner.
3. Medications used for fibromyalgia such as milnacipran (Savella), pregabalin (Lyrica), and duloxetine HCl (Cymbalta) should be avoided in pregnancy, and cyclobenzaprine, amitriptyline, and gabapentin should be only considered if the benefit greatly outweighs the risks.

B. Geriatrics:
1. Fibromyalgia is common in older adults and can be more painful for this population due to other coexisting conditions, such as OA. Fibromyalgia pain that waxes and wanes in a younger population may be a constant in the older adult population.
2. Fibromyalgia prevalence has accelerated in the geriatric population. Studies show that older adults suffering with fibromyalgia are more depressed and less physically active. Treatment options that are most appropriate include antidepressants titrated according to the Beers Criteria as chronic use of steroids/NSAIDs could lead to adverse effects.
3. Fibromyalgia syndrome is associated with increased risk of falls and fractures. Calcium and vitamin D levels should be monitored. Vitamin D_3 recommendation for clients older than 70 years of age is 24,000 IU/mo.

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GOUT

DEFINITION

A. Gout is an inflammatory disease of the joint caused by high concentrations of uric acid leading to deposition of monosodium urate crystals in the joints and bones. Acute gout attacks must be treated properly to avoid progression to chronic gout, which can lead to permanent joint damage.

B. Three stages of gout:

1. Acute gouty arthritis: a sudden onset of severe joint pain, redness, warmth, and swelling, which may last from days to weeks.

2. Intercritical gout: period without gout flares.

3. Chronic/tophaceous gout: inadequately treated gout episodes that eventually result in urate crystal deposits (tophaceous deposits) in the joints, which can cause joint destruction, deformity, and disability.

INCIDENCE

A. Gout is most common in males aged 30 to 60 years. Females become increasingly susceptible to gout after menopause. It is estimated that gout occurs in approximately 3% of the adult population in the United States.

PATHOGENESIS

A. Primary: High levels of uric acid result from either increased production or decreased excretion rates of uric acid leading to deposition of monosodium urate crystals, triggering an inflammatory response.

B. Secondary: Hyperuricemia results from primary disease processes such as hypertension, renal failure, kidney disorders, and so forth. See the "Predisposing Factors" section.

C. Medications/toxins can also cause hyperuricemia.

PREDISPOSING FACTORS

A. Chronic conditions: hyperuricemia, chronic kidney disease (CKD), hypertension, diabetes mellitus, metabolic syndrome, ischemic cardiovascular disease (CVD), hyperlipidemia, obesity, osteoarthritis (OA).

B. Sex (males older than 30 years, postmenopausal females).

C. Medications that alter uric acid level (thiazide and loop diuretics, acetylsalicylic acid, alcohol, nicotinic acid, ethambutol, pyrazinamide).

D. Dietary factors (high intake of beer, meat/fresh seafood products, and sweetened beverages).

E. Family history of gout.

F. Lead poison.

COMMON COMPLAINTS

A. Redness, swelling, warmth, and/or pain in the joint (initially, usually one joint only—often the great toe known as podagra or first metatarsophalangeal joint). The pain is likely to be most severe in the first 12 to 24 hours.

B. History of having severe joint pain with inflammation in other joints, followed by pain-free episodes.

OTHER SIGNS AND SYMPTOMS

A. Tophi are seen from several years of untreated gout.

B. Fever and chills may be present in acute stages.

SUBJECTIVE DATA

A. Note the onset of symptoms and the course of development.

B. Review client's history of gout.

C. Determine what makes the symptoms worse or better.

D. List medications/therapies used and the effectiveness of each medication/therapy.

E. Review recent dietary preferences (alcohol, meat, seafood, sweetened beverages).

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure (BP).

B. Inspect:

1. Inspect the appearance and mobility of involved joint(s).

2. Note the presence of tophi on other joints.

C. Palpate:

1. Palpate joints for tenderness, pain, and warmth of skin.

DIAGNOSTIC TESTS

A. Joint fluid aspiration for urate crystals is the gold standard in diagnosing gout. Inspect fluid with a polarized light microscopy to identify uric acid crystals.

B. Complete blood count (CBC): elevated white blood cell (WBC) count.

C. Erythrocyte sedimentation rate (ESR): elevated in gout.

D. Serum uric acid level: uric acid greater than 6.8 mg/dL. Serum uric acid level may be normal during acute attacks. Perform test at least 2 weeks after acute attack or when flare has resolved.

E. Rheumatoid factor (RF): titer.

F. X-ray or MRI: identifies bone cysts/gout tophi; may be negative in early cases.

DIFFERENTIAL DIAGNOSES

A. Gout.

B. Infectious arthritis.

C. Rheumatoid arthritis (RA).

D. Hyperparathyroidism.

E. Pseudogout (calcium pyrophosphate deposition disease) commonly occurs in the knee, wrist, or other joints.

F. Bursitis.

G. Cellulitis.

PLAN

A. General interventions:

1. Rest the affected joint area; no heavy lifting or weight-bearing activity.

2. Avoid medications and dietary products that increase uric acid levels.

3. Manage chronic conditions associated with gout.

B. Client teaching: *See Client Teaching Guide for this chapter, "Gout."*

1. Increase fluid intake to at least eight glasses of water daily.

2. Avoid alcohol intake and excessive meat and seafood products that trigger flares.

3. Reinforce proper medication management and compliance of the prescribed medications to prevent the development of chronic tophaceous gout.

4. Avoid medications that increase uric acid levels (aspirin).

C. Pharmaceutical therapy:

1. Analgesia:

a. Nonsteroidal anti-inflammatory drug (NSAIDs) is most effective if started within 48 hours of presenting symptoms:

i. Indomethacin 50 mg Q8H for eight doses, then 25 mg Q8H until pain-free for 1 to 2 days. Normal course is 5 to 7 days.

ii. Naproxen, 750 mg initially, followed by 500 mg Q12H.

iii. NSAIDs are contraindicated in clients with a diagnosis of renal insufficiency, heart failure, peptic ulcer disease, NSAID allergy, and anticoagulation therapy.

b. Colchicine (Colcrys) is most effective if started within 12 to 24 hours of presenting symptoms: 1.2 mg initial dose, followed by 0.6 mg 1 hour later. Continue 0.6 mg one to two times daily until symptoms resolve. Medication may be stopped after the client is free from symptoms after 2 to 3 days. Colchicine may also be used to prevent attacks. Colchicine warnings: Be aware of contraindications. Caution regarding drug interactions. Intolerable side effects of colchicine include nausea, vomiting, and diarrhea.

For long-term use, colchicine 0.6 mg/d may be used in clients who have more than one attack per year. See package insert for recommendations for more frequent use.

c. Corticosteroids may be used in clients who cannot tolerate oral medications.

i. Intraarticular steroid injection of methylprednisolone acetate may be given.

ii. Oral steroids may also be prescribed in clients who cannot take NSAIDs or colchicine and who cannot tolerate intraarticular steroid injection. Prednisone 40 mg daily for 5 days or tapered packs can be effective. Be aware of rebound effects.

2. For hypersecretion of uric acid (frequent attacks of ≥2 yearly, tophi on exam or imaging, CKD stage 2 or worse, past urolithiasis), long-term therapy is needed to reduce uric acid production:

a. Allopurinol (Zyloprim) is the drug of choice. Starting dose 100 mg/d and titrate up over several weeks to a maximum of 800 mg/d, but adjust dose for clients with renal impairment. The goal is to keep the uric acid level less than 6.0 mg/dL.

b. Febuxostat (Uloric) is also used to lower blood uric acid levels. The recommended dose is 40 mg/d with or without food; maximum dose is 80 mg/d. Initial laboratory studies include liver enzymes before beginning Uloric. Common side effects include liver problems, nausea, gout flares, joint pain, and rash.

3. For reduced excretion rates of uric acid, consider probenecid (Benemid) 250 mg PO BID for 1 week, then increase to 500 mg PO BID. Recommend an increased fluid intake during therapy. Avoid if creatinine clearance (CrCl) is less than 50.

4. Chronic gout: If clients have three or more attacks per year, consider long-term therapy, low dose of NSAIDs, or allopurinol for 2 to 12 months.

a. Alternative medications for intolerance, renal insufficiency, and extensive tophi include oxypurinol, febuxostat, and uricase. Refer to rheumatology specialist.

FOLLOW-UP

A. Clients with acute gout flare-ups should be evaluated within 24 hours of symptom onset.

B. Schedule follow-up visit in 1 month to reevaluate status.

C. Monitor serum urate during medication titration every 2 to 5 weeks to target.

D. Chronic gout: Obtain yearly uric acid levels; before initiating long-term therapy, obtain baseline blood urea nitrogen (BUN), serum lipid profile, and CBC and periodic liver enzymes.

CONSULTATION/REFERRAL

A. Consult and refer to a physician or rheumatologist for aspiration of joint fluid and newer treatment options.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Colchicine is not recommended.

B. Geriatrics:

1. Reformation in joint areas may be seen in clients who have a history of gout from uric acid deposits.

2. Complications of chronic gout include nephrolithiasis and chronic urate nephropathy.

3. Avoid indomethacin in older adult clients due to the increased risk of adverse effects with other medications when compared with other NSAIDs.

4. NSAIDs are not recommended in older adults with a history of heart failure, gastrointestinal (GI) ulcers/disease, and renal impairment.

5. Individual considerations must be given in this population regarding GI, cardiac, renal, and liver disease.

6. Severe gout symptoms ≥2 years in duration are associated with CVD in the adult and geriatric population. Studies have suggested that preventing gout-related inflammation could be clinically beneficial to cardiovascular health. Uricosuric agents that may be helpful in preventing CVD in older clients with gout are fenofibrate and losartan.

7. Allopurinol can have a negative interaction with warfarin, cyclosporine, chlorpropamide, and thiazide diuretics.

8. The Beers Criteria recommends avoiding colchicine in geriatric clients with CrCl less than 30 mL/min secondary to increased risk of bone marrow toxicity, GI complications, and neuromuscular adverse effects. The use of probenecid should also be avoided in geriatric clients with CrCl less than 30 mL/min due to loss of efficacy.

9. Older adult clients with a severe gout attack are often misdiagnosed with septic arthritis because geriatrics clinically present indistinguishable symptoms of fever, leukocytosis, and C-reactive protein (CRP) elevation with gouty inflammatory response. Clinical guidelines recommend administering empirical antibiotics and observing clients until culture and sensitivity results are available before proceeding with invasive joint lavage. Studies revealed that joint lavage is associated with prolonged pain and increased length of hospital stay in clients ≥65 years old.

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OSTEOARTHRITIS

DEFINITION

A. Osteoarthritis (OA), formerly known as degenerative joint disease, is a common chronic arthritic disease of the joints that can cause pain and activity limitation. OA is characterized ▶

by destruction of the cartilage with resultant decrease in the joint spaces and reactive bony overgrowth. The most common form of OA is the primary or idiopathic condition that often affects adults and older adults. Secondary OA can occur with predisposing conditions including trauma, septic arthritis, inflammatory arthritis, metabolic disorders, or congenital or acquired joint abnormalities.

INCIDENCE

A. It is estimated that up to 12% of the general population between the ages of 25 and 74 years have OA. The incidence increases with age, as up to 85% of the general population older than 65 years of age have radiographic changes suggestive of OA.

PATHOGENESIS

A. Damage to the articular cartilage and subchondral bone may be a result of an increased stress on the joints from overuse, obesity, or trauma leading to chondrocyte damage.
B. Chondrocytes release proteolytic enzymes to assist in repair of the cartilage, but these enzymes eventually erode the cartilage overtime.
C. In OA, the damaging remodeling process of chondrocytes and erosive release of enzymes result in the weakening and destruction of the subchondral bone. Joint destruction and reactive bony overgrowth most frequently occur in knees, hands, hips, and spine.

PREDISPOSING FACTORS

A. Increasing age: among clients older than 55 years.
B. Sex: Females are more commonly affected and exhibit greater disease severity.
C. Genetic predisposition; distal interphalangeal (DIP) and proximal interphalangeal (PIP) joints involvement.
D. Trauma such as previous fractures, ligamentous injuries, or occupationally related repetitive stress.
E. Altered joint anatomy or instability.
F. Obesity: Mechanical stress on the knee may increase OA.
G. Secondary inflammation such as infections, inflammatory arthropathies, and metabolic disorders.

COMMON COMPLAINTS

A. Unilateral joint pain frequently involving the joints of the hands, neck, lower back, knees, and hips.
B. Morning stiffness lasting less than 1 hour.

OTHER SIGNS AND SYMPTOMS

A. Unilateral joint pain and/or deformity involving the DIP and PIP joints, first carpometacarpal joint, hips, knees, cervical and lumbar spine, and first metatarsophalangeal joint.
B. Mild OA, or early disease, joint pain that increases with movement and decreases with rest.
C. Severe OA, or late disease, joint pain that is present with rest and can progress to involve multiple joints.
D. Activity limitation (abnormal gait and/or impaired manual dexterity).

SUBJECTIVE DATA

A. Elicit the client's age at onset of pain.
B. Has the pain gradually gotten worse over the months or years?
C. How long does the pain last in the morning? Does the joint pain get worse with movement and better with rest?
D. What joints are involved?
E. Is the joint pain described as "aching"?
F. What does the client take to relieve the pain?
G. Is there any joint deformity, redness, swelling, or warmth?
H. Is there any decrease in range of motion (ROM) of the joint?
I. Is there any family history of OA?

PHYSICAL EXAMINATION

A. Check pulse and blood pressure (BP).
B. Inspect the joints for enlargement, edema, and erythema.
C. Palpate:
  1. Palpate the joints, noting temperature, edema, and tenderness. Joints are cool. Bony enlargement may be present in the PIP (Bouchard nodes) or DIP (Heberden nodes) joints and other weight-bearing joints.
  2. Palpate extremities. Perform assisted and active ROM exercises. With examination, limited ROM of the joint and/or pain on palpation may be present, along with crepitus.

DIAGNOSTIC TESTS

A. Erythrocyte sedimentation rate (ESR): OA does not cause an increase in ESR.
B. Chemistry profile.
C. Complete blood factor (CBC).
D. Rheumatoid factor (RF).
E. Routine radiography confirms disease severity and presence of joint narrowing.
F. CT scan or MRI is considered with nerve impingement syndrome (spine) or spinal stenosis.

DIFFERENTIAL DIAGNOSES

A. Inflammatory OA.
B. Rheumatoid arthritis (RA).
C. Gout or pseudogout.
D. Septic arthritis.
E. Bursitis or tendonitis.
F. Systemic lupus erythematosus (SLE).
G. Fracture or trauma.

PLAN

A. General interventions:
  1. Confirm diagnosis.
  2. Provide the client with support and education to improve client well-being and reduce discomfort.
  3. Physical therapy and/or occupational therapy should be initiated, if indicated.
B. Client teaching: *See Client Teaching Guide for this chapter, "Osteoarthritis."*
  1. Reinforce the importance of joint protection; avoid repetitive stress or trauma.
  2. Encourage a combination of aerobic and strengthening exercises tailored to individual needs.
  3. Encourage weight loss if indicated. Joint pain can be significantly reduced with weight loss of at least 10% of body weight.
  4. Consider the use of assistive walking devices, braces, and foot orthotics if appropriate.
C. Pharmaceutical therapy:
  1. First-line agents: The goal of treatment is to preserve joint mobility. First-line agents should be used in a stepwise approach.
    a. Acetaminophen up to 1 g QID; in early disease, this may be given on an as-needed basis.
      i. Pay special attention to the maximum dosage per day as acetaminophen is commonly combined with other over-the-counter medications to treat pain and cold symptoms.

b. Nonsteroidal anti-inflammatory drugs (NSAIDs) if acetaminophen has failed to control the pain. Consider renal function and caution use in clients with risk factors for peptic ulcer disease (PUD) and cardiovascular disease (CVD). None of these agents should be taken on an empty stomach to avoid gastric erosion.

i. Naproxen 220 to 375 mg one to two times daily.

ii. Inflammatory OA: Consider naproxen 375 to 500 mg BID. Recommend taking this medication for 2 to 4 weeks for maximum effects.

iii. Ibuprofen 200 to 400 mg TID may also be considered. Doses may be increased at a gradual pace for maximum benefit as tolerated. If one NSAID does not work, consider other NSAIDs.

1) Cox-2 inhibitors, such as celecoxib (Celebrex), may also be considered. Celebrex 200 mg daily to BID as tolerated.

2) Meloxicam (Mobic) 7.5 to 15 mg daily.

c. For high-risk clients with comorbidities, an H2 receptor blocker or a proton pump inhibitor may reduce the risk of gastritis and prevent NSAID-induced duodenal ulcers. Consider Arthrotec 50 mg/200 mcg one tablet two to three times a day, or naproxen/esomeprazole 375 mg/20 mg or 500 mg/20 mg BID.

d. Misoprostol may be considered in clients who are at high risk of gastric ulcers. It should not be used by pregnant persons. It is considered high risk for fetal death and possible congenital abnormalities.

e. Topical creams:

i. Diclofenac gel (Voltaren) may be applied to the affected area; recommended for clients with severe pain who are not able to tolerate oral NSAIDs.

ii. Capsaicin cream may be applied to the affected area. Capsaicin creams may cause local burning at the site of application for the first several days.

2. Second-line agents, such as intraarticular corticosteroid injections, may prevent some joint erosion and reduce pain. The same joint should not be injected more than three to four times a year in 3-month intervals. If the joint is injected at this frequency for more than 1 year, alternative options, such as surgery, should be considered. Narcotics may provide relief from more severe OA pain, but they carry a risk of dependence. These should be reserved for clients who do not respond to other treatments and are not surgical candidates. Follow state protocols for scheduled pain medications.

3. The use of glucosamine and chondroitin has not been established and is not recommended for clients due to limited data on efficacy. If the client does not notice any relief within the first 6 months of use, recommend discontinuing the product.

4. Physical therapy to create an individualized exercise regimen to strengthen muscles, increase ROM, and reduce pain.

5. Lubrication injections (Hyalgan or Synvisc) may also be considered. Referral to a rheumatologist should be considered for this treatment.

FOLLOW-UP

A. Follow-up is based on disease severity and therapeutic treatment. If the client is treated with first-line agents, follow up on pain control, nonpharmacologic interventions, and possible side effects of medications within 2 to 4 weeks.

CONSULTATION/REFERRAL

A. Referral to an orthopedic surgeon may be considered for moderate to severe pain, as indicated.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. NSAIDs should not be used in pregnancy unless clearly indicated. Misoprostol should be used with great caution in persons of childbearing age due to its potential for fetal abnormalities and abortive properties.

B. Adults and geriatrics:

1. Clients on chronic NSAIDs should be monitored closely for toxicity, such as renal insufficiency, gastritis, PUD, and cardiovascular complications. Older adult clients and those with preexisting gastrointestinal disease, diabetes, congestive heart failure, and cirrhosis should be monitored closely.

2. For those with multiple joint involvement and comorbidities limiting the use of NSAIDs or have inadequate response to NSAIDs, duloxetine 30 mg orally once daily for a week and increasing to 60 mg orally once daily may be efficacious.

3. Topical therapies are preferred in clients over age 75 due to increased risk for complications from oral NSAIDs.

4. Studies indicate that clients who adhered to the Mediterranean diet have a lower chance of developing OA. The Mediterranean diet is high in beta carotene, omega 3, and magnesium, which may reduce inflammatory markers and OA-related pain. In addition, clients can lose weight following the Mediterranean diet, which also alleviates burden on the knees and reduces the inflammatory response.

5. Sedentary lifestyle contributes to OA and increases joint burden. Studies indicate that pain-coping skill training for the geriatric population improved self-efficacy in controlling OA-related knee pain, thereby increasing physical functions and enhancing quality of life.

6. Use with caution or avoid splinting/orthotics for long periods (≥6 weeks) due to clients' vulnerability to periarticular muscle weakness and wasting. Splints worn at night may reduce pain and prevent unintentional injury.

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OSTEOPOROSIS/KYPHOSIS/FRACTURE

DEFINITION

A. Osteoporosis is a condition of reduced bone mass resulting in an increased risk of bone fragility and fracture. The World Health Organization (WHO) has defined it as a measurement of bone mineral density (BMD) ≥2.5 standard deviations below the normal mean for healthy, young females (T-score of –2.5 or lower) measured by dual-energy x-ray absorptiometry (DXA, DEXA). Measurements of BMD are performed on the lumbar spine, hip, and forearm. ▶

B. Clinical diagnosis of osteoporosis can also be defined as having a fragility fracture of the spine, hip, wrist, pelvis, rib, and/or humerus without evidence of a BMD.

C. Osteopenia is defined as low bone mass of a BMD that is between 1 and 2.5 standard deviations below the mean as T-score between −1.0 and −2.5.

D. Kyphosis (dowager's hump) is the forward curvature of the thoracic spine. It is estimated that kyphosis occurs in approximately 20% to 40% of clients older than 60 years of age. Kyphosis may occur due to many different causes, including vertebral fractures, muscle weakness, degenerative disc disease, postural changes, and genetic/metabolic changes. Kyphosis is associated with other conditions such as decreased pulmonary function, back pain, increased risk of fracture of the spine, limited mobility, and an increase in mortality.

E. Osteoporotic fracture occurs from a fall while standing at normal height or less, without any type of trauma and/or while performing daily activities. A vertebral compression fracture is the most common type of osteoporotic fracture.

F. Vertebral fractures: There are three primary types of vertebral fractures:

1. Biconcave deformity.
2. Wedge fracture.
3. Compression fracture.

INCIDENCE

A. There are approximately 25 million people in the United States diagnosed with osteoporosis; 80% of those are female. Females are diagnosed with osteoporosis six to eight times more often than males and this is thought to be related to estrogen deficiency. The peak incidence of fractures in males occurs 10 years later in life than for females, at roughly 70 years of age.

B. Approximately 50% to 60% of 50-year-old females sustain osteoporosis-related fractures during their remaining life. Spinal fractures occur in 25% of White females by age 65 years, causing pain, deformity, and disability. The most common fractures include 25% at distal radius (Colles fracture), 50% in the vertebrae, and 25% in the hip.

C. Approximately 33% of all females and 17% of males suffer a hip fracture before age 90 years, and 20% of those who sustain a fracture die within 3 months of the event.

PATHOGENESIS

A. Primary osteoporosis is due to aging process or menopause.

1. Aging-related osteoporosis occurs mainly because of the rate of bone resorption becomes greater than the rate of bone formation.
2. Estrogen deficiency is the principal mechanism of osteoporosis in menopause.

B. Secondary osteoporosis is a consequence of underlying health conditions or medications.

1. Health conditions including hyperthyroidism and chronic renal failure are common conditions associated with secondary osteoporosis.
2. Long-term use of systemic corticosteroids and excessive thyroid replacement therapy are important causes of secondary osteoporosis.

PREDISPOSING FACTORS

A. Hypogonadal states, particularly menopause.
B. Small body frame, low body weight (<127 lbs).
C. Current smoking.
D. Low calcium or vitamin D intake.
E. Physical inactivity; lack of weight-bearing exercise.
F. Family history of osteoporosis.
G. Excessive alcohol intake.
H. Asian or White.
I. Advanced age.
J. Previous fracture.
K. Secondary causes:

1. Hyperparathyroidism.
2. Hyperthyroidism.
3. Cushing syndrome.
4. Malignancy (multiple myeloma, lymphoma, leukemia).
5. Thyroid replacement therapy.
6. Corticosteroid therapy.
7. Chronic renal or liver disease.
8. Type 1 diabetes mellitus.
9. Anorexia nervosa.

COMMON COMPLAINTS

A. Loss of height.
B. Kyphosis, or dowager's hump.
C. Back pain (may indicate a vertebral compression fracture).

OTHER SIGNS AND SYMPTOMS

A. Cervical lordosis.
B. Fracture with little or no trauma.
C. Crush fracture of vertebra.
D. Pain, especially after minor injuries.

SUBJECTIVE DATA

A. Explore history of the following:

1. Loss of height. Ask the client to compare current height with height written on the driver's license if height is unknown.
2. History of low bone mass (elevated T-score) result in the past.
3. Early menopause, oophorectomy, postmenopause, or amenorrhea.
4. European or Asian family origin.
5. Family history of spinal fractures and osteoporosis.
6. Sedentary lifestyle with little weight-bearing activity.
7. Endocrine disorders.
8. Review medications taken in the present and past, including over-the-counter (OTC) and herbal supplements, with attention to medications such as corticosteroids, barbiturates, heparin, and thyroid hormone.
9. Dietary review for low calcium and vitamin D intake add period.
10. Smoking, alcohol, caffeine, and high protein intake.
11. Renal disease/dialysis add period.

B. Determine onset, duration, location, and characteristics of back pain if present.

PHYSICAL EXAMINATION

A. Check pulse, blood pressure, height, and weight.
B. Inspect:

1. Compare present height with previous height.
2. Observe presence of dorsal kyphosis.
3. Observe physical abnormalities that interfere with mobility.

C. Palpate:

1. Palpate joints and over the back for pain.

DIAGNOSTIC TESTS

A. Laboratory studies:

1. Females: complete metabolic panel (CMP; calcium, phosphorus, albumin, total protein, creatinine, liver enzymes, ▶

alkaline phosphatase, electrolytes), thyroid-stimulating hormone (TSH), parathyroid hormone (PTH), and 25-hydroxyvitamin D (25[OH]D) level.

2. Males: CMP (calcium, phosphorus, albumin, total protein, creatinine, liver enzymes, alkaline phosphatase, electrolytes), TSH, 25(OH)D level, and testosterone level.

3. Complete blood count (CBC), erythrocyte sedimentation rate (ESR), and serum protein electrophoresis to rule out multiple myeloma and leukemia, if concerned.

B. DEXA scan for measuring BMD:

1. The DEXA scan usually measures bone density of the lumbar spine and hip. Medicare will reimburse for this test to be performed every 2 years. If the spine is compressed or has severe scoliosis, BMD values may not be valid for the lumbar spine. Consider assessing the forearm for radial interpretation.

2. The result of this procedure is read in "T-scores" or "Z-scores." T-scores are evaluated for postmenopausal females and males 50 years and older. A T-score is a number given to identify the amount of bone present when compared with healthy young adults of the same sex. Z-scores are recommended for premenopausal females, males younger than 50 years, and children. A Z-score is a score given to identify the amount of bone present when compared with other people of the same age and sex.

3. The WHO Fracture Risk Assessment Tool (FRAX) was developed to estimate the risk of osteoporosis-related fractures in the next 10 years. This validated tool is available online (www.sheffield.ac.uk/FRAX/) and may be used with the BMD results to determine who needs to be treated with medication for prevention of fracture when treatment is unclear. According to the National Osteoporosis Foundation, FRAX should be used in clients who have T-scores between −1.0 and −2.5 in the spine or total hip region. The FRAX tool can also be used in clients who have been treated with prior antiresorptive medications if they have been off these medications for more than 2 years. The tool is validated to be used in clients not currently on antiresorptive medications.

C. Radiography:

1. X-ray the lumbar and thoracic spine for suspected vertebral fracture and/or for height loss greater than 2 in. Consider a CT to assess for instability of a wedge fracture. An MRI is recommended to assess for the extent of a compression fracture and/or possible malignancy.

DIFFERENTIAL DIAGNOSES

A. Osteoporosis.

B. Osteoarthritis (OA).

C. Infection of the spine.

PLAN

A. General interventions:

1. Lifestyle changes to minimize potential risk factors should be introduced to the client.

2. Educate the client regarding proper diet with calcium and vitamin D intake daily along with regular weight-bearing exercises.

B. Client teaching: *See Client Teaching Guide for this chapter, "Osteoporosis."*

1. Educate the client regarding calcium and vitamin D intake in diet:

a. Recommended daily calcium intake:

i. Males 19 to 70 years: 1,000 mg/d.

ii. Males older than 70 years: 1,200 mg/d.

iii. Females 19 to 50 years: 1,000 mg/d.

iv. Females older than 50 years: 1,200 mg/d.

v. Food sources high in calcium include salmon or sardines with bones, low-fat yogurt and skim milk, green vegetables, and cheese.

b. Recommended vitamin D intake:

i. Ages younger than 50 years: 400 to 600 IU/d.

ii. Ages 51 to 70 years: 600 to 1,000 IU/d.

iii. Ages 71 years and older: 800 to 1,000 IU/d.

iv. Food sources high in vitamin D include vitamin D-fortified milk and cereals, fish liver oils, cod liver oil, mushrooms, herring, catfish, salmon, sardines, egg yolks, cheese, and beef liver.

2. Advise against potential risk factors such as alcohol excess and cigarette smoking.

3. Prescribe regular moderate exercise, such as 30 minutes of weight-bearing exercises (walking, jogging, dancing) at least three times per week. Walking 50 to 60 minutes three times per week provides optimal benefits.

4. Fall prevention: Advise the client to avoid medications that may cause drowsiness or postural hypotension and precipitate falls. Use extra light at night in the bathroom to help prevent falls. Remove all loose rugs and clutter from the home. Install hand rails on steps in the home to ensure safety.

5. Discuss safety issues and fall prevention strategies with the client and family. *See Client Teaching Guide for Chapter 3, "Safety Issues: Fall Prevention."*

C. Pharmaceutical therapy:

1. Calcium supplements.

Calcium supplements may be contraindicated in clients who have a history of renal stones.

2. Calcium carbonate (Os-Cal) 500 mg is given orally one to three times per day, or Tums one tablet orally two to three times a day. See the "Plan" section for recommendations.

3. Vitamin D supplements: See the "Plan" section for recommendations. Clients diagnosed with vitamin D deficiency should ingest an increased dosage, according to the deficiency.

4. Bisphosphonates: first-line therapy recommended for postmenopausal females to reduce fracture risk in osteoporosis.

a. Reduce bone resorption and bone loss by preventing osteoclast activity.

b. May be given daily, weekly, or monthly according to individual medication.

i. Alendronate sodium (Fosamax): 10-mg tablet oral daily; 70-mg tablet or liquid oral weekly; alendronate sodium/cholecalciferol (Fosamax Plus D) 70-mg tablet + 2,800 IU vitamin D tablet oral weekly or 70-mg tablet + 5,600 IU vitamin D tablet oral weekly. If osteoporosis is due to corticosteroid therapy, 5 to 10 mg oral daily.

ii. Risedronate sodium (Actonel): available as 5-mg oral daily, 35-mg oral weekly, or 150-mg oral monthly tablets. Check serum creatinine, phosphorus magnesium, and calcium levels prior to beginning therapy.

iii. Zoledronic acid (Reclast): 5 mg/100 mL (intravenous [IV]) infusion once yearly as 15-minute infusion. Check serum creatinine, phosphorus, magnesium, and calcium levels before infusion.

iv. Ibandronate (Boniva): available 2.5 mg oral once a day in the morning or 150 mg oral once a day monthly. Check serum creatinine, phosphorus magnesium, and calcium levels prior to beginning therapy.

c. Instruct the client to take oral medication with 6 to 8 oz of water at least 30 minutes (60 minutes for ibandronate [Boniva]) before breakfast or any medication for the day. Advise the client to remain standing or sitting upright for 30 minutes (60 minutes for ibandronate) after taking medication. No food should be ingested for 30 minutes (60 minutes for ibandronate) after taking the medication. Precautions should be used for clients who have upper gastrointestinal (GI) side effects.

d. Studies support the efficacy of treatment with bisphosphonates for up to 5 years. After 5 years of treatment, reassess the treatment options. Treatment beyond 5 years of bisphosphonate therapy is highly individualized.

5. Receptor activator of nuclear factor kappa B ligand (RANKL) inhibitor:

a. Acts by inhibiting osteoclast formation, decreasing bone resorption, and increasing bone density:

i. Denosumab (Prolia) 60-mg subcutaneous injection once every 6 months. Clients with chronic kidney disease and/or those at risk of hypocalcemia should have a serum calcium level checked 10 to 14 days after the administration.

ii. Denosumab is recommended as an alternative initial treatment option for postmenopausal females with osteoporosis and high risk for osteoporotic fracture.

6. Selective estrogen receptor modulator (SERM):

a. Acts by decreasing bone resorption, increasing bone density, and decreasing risk of fractures.

i. Raloxifene (Evista): 60 mg orally daily.

ii. Long-term use of raloxifene may reduce risk of breast cancer but increase risk of venous thromboembolism and increased side effects of vasomotor symptoms including hot flashes.

7. Calcitonin:

a. Calcitonin is not the first-line treatment for osteoporosis. Studies reveal it may be less effective when compared with bisphosphonate and PTH therapy. Long-term use also has concerns for increased incidence of malignancy.

8. Medications for rebuilding bone:

a. PTH: Recombinant human PTH rebuilds bone density and increases strength of bone. Approved for severe osteoporosis for postmenopausal females and males with a diagnosis of osteoporosis who have failed antiresorptive therapy and are not able to tolerate bisphosphonates. See package insert for contraindications.

b. Teriparatide (Forteo) 20 mcg prefilled, 28 day pen/syringe; subcutaneous injection daily: This is taken for a maximum of 2 years. Check calcium and renal function before prescribing. It is contraindicated in clients with a history of kidney stones and history of radiation therapy. See package insert.

c. Abaloparatide (Tymlos): This medication is similar to PTH and can only be taken for a maximum of 2 years.

d. Romosozumab (Evenity): This is the newest treatment for osteoporosis on the market for rebuilding bone, and is given by injection monthly at the provider's office and can only be taken for a maximum of 1 year.

9. Hormone replacement therapy (HRT).

Estrogen therapy and estrogen/progesterone therapy are available as tablets or transdermal patches and come in a wide variety of doses. HRT is approved for prevention of bone loss, but not approved for treatment of osteoporosis.

FOLLOW-UP

A. If the client is on calcium supplements, check urinary calcium excretion two times per year. If it is below 250 mg/d, nephrocalcinosis and the risk of renal stones are decreased.

B. For those with a T-score of −2.00 to −2.49 at any location or have any risk factors for bone loss, a repeat bone density testing is recommended every 2 years to evaluate effectiveness of medical plan.

C. For females 65 years and older with a baseline T-score of −1.50 to −1.99 at any location and have no risk factors for bone loss, DEXA is repeated every 3 to 5 years.

D. For females 65 years and older with a baseline T-score of −1.01 to −1.49 at any location and have no risk factors for bone loss, DEXA is repeated every 10 to 15 years.

CONSULTATION/REFERRAL

A. If fracture is suspected, consult with a physician.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Assessment and treatment for osteoporosis must be performed routinely to prevent the risk of fracture, which may increase the risk of morbidity and mortality rates in this population.

2. The 10th Annual National Survey of Older Americans Act participants revealed that older adult participants with osteoporosis tended to have a lower self-rated health status and more physical limitations than those without osteoporosis. Studies exemplified that diet can help lessen the severity of osteoporosis in geriatrics. Older adults must have continual nutrition assessments (during physicals and/or regular visits) because undernutrition is a risk in this population.

3. Current literature review and meta-analysis suggests that recombinant human growth hormone treatment (low dose daily or 3x/wk) may be an option in preventing fractures in postmenopausal females 61 to 69 years old with osteoporosis and remained beneficial during the 10-year follow-up. Statistically, the therapy reduced fracture risk by 37%. Side effects have been reported as minor, and symptoms subsided after lowering the dose. When geriatric clients inquire about this therapy, collaborate/refer to an endocrinologist to help manage the treatment and monitor results.

4. The U.S. Preventive Services Task Force recommends that females ≥65 years of age undergo bone measurement testing for screening. Most clients do not know they have osteoporosis until they suffer a fracture.

5. It is important to remember to also assess males for osteoporosis. The mortality rate post fracture for geriatrics with osteoporosis was 18.7% for males and 13.9% for females. Counsel clients after the initial fragility fracture for safety and preventive measures with fall risks; implement shared decisions regarding treatment for osteoporosis. ▶

6. Recommended total calcium supplement/diet: 1,000 mg/d for males ≤70 years; 1,200 mg/d for females ≥50 years and males ≥70 years.

7. Although bisphosphonates are first-line treatment, it is contraindicated in clients with esophageal abnormalities and not recommended for those with creatinine clearance ≤30 to 35 mL/min. A routine dental assessment is not mandated before initiating bisphosphonate therapy; however, delay treatment a few months postdental surgery until mandibular and facial bones have healed.

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POLYMYALGIA RHEUMATICA

DEFINITION

A. Polymyalgia rheumatica (PMR) is an inflammatory condition, with an insidious or abrupt onset, that causes morning muscle stiffness, pain, and decreased range of motion (ROM), primarily in the hips, shoulders, and neck.

INCIDENCE

A. PMR is more common among people of European origin and is rarely seen in the African American, Asian, and Hispanic populations.

B. It is frequently seen in adults older than 50 years, with an increased incidence in the 70- to 80-year-old population. The incidence rates of those 50 years and older range from 58 to 96 per 100,000 annually in populations with predominantly European ancestry.

C. Females are affected two to three times more often than males.

D. PMR is occasionally associated with giant cell arteritis (GCA). Approximately 15% of diagnosed clients with PMR will also develop GCA. Approximately half of clients diagnosed with GCA will be diagnosed with PMR.

PATHOGENESIS

A. The cause of PMR is not well-understood. Environmental and genetic factors have been shown to play a role in PMR. Studies speculate that environmental triggers, such as viruses, may cause the onset of symptoms.

B. There are several similarities between PMR and GCA.

PREDISPOSING FACTORS

A. Age (older than 50 years).

B. Sex (females are affected two to three times more often than males).

C. Ethnicity (those of Northern European origin have a higher rate of PMR).

COMMON COMPLAINTS

A. Early morning symmetrical joint stiffness and pain, worse about 20 to 30 minutes after waking; onset of symptoms can range from a day to 2 weeks.

B. Stiffness and pain commonly occur in the shoulders, hips, and neck.

C. Decreased ROM of joints.

D. Muscle pain.

E. Weakness of joints and muscles.

OTHER SIGNS AND SYMPTOMS

A. Up to 50% will experience systemic symptoms.

1. Low-grade fever; high fever is uncommon and should suspect GCA.

2. Fatigue.

3. Malaise.

4. Anorexia.

5. Weight loss.

POTENTIAL COMPLICATIONS

A. Difficulty performing daily activities, such as getting up out of a chair, dressing, and bathing.

B. Impaired sleep and decreased quality of life.

C. Overall, a decrease in general health due to limitations.

SUBJECTIVE DATA

A. Ask the client if there was an activity that brought about or preceded the episode of joint pain.

B. Inquire about any recent illness or injury.

C. Ask the client to describe the onset, duration, and intensity of pain, noting what particular joints are involved.

D. Inquire about the duration of pain and stiffness in the mornings.

E. Does the client notice a decrease in ROM of joints? Are there activities that the client is unable to perform?

F. Ask the client to list all medications currently being taken, particularly substances not prescribed and over-the-counter products. What medications improve pain?

G. Has the client noticed a problem with sleeping since the symptoms began?

PHYSICAL EXAMINATION

A. Check vital signs: temperature, pulse, respirations, and blood pressure (BP).

B. Inspect:

1. Hands, wrists, elbows, shoulders, hips, and neck for erythema and synovitis.

C. Auscultate:

1. Heart.

2. Lungs.

D. Palpate:

1. Palpate shoulders, neck, and hip joints for diffuse swelling and pain. Marked swelling is not usually seen in clients with PMR.

2. Assess ROM of the shoulders, hips, back, and neck. Active ROM may be restricted due to pain. Perform passive ROM of the shoulders and hips.

3. Assess for muscle tenderness of arms, thigh, and neck.

4. Assess muscle strength of upper and lower extremities and neck.

5. Assess temporal arteries for signs of inflammation; can also listen to temporal arteries for bruits.

DIAGNOSTIC TESTS

A. Complete blood count (CBC): normocytic anemia and thrombocytosis may occur.
B. Erythrocyte sedimentation rate (ESR): elevation.
C. C-reactive protein (CRP), noncardiac: elevation.
D. Rheumatoid factor (RF), antinuclear antibody (ANA), and anticyclic citrullinated peptide antibody: negative.
E. Liver function tests (LFTs): occasionally elevated.
F. X-rays, ultrasounds, or MRI of the affected joints.

DIFFERENTIAL DIAGNOSES

A. PMR.
B. Rheumatoid arthritis (RA).
C. GCA.
D. Fibromyalgia (symptoms would have been present for years).

PLAN

A. General interventions:
1. Treat the client with oral steroid therapy to improve symptoms, starting at a dose adequate to resolve symptoms. Treatment includes slowly tapering the steroids over a period of months to keep the client in remission.
B. Client teaching: *See Client Teaching Guide for this chapter, "Polymyalgia Rheumatica."*
1. Educate the client and family that PMR is primarily a self-limiting condition that will improve slowly over time.
2. Treatment will include several months of low-dose prednisone therapy, tapering slowly until completion of steroid therapy. Tapering slowly and patience will prevent recurrent attacks and rebound flares.
C. Pharmaceutical therapy:
1. Low-dose prednisone is used to treat the symptoms. Steroids should be given to relieve symptoms (starting prednisone dose may be 12.5–25 mg orally [PO] daily) and then tapered slowly to an oral dose of 10 mg/d equivalent within 4 to 8 weeks. Once in remission, continue to taper oral dose by 1 mg every 4 weeks until discontinuation. Gradually decreasing the steroid dose by very small increments over time has the best results, minimizing the possibility of relapse.

FOLLOW-UP

A. A follow-up appointment is recommended 2 weeks after the initial appointment.
B. If symptoms are improving with the steroid therapy, a follow-up appointment is recommended every 1 to 2 months until remission is achieved and the client has completed the steroid therapy. Treatment may take up to 1 year or longer.
C. Another condition to consider with the symptoms is remitting seronegative symmetrical synovitis with pitting edema. This condition presents with hand and distal extremity swelling, noting marked pitting edema in extremities. It is commonly seen in clients older than 50 years of age, with an acute onset of swelling and pain. Symptoms respond quickly to the use of low-dose steroids. Laboratory testing for RA is negative.
D. Clients on long-term steroids should have a bone mass density (BMD) performed to evaluate for osteoporosis.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. The American College of Rheumatology (ACR) classification criteria for PMR for older adults are as follows:
a. Age ≥50 years, bilateral shoulder pain, and abnormal CRP/ESR.
b. Morning stiffness that lasts ≥45 minutes = 2 points.
c. Limited hip ROM and pain = 1 point.
d. Absent RA factor and/or ANA = 2 points.
e. No peripheral joint pain = 1 point.
i. A total of ≥4 points has a great sensitivity for discriminating RA from PMR.
2. Tips for geriatric clients include: simplify daily routine, exercise in ways to help joint flexibility like tai chi or yoga, pain-coping strategies including meditation, sharing feelings with others, getting plenty of sleep, and relaxation techniques.

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PSEUDOGOUT

DEFINITION

A. An acute inflammatory condition primarily affecting the larger joints in which calcium pyrophosphate dihydrate crystals deposit in the connective tissues.

INCIDENCE

A. The incidence of acute attacks is about equal in both males and females.
B. Females diagnosed with osteoarthritis (OA) with calcium pyrophosphate dihydrate crystal deposition disease have a higher incidence.
C. Approximately 50% of cases occur in clients older than the age of 84 years, 36% occur during the ages of 75 to 84 years, and 15% occur in those 65 to 74 years old.
D. Joints affected: The knee is affected approximately 50% of the time; other joints affected include wrists, shoulders, ankles, feet, and elbows.

PATHOGENESIS

A. Pseudogout occurs from the crystal formation in the cartilage that is shed into the affected joint. Excessive cartilage pyrophosphate production leads to CPP accumulation, and CPP crystals are formed and deposited in the joint.

PREDISPOSING FACTORS

A. Older adults.
B. Joint trauma or surgery.
C. Hospitalization/illness.
D. Familial chondrocalcinosis.
E. Endocrine/metabolic disorders:
1. Gout.
2. Hyperparathyroidism.
3. Hemochromatosis.
4. Hypophosphatasia.
5. Hypothyroidism.
6. Hypomagnesemia.
7. Gitelman syndrome.
8. Hemosiderosis.

COMMON COMPLAINTS

A. Asymptomatic (may be visible on x-ray).
B. Acute attack of pain and swelling of affected joint, commonly in a large joint.
C. Erythema. ▶

D. Decreased range of motion (ROM) secondary to severe pain; inability to bear weight.

OTHER SIGNS AND SYMPTOMS

A. Fever.
B. Pain, swelling, and redness in multiple joints.

SUBJECTIVE DATA

A. Ask the client about the onset, progression, and duration of symptoms.
B. How many joints are affected?
C. Is this the first time this has occurred, or is this a chronic problem?
D. Ask the client to describe the onset of symptoms, in the order of occurrence.
E. In addition to today, has this occurred in other joints?
F. Does the client have a history of rheumatoid arthritis (RA), OA, or other types of arthritis?
G. Any previous injury to the joint?
H. What prior treatment has the client used for pain or fever?

PHYSICAL EXAMINATION

A. Check vital signs and temperature as indicated.
B. Inspect:
  1. Inspect the affected joint for erythema, edema, and synovitis.
  2. If erythema is present, note location of erythema and evaluate if erythema extends beyond joint area.
  3. When examining the joint, note facial grimaces and guarding with examination.
  4. If weight-bearing joint is affected, note the client's ability to bear weight.
C. Auscultate:
  1. Lungs and heart.
D. Palpate:
  1. Palpate affected joint, noting pain and swelling in the joint.
  2. Assess ROM in joint if possible.

DIAGNOSTIC TESTS

A. Complete blood count (CBC).
B. Erythrocyte sedimentation rate (ESR).
C. Serum C-reactive protein (CRP).
D. Comprehensive metabolic profile (CMP).
E. Magnesium level.
F. Thyroid-stimulating hormone (TSH).
G. Iron studies (iron, total iron-binding capacity, ferritin level).
H. Joint aspiration for synovial fluid. Microscope evaluation and analysis for crystals for definitive diagnosis.
I. X-ray of joint may demonstrate the extent of joint damage and reveal linear and punctate calcifications in articular hyaline or fibrocartilage, also known as chondrocalcinosis.

DIFFERENTIAL DIAGNOSES

A. Pseudogout: joint aspiration of synovial fluid recommended for diagnosis. Diagnosis made by crystals identified by polarized light microscopy or radiographic changes of CPP crystals noted in the tissue or synovial fluid.
B. Gout.
C. Septic arthritis.
D. RA.
E. OA.

PLAN

A. General interventions:
  1. Acute attacks should be treated with rest, immobilization, and education.
  2. Differentiate between acute and chronic attacks and treatment.
  3. Recurrent attacks should have long-term management.
B. Client teaching:
  1. For acute attacks, encourage rest and elevation of affected joint.
  2. Encourage no weight-bearing until symptoms subside. Suggest the use of crutches or cane for assistance.
  3. Cool compress may be used as needed for comfort.
  4. As symptoms improve, suggest ROM exercises for affected joint.
  5. Once symptoms have improved, weight-bearing activities may resume.
  6. Symptoms should continue to improve over the next 7 to 10 days. Full recovery should be expected.
C. Pharmaceutical therapy:
  1. For acute attack, with one to two joints affected and no signs of infection:
    a. Large joints (knees/shoulders): joint aspiration and glucocorticoid steroid injection: triamcinolone acetonide 30 to 40 mg mixed with 1% procaine, 1 to 2 mL. Smaller joints require less steroid.
  2. If not meeting the criteria discussed earlier, the next treatment option recommended is the use of nonsteroidal anti-inflammatory drug (NSAID) medications:
    a. Naproxen (Naprosyn) 500 mg BID with food.
    b. Indomethacin (Indocin) 50 mg TID with food (not recommended in high-risk clients with heart failure, gastrointestinal [GI] disease, or decreased renal function).
    c. Sulindac 200 mg BID with food.
  3. If NSAIDs are contraindicated, use colchicine 1.2 mg within 24 hours of attack, followed by 0.6 mg 1 hour later, then 0.6 mg BID.
  4. If injection and use of NSAIDs are contraindicated, use oral prednisone 30 to 50 mg daily until flare begins to resolve, then taper prednisone over the next 7 to 10 days.
  5. Recurrent attacks (more than three attacks in 1 year) should be treated with colchicine. Monitor liver and kidney function. Consult with a physician and/or refer to a rheumatologist.

FOLLOW-UP

A. The client should return to the office in 48 to 72 hours after diagnosis and treatment.
B. Client follow-up in 1 week is recommended for continued care.
C. If symptoms are not improving or are worsening, client should return to the clinician's office or present to the ED for further evaluation and treatment.

CONSULTATION/REFERRAL

A. Refer all clients who appear to have a septic joint to the ED for evaluation and treatment. Refer to an orthopedic surgeon for consult.
B. Consider referring clients with pseudogout to rheumatology for evaluation and continued care.

INDIVIDUAL CONSIDERATIONS

A. Adults:
  1. Most commonly seen in adults older than 60 years.
  2. Precautions should be used in treating clients with long-term NSAIDs. Active GI ulcers or history of GI disease should avoid NSAID use. ▶

3. Clients on blood thinner medications should avoid the use of NSAIDs.
4. All precautions for the use of NSAIDs in adults should be considered, including chronic kidney disease.
5. Clients diagnosed with diabetes who are prescribed oral or injection steroids should be advised that steroids may increase blood sugar and to monitor blood sugar values at home as indicated.

B. Geriatrics:
1. Most common in the older adult.
2. Precaution should be used with using NSAIDs in the older adult.
3. Monitor renal function.
4. Monitor GI ulcers and bleeding.
5. Diet modifications to avoid salt, table sugar, corn syrup and sugary drinks, and high purine foods.
6. Encourage good hydration with water.

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PSORIATIC ARTHRITIS

DEFINITION

A. Psoriatic arthritis (PsA) is a complex inflammatory condition of the joints that presents with symptoms of joint pain, stiffness, and swelling around the tendons and ligaments. Other symptoms include psoriasis and inflammatory back pain. PsA is a chronic disease and, if left untreated, can cause irreversible joint damage.

B. Five different types of PsA:
1. Symmetrical polyarthritis.
2. Asymmetric oligoarthritis (fewer than five small joints and/or large asymmetric joints involvement).
3. Distal interphalangeal (DIP) joint involvement.
4. Arthritis mutilans (destructive and deforming, causes resorption of the phalanges).
5. Axial arthritis (spondyloarthritis).

C. Based on the Classification of Psoriatic Arthritis (CASPAR) study, classification criteria were developed in 2006 to assist in diagnosing clients with PsA. The client must score at least three points from the following list:
1. Skin psoriasis:
a. Present: 2 points.
b. History of skin changes: 1 point.
c. Family history of psoriasis if client is not affected: 1 point.
2. Nail lesions (onycholysis, pitting): 1 point.
3. Dactylitis (present or past): 1 point.
4. Rheumatoid factor (RF): negative: 1 point.
5. X-ray evidence of new bone formation near a joint: 1 point.

INCIDENCE

A. Occurs in approximately 1% to 2% of the U.S. population.
B. It is estimated to occur in approximately 4% to 30% of clients diagnosed with psoriasis.
C. Incidence rate of approximately 6 per 100,000 per year.

PATHOGENESIS

A. Individuals are genetically susceptible to PsA. It is thought that the immune system is precipitated by environmental triggers, which may include infections and/or traumas. The immune system is stimulated, activating T-cells that produce inflammatory cytokines and mediators that attack the joints, causing inflammation, pain, and/or erosions that can potentially destroy the joint. This response occurs in the joint and causes damage to the ligament and/or tendon attachment area occurs (enthesis), as well as produces bone spurs and changes in the skin and/or nails.

PREDISPOSING FACTORS

A. Sex: Males and females are affected equally.
B. Commonly seen in adults ranging from 30 to 55 years of age.
C. The client has a first-degree relative with psoriasis.
D. More common in Caucasians than African American and Native American populations.

COMMON COMPLAINTS

A. Pain and stiffness in joints, usually lasting longer than 30 minutes in the morning after wakening.
B. Pain and stiffness that usually improve with activity.
C. Enthesopathy (pain and inflammation at the ligament or tendon attachment site of the bone, commonly seen on the Achilles tendon, plantar fascia, or tibial tuberosity area).

OTHER SIGNS AND SYMPTOMS

A. Skin and nail psoriasis, nail pitting.
B. Uveitis.

SUBJECTIVE DATA

A. Does the client have a history of skin rash, plaques, and so forth? If present, ask the client when psoriasis began. What treatments have been used, including topical, light therapy, oral, and subcutaneous treatments?
B. Does the client complain of photosensitivity?
C. Ask the client to describe joint changes, such as swelling, pain, redness, and so forth. What joints are affected with pain and swelling?
D. Have any treatments used for psoriasis improved joint pain?
E. What medications or treatments are being used for joint pain and swelling? What is the result of medications/treatments being used?
F. Does the client have a history of back pain? If so, when did symptoms begin? Ask the client to describe their back pain. Does pain improve or worsen with physical activity?
G. Does the client have a history of frequent infections or high fevers?
H. Any history of frequent eye infections?

PHYSICAL EXAMINATION

A. Check vital signs and temperature as indicated.
B. Inspect:
1. Inspect the skin for plaques or rash.
2. Inspect the nails for pitting.
3. Inspect the joints for inflammation, synovitis, and erythema.
4. Inspect the eyes for erythema and injection.

C. Auscultate:
1. Heart.
2. Lungs.

D. Palpate:
1. Palpate all joints (hands, wrists, feet, elbows, ankles, knees, and shoulders) for synovitis/pain.
a. Dactylitis (swelling of the entire finger/toe that appears as a "sausage digit") occurs in approximately 50% of clients diagnosed with PsA. ▶

b. Enthesopathy is commonly seen in the Achilles tendon, plantar fascia, or tibial tuberosity.

2. Note range of motion (ROM) of all joints.

3. Palpate lumbar/thoracic spine.

4. Palpate sacral/iliac joint for tenderness.

DIAGNOSTIC TESTS

A. Erythrocyte sedimentation rate (ESR): may be elevated or normal.

B. C-reactive protein (CRP): may be elevated or normal.

C. Complete blood count (CBC).

D. RF: commonly negative in these clients; only 2% to 16% of clients with PsA will have a positive RF.

E. X-rays of affected joints.

DIFFERENTIAL DIAGNOSES

A. PsA.

B. Rheumatoid arthritis (RA).

C. Ankylosing spondylitis (AS).

D. Osteoarthritis (OA).

E. Gout.

F. Reactive arthritis.

PLAN

A. General interventions:

1. Early identification is necessary to prevent irreversible joint damage.

B. Client teaching:

1. Nonpharmacologic treatment options include the following:

a. Exercise on most days of the week. ROM exercises are encouraged for joint stiffness.

b. Heat application for stiffness and ice to affected joint for swelling may be used.

c. For extended joint stiffness/pain, recommend physical therapy for assessment and treatment.

C. Pharmaceutical therapy:

1. First-line treatment: The European League Against Rheumatism (EULAR) recommends the use of nonsteroidal anti-inflammatory drugs (NSAIDs) for first-line treatment. Precautions should be given for all clients using NSAIDs (cardiovascular, renal, and gastrointestinal [GI] risks should be considered). NSAIDs are used for mild symptoms.

2. First-line adjunctive therapy: Local glucocorticoid injections may be considered for a short period of time.

3. Second-line treatment: Disease-modifying antirheumatic drugs (DMARDs), such as methotrexate (MTX), sulfasalazine, and leflunomide, are used. These medications should be prescribed by a rheumatology specialist.

4. Third-line treatment: This includes biologic tumor necrosis factor (TNF) inhibitors (adalimumab, certolizumab pegol, etanercept, golimumab, and infliximab), and these medications should be prescribed by a rheumatology specialist.

FOLLOW-UP

A. Follow-up is recommended in 2 weeks if prescribing NSAIDs for treatment of early disease.

B. Recommend follow-up every 3 months if not referred to rheumatology.

CONSULTATION/REFERRAL

A. Clients diagnosed with PsA who are not controlled with NSAIDs or who have evidence of joint changes should be referred to a rheumatology specialist for evaluation and treatment. NSAIDs do not prevent joint damage, and prevention of irreversible joint damage is imperative.

B. Clients presenting with uveitis should be referred to an ophthalmologist for evaluation and treatment.

C. Clients diagnosed with severe joint damage should be referred to orthopedics for evaluation and possible surgical options.

D. Clients diagnosed with severe skin psoriasis should be referred to dermatology for evaluation and treatment.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. The use of oral steroids is not recommended for clients with PsA. Oral steroids have not been found to be effective; they also present a high risk of skin psoriasis flare when tapering the steroid dose.

2. Clients diagnosed with PsA are at higher risk for cardiovascular disease (CVD) and should be screened and monitored annually.

B. Geriatrics.

1. When prescribing NSAIDs, monitor renal, cardiac, and GI systems for adverse events with use.

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RAYNAUD PHENOMENON

DEFINITION

A. Raynaud phenomenon (RP) is an idiopathic disease in which an exaggerated vascular response occurs in extreme circumstances (heat, cold, and stress). It is manifested by bilateral blanching of the skin that is well-demarcated, discomfort in the fingers, then cyanosis, and eventually erythema after warming the digits.

B. Cold hands and feet are very common complaints. RP involves both cutaneous color change and cool skin temperature. Although the hands are the most common area of attacks, RP can also occur in the toes, ears, nose, face, knees, and nipples.

C. A Raynaud attack typically begins in a single finger and spreads symmetrically; however, the thumb is often spared.

D. RP may be either primary or secondary. Spontaneous remission may occur with primary RP.

E. Clinical characteristics associated with primary RP:

1. Symmetric episodic attacks.

2. No evidence of peripheral vascular disease.

3. No tissue gangrene, digital pitting, or tissue injury.

4. Negative nailfold capillary examination.

5. Negative antinuclear antibody (ANA) test.

6. Normal erythrocyte sedimentation rate (ESR).

F. Clinical characteristics associated with secondary RP: ▶

1. Age of onset older than 40 years.
2. Painful severe attacks with signs of ulceration/ischemia.
3. Ischemic signs/symptoms proximal to the fingers or toes.
4. Asymmetric attacks.
5. Abnormal laboratory, suggesting vascular or autoimmune disorders.

INCIDENCE

A. 3% to 20% in females.
B. 3% to 14% in males.
C. 3% in African Americans.
D. Wide global differences from the United States.

PATHOGENESIS

A. Primary RP: This occurs alone, without any other disease process. Speculated theories include digital vasospasm due to increased response of alpha-2 adrenergic receptors and a high sympathetic vascular tone.
B. Secondary RP: Symptoms occur as a secondary manifestation from other diseases or products.
C. Symptoms may be unilateral and affect only one or two fingers. Secondary RP usually has a higher morbidity than the primary disease.
1. Autoimmune disorders (connective tissue disorders, sclerosis, systemic lupus erythematosus [SLE[, rheumatoid arthritis [RA], vasculitis, Sjögren syndrome, dermatomyositis, polymyositis, etc.).
2. Atherosclerotic diseases.
3. Hematologic disorders (polycythemia, cryofibrinogenemia, etc.).
4. Metabolic/endocrine disorders (diabetes mellitus, pheochromocytoma, myxedema).
5. Neoplastic syndromes (lymphoma, leukemia, polycythemia, monoclonal/type 1 cryoglobulinemia).
6. Infections (hepatitis B, hepatitis C, *Mycoplasma* infections).
7. Environmental exposures/neurologic changes (frostbite, vibratory injuries from use, lead exposure, vinyl chloride exposure, arsenic exposure, organic solvents [xylene, toluene, acetone, chlorinated solvents], carpel tunnel syndrome).
8. Certain medications/drugs (cyclosporines, antineoplastics, oral contraceptives, narcotics, ergot alkaloids, bromocriptine, beta-adrenergic blocking agents, and nicotine).

PREDISPOSING FACTORS

A. Primary RP:
1. Female.
2. Onset of symptoms after menarche (15–30 years).
3. Smoking.
4. Emotional stress.

B. Secondary RP:
1. Onset after age 40 years.
2. Male.

C. Family history: multiple family members.
D. Frostbite.
E. Vascular trauma (distal ulnar artery).
F. Vibration-induced/occupation exposure (jackhammers, pneumatic drills, weed eaters).
G. Medication-associated RP (Table 22.1).

TABLE 22.1 DRUGS THAT MAY INDUCE RAYNAUD PHENOMENON

Amphetamines	Clonidine	Interferon-Alpha
Beta-blockers	Cocaine	Nicotine
Bleomycin	Cyclosporine	Vinblastine
Cisplatin	Ergot	Vinyl chloride

COMMON COMPLAINTS

A. Paleness of the fingertips after exposure to cold temperatures, followed by redness and discomfort after warming fingers.
B. "White attack": sharp, demarcated color of skin pallor.
C. "Blue attack": cyanotic skin.
D. White or blue attack, usually lasting 15 to 20 minutes.
E. Reactive hyperemia: erythema and reperfusion in recovery.
F. Age of onset between 15 and 30 years.

OTHER SIGNS AND SYMPTOMS

A. Paresthesias and numbness.
B. Clumsiness of the aching hand/finger.
C. Loss of pulp in pads of fingers (severe cases).

SUBJECTIVE DATA

A. Determine the age of onset, time, duration, and course of presenting symptoms.
B. Question the client regarding location and symptoms, noting blanching, followed by erythema and pain after hands are warmed.
C. Note frequency and severity of attacks.
D. Review the presence of any other skin alterations that have occurred. Does client also note skin mottling of the arms and legs? Livedo reticularis is a lilac or violet mottling or reticular pattern that occurs during a cold response.
E. Ask the client to identify any events that precipitate occurrences and what makes symptoms worse or better:
1. Air conditioning.
2. Grocery cold/freezer food sections.
3. Cold weather.
4. Cold water.
5. Emotional stress.
6. Sudden startling.

F. Identify any other symptoms that occur at the same time, such as migraine headaches.
G. Review the client's complete health history for underlying disorders, such as hypothyroidism and autoimmune disorders/connective tissue disease.
H. Review current or past occupations (especially those that include the use of vibratory tools).
I. Review current medications, stimulants, herbals, and over-the-counter (OTC) medications.

PHYSICAL EXAMINATION

A. Check pulse, respirations, and blood pressure (BP).
B. Inspect:
1. Dermal examination: Note malar/petechial rash, telangiectasias, digital pallor, or erythema. Examine for any ulceration or signs of ischemia.
2. Inspect joints for swelling or redness and overall ischemic changes. ▶

3. Examine several fingernails using a microscope or ophthalmoscope; examine capillaries at nail fold.
   a. Normal: fine red capillaries, lined in the same direction.
   b. Abnormal: capillaries dilated, tortuous, and irregularly spaced; avoid using the index finger for evaluation.

C. Auscultate:
1. Heart.
2. Lungs.

D. Palpate:
1. Palpate the joints for tenderness and peripheral pulses bilaterally.

E. Neurologic examination:
1. Sensory function:
   a. Sensory discrimination (hot/cold, sharp/dull).
   b. Location of sensation (proximal/distal to previous stimuli).
   c. Vibratory sensation with tuning fork (distal to proximal joints).
   d. Graphesthesia (draw a number or letter in the palm of the hand with a blunt object, such as a pencil, applicator stick, or pen and have the client identify the letter/number).

DIAGNOSTIC TESTS

A. There is no gold standard diagnostic test.

B. History alone is accepted as diagnostic as no office test application consistently triggers an attack. A history of at least two color changes, pallor, and cyanosis after cold exposure is adequate for the diagnosis of RP. Ask the client to take a picture when symptoms occur and bring it into the office for evaluation.

C. The cold water challenge test is no longer recommended.

D. Tools to assess vascular response (usually not readily available):
1. Nailfold capillaroscopy.
2. Videomicroscopy.
3. Thermography.
4. Angiography.
5. Laser Doppler.
6. Direct measures of the skin temperature and local blood flow.

E. Laboratory tests:
1. ANA.
2. ESR.
3. Thyroid profile if hypothyroidism is suspected.
4. Other tests are used, depending on suspected etiology; testing should be guided by results of history and physical examination:
   a. Complete blood count (CBC).
   b. Chemistry profile with renal and liver function.
   c. Urinalysis.
   d. Rheumatoid factor (RF).
   e. Complement (C3 and C4).
   f. ANA.

DIFFERENTIAL DIAGNOSES

A. RP (primary vs. secondary).

B. Scleroderma.

C. SLE.

D. Occupational trauma.

E. Medication-induced.

F. Peripheral vascular disease.

G. Neurovascular processes (diabetes, atherosclerosis, thromboangiitis obliterans).

PLAN

A. General interventions:
1. If ulcerations are present, monitor for secondary infections. Consider topical/systemic antibiotics if secondary infection. Debridement may be necessary.
2. Biofeedback and relaxation techniques are frequently used for treatment.

B. Client teaching:
1. Stress the importance of not smoking.
2. Keep the body warm:
   a. If in extreme temperatures, wear extra clothing (thermal underwear) to maintain temperature.
   b. Wear mittens instead of gloves to protect hands and keep them warm.
   c. Wear a hat to conserve heat.
3. If possible, stop all medications that could induce symptoms. Other drugs that should be avoided include the following:
   a. Decongestants and stimulants.
   b. Herbs that contain ephedra.
   c. Medications used for migraine headaches; for example, serotonin agonists such as sumatriptan, or caffeine plus ergotamine.
4. If vibration injury is present, stop the repetitive activity that induces symptoms. Consider alternative methods of work. If unable to totally stop the activity, reduce time spent using the vibratory equipment.
5. Emotional stress can be a trigger due to the vasoconstriction of the sympathetic nervous system being triggered. Counsel the client regarding stress management and treatment (nonpharmacologic/pharmacologic) options that may be beneficial.

C. Pharmaceutical therapy:
1. Therapy may be required only during the winter months.
2. Slow-release or long-acting dihydropyridine calcium channel blockers are used. Doses may be adjusted every 4 weeks or as tolerated. Monitor BP and evaluate adverse effects of hypotension: headaches, dizziness, flushing tachycardia, and peripheral edema.
   a. Nifedipine 30 to 180 mg/d.
   b. Amlodipine 5 to 20 mg/d.
3. Low-dose aspirin antiplatelet therapy 75 to 81 mg/d may be considered in secondary RP with a history of ischemic ulcers or other thrombotic events. Clopidogrel or dipyridamole can be used if aspirin is contraindicated.
4. Vasodilators (sildenafil) or endothelin receptor antagonists (bosentan) may be useful in refractory cases with associated digital ulcers/infarcts. However, these medications are not Food and Drug Administration (FDA)-approved for this treatment.
5. Topical nitrates are alternative treatment options. Nitroglycerin 2% ointment may be applied to affected areas. Topical ointments may result in adverse effects of hypotension and dizziness.

D. Surgical therapy (only recommended for clients resistant to initial therapies):
1. Temporary sympathectomy involving a local chemical block with lidocaine or bupivacaine (without epinephrine) relieves the pain.
2. Chemical and cervical sympathectomy may be used for severe cases with digital ischemia for temporary measures.
3. Vascular reconstruction is an option.

▶

FOLLOW-UP
A. Follow up in 1 month or as needed given client symptoms.

CONSULTATION/REFERRAL
A. Consult a physician if signs of ischemia are present and/or if refractory symptoms are present.
B. Refer to a rheumatologist if there is a moderate/high suspicion of secondary RP.
C. Refer for vascular surgical therapies if needed.

INDIVIDUAL CONSIDERATIONS
A. Adults:
1. The onset of RP after 40 years of age is commonly associated with an underlying disease.
B. Geriatrics:
1. Recommend geriatric clients to wear gloves and socks in cold weather and have jackets/sweaters for sudden temperature changes, such as outdoor settings, restaurants, or freezer section of grocery stores.
2. When RP occurs, instruct older adults to protect aging, thinning skin, and make sure to rewarm hands with *moderately* warm water or by placing hands in the axilla area.
3. Older adults are particularly susceptible to triggers causing vasoconstriction, such as vibratory tools, decongestants, herbs/medications, emotional stress, and secondhand smoke.
4. Due to neuropathies and cognitive decline, some older adult clients may not be aware of the changes with fingers, which could increase risk for delayed diagnosis. If ulcerations occur, clinical guidelines indicate intravenous (IV) antibiotics. Severe cases will require surgical intervention.

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RHEUMATOID ARTHRITIS

DEFINITION
A. Rheumatoid arthritis (RA) is a chronic systemic disease that involves polyarticular inflammation of the joints. The disease onset is generally insidious, with a slow development of stiffness and tenderness of one or more joints over a period of weeks to months and eventually leading to the involvement of multiple symmetrical joints. Typically, inflammatory changes in the interphalangeal joints of the fingers and thumbs are noted; however, other joints involved include the elbows, shoulders, ankles, knees, and toes. Left untreated, the client is at high risk of joint deformity and disability. Treatment goals of RA include remission, preventing functional decline of the client, and halting progression of the disease.
B. RA is not limited to the joints; extraarticular features of RA include anemia, pleuropericarditis, neuropathy, myopathy, splenomegaly, Sjögren syndrome, scleritis, vasculitis, and renal disease. Most clients with extraarticular symptoms also have the classic RA joint symptoms. Clients with RA are at increased risk of development of carpal tunnel syndrome, stroke, an osteoporotic fracture, and renal disease (secondary to drug toxicity).
C. The American College of Rheumatology (ACR) criteria for RA uses a weighted scoring system to classify new presenting clients as having RA. A score ≥6/10 indicates a need to evaluate the client for RA. Individuals who have had at least one swollen joint without other known etiology should be screened as early diagnosis and treatment reduces the risk of joint complications associated with RA. Scoring for the RA criteria is available on the ACR website at www.rheumatology.org/Practice-Quality/Clinical-Support/Criteria.
D. Classification criteria for screening for RA:
1. Duration of symptoms for more than 6 weeks.
2. Joint swelling involvement (synovitis).
3. Serology: rheumatoid factor (RF) and/or anti-citrullinated peptide/protein antibody (CCP).
4. Acute-phase reactants: C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR).
E. Four stages of RA:
1. Stage 1: no symptoms or signs; normal activity; RF and/or CCP antibody are present.
2. Stage 2: morning stiffness, warmth at joint, normal activities of daily living (ADLs), minimal limitation in joint use; increased T-cells, B-cells, antibody production, and synovial cells are observed.
3. Stage 3: morning stiffness, warmth at joint, and extraarticular manifestations; marked limitation in ADL. Increased T-cells, B-cells, antibody production, and synovial cells are observed.
4. Stage 4: same as stage 3 plus proliferating synovial membrane involved, causing injury to the bone, tendons, and cartilage. Client is now incapacitated or confined to a wheelchair.

INCIDENCE
A. It is estimated that RA occurs in approximately 1.5 million adults in the United States. RA occurs at twice the rate in females compared with males. The prevalence of RA is approximately 1.06% in females compared with 0.61% in males.

PATHOGENESIS
A. Although the cause is unknown, RA is thought to be a result of predisposing genes, autoimmune, and environmental triggering events such as infections and toxins. Inflammatory cells proliferate in the synovial joints, causing abnormal tissue antibody production and ultimately joint destruction.

PREDISPOSING FACTORS
A. Family history, including 15% prevalence in monozygotic twins.
B. Female.
C. Ages 30 to 50 years.
D. Cigarette smoking (strongest environmental trigger).
E. Recent systemic illness or trauma.

COMMON COMPLAINTS
A. Joint pain.
B. Morning stiffness in joints for at least 1 hour that has been present for more than 6 weeks.
C. Swelling and warmth in at least three joints or more for at least 6 weeks. Common joints affected include the wrists and hands, the metacarpal phalangeal (MCP) joints, and the proximal interphalangeal (PIP) joints.
D. Symmetrical involvement of the affected joints.

OTHER SIGNS AND SYMPTOMS
A. Fatigue.
B. Malaise.
C. Subcutaneous nodules.
D. Joint deformities: ▶

1. PIP joints: boutonniere deformities.
2. Fingers: swan-neck contractures, ulnar deviation.
3. Wrists: loss of extension.
4. Hips: loss of internal rotation, followed by flexion contractures.
5. Knees: suprapatellar pouch distension.
6. Elbows: decreased extension, olecranon bursitis.
7. Shoulders: limited range of motion (ROM).
8. Cervical spine: subluxation rare.
9. Temporomandibular joint: pain with biting.

E. Depression.
F. Low-grade fever.
G. Weight loss.
H. Myalgia.
I. Anemia.
J. Carpal tunnel syndrome.
K. Bone loss.

SUBJECTIVE DATA

A. Review when joint pain began and identify the joints involved. Are pain and stiffness worse in the morning? How long does it last? Is it symmetrical?
B. Elicit the client's description of pain and a description of how the pain interferes with ADLs (walking, climbing stairs, using the toilet, getting up from a chair, opening a jar).
C. Review family history of RA.
D. Has the client ever had any type of injury to the specific joint area? Rule out recent injuries.
E. Identify what makes the pain worse and alleviating factors. In clients with RA, activity typically alleviates symptoms, which is indicative of inflammation.
F. Review list of medications, including herbal and over-the-counter (OTC) medications. What therapies have specifically been used and what were the results?
G. Review the client's history for recent infections.
H. Does the client use any assistive devices, including cane, crutches, walker, wheelchair/power mobility device, kitchen devices/grips, and so forth?

PHYSICAL EXAMINATION

A. Check temperature, if indicated, pulse, respirations, blood pressure (BP), and weight.
B. General observation:
1. Observe the client getting up and down in the chair.
2. Observe the client walking (may tend to bear weight on heels and hyperextend toes secondary to tenderness to the metatarsophalangeal joints).
3. Observe the client handling objects in the hands.
4. Observe for signs of depression.

C. Inspect:
1. Inspect all joints, noting deformities, erythema, and temperature. (Heat and redness are not prominent features of RA.)
2. Evaluate for pitting edema in the hand (may have a "boxing glove" appearance).
3. Inspect for subcutaneous rheumatoid nodules (elbow is the most common site).
4. Evaluate skin for ulcerative lesions (secondary from venous stasis and neutrophilic infiltration), skin atrophy and ecchymoses from glucocorticoids, and petechiae (side effect from medications causing thrombocytopenia).
5. Eye examination:
   a. Episcleritis: acute redness and pain without discharge.
   b. Scleritis: deep ocular pain with dark red discoloration.

D. Auscultate:
1. Auscultate the heart.
2. Auscultate the lungs (at risk of infectious complications from immunosuppression and pulmonary toxicity from methotrexate [MTX], if currently being treated with medications).

E. Percuss:
1. Perform a patellar tap to evaluate synovial thickening/effusion of the knee.

F. Palpate:
1. Palpate the lymph nodes.
2. Perform oral examination to palpate the salivary glands (may have lymphocytic infiltration).
3. Palpate all joints to evaluate for tenderness with pressure, pain with movement of the joint, and "bogginess" of the joint (synovial thickening).
4. Palpate the popliteal fossa for evidence of a popliteal (Baker) cyst.
5. Examine abdomen for splenomegaly.

G. Musculoskeletal examination:
1. Assess grip (a reduced grip is a useful parameter in evaluating disease activity and progression). Is the client able to close fingers to make a fist?
2. Assess the strength of the extremities.
3. Assess the ROM (active and passive).

DIAGNOSTIC TESTS

A. Complete blood count (CBC) and platelets.
B. ESR.
C. CRP.
D. RF, quantitative.
E. CCP antibody.
F. Uric acid level.
G. Synovial fluid (optional).
H. Plain film radiography of the hands, wrists, and feet as a baseline and to monitor disease progression.

DIFFERENTIAL DIAGNOSES

A. RA.
B. Osteoarthritis (OA).
C. Psoriatic arthritis (PsA).
D. Crystalline arthritis (gout and pseudogout).
E. Polyarthritis.
F. Reactive arthritis.
G. Acute viral/infectious process:
1. Lyme disease.
2. Hepatitis B.
3. Hepatitis C.
4. Parvovirus B19.

H. Sjögren syndrome: keratoconjunctivitis sicca, splenomegaly, and lymphadenopathy.
I. Sarcoidosis.
J. Polymyositis.

PLAN

A. General interventions:
1. Focus on exercise and joint mobility to maintain functional abilities.
2. Encourage smoking cessation, especially among females on glucocorticoids due to the risk of increased bone loss/fracture.

B. Client teaching:
1. Once diagnosed, it is recommended that treatment with medications begin to prevent joint damage. First-line medications include disease-modifying antirheumatic drugs ▶

(DMARDs). Depending on what medication is used, educate the client regarding benefits, risks, side effects, and precautions for medications. If MTX is used, educate the client that alcohol should be avoided to prevent hepatotoxicity.

2. Stress the importance of returning for laboratory follow-up while on DMARDs.

3. Discuss vaccinations, especially pneumonia and influenza vaccines. Emphasize the importance of keeping all vaccinations up to date. Most treatment medications suppress the immune system and increase the risk of infections.

C. Pharmaceutical therapy:

1. Early initiation of DMARDs is important in reducing risk of joint complications. NSAIDs and/or glucocorticoids may be initiated for symptom control of inflammation while DMARDs take effect.

a. Timely referral to a rheumatologist as safety precautions and assessments is necessary before starting DMARDs.

i. MTX is given on a weekly basis starting with 7.5 to 15 mg/wk and is increased as tolerated to control symptoms. Titration: Increase the dose after 4 weeks at a rate of 2.5 to 5 mg/wk every month to a maximum dose of 25 mg/wk. MTX should not be given to clients who desire to become or who are pregnant, and clients with liver disease and severe renal impairment (estimated glomerular filtration rate [eGFR] <30 mL/min).

ii. Other DMARDs include sulfasalazine (Azulfidine) 500 to 1,000 mg is given daily initially; hydroxychloroquine (Plaquenil) 200 to 400 mg is given daily as maintenance. Before starting clients on hydroxychloroquine, the client must be counseled regarding the possible risks of the medication, including retinal damage that can lead to blindness. A baseline eye examination must be performed, followed by eye examination every 6 months to monitor for retinal changes; leflunomide (Arava) 10 to 20 mg/d as maintenance. Monitoring blood work while on leflunomide every 3 months is recommended.

b. Nonsteroidal anti-inflammatory drugs (NSAIDs) and pain-relieving medications for analgesic benefit:

i. Ibuprofen 600 to 800 mg every 6 to 8 hours as needed with food.

ii. Naproxen 500 mg orally Q12H as needed with food.

iii. Celecoxib (Celebrex) 200 mg PO daily with food.

iv. Acetaminophen (Tylenol) 500 to 1,000 mg orally every 4 to 6 hours as needed.

1) Avoid or caution use in comorbid conditions, such as a history of heart failure, renal disease, and peptic ulcers.

2) Examine all current medications, especially those prescribed by other providers and consider possible drug–drug interactions. Examples include NSAIDs, antacids, anticoagulants, oral hypoglycemic agents, antihypertensive/diuretics, lithium, MTX, and diphenylhydantoin (Dilantin).

c. Glucocorticoids for symptom management and control:

i. Oral glucocorticoids, prednisone 5 to 10 mg/d, may be added for active joint inflammation. Prednisone may be used up to 6 months, but shorter courses are recommended due to the consequence of long-term steroid use. Steroids should be tapered over a period of a few months and then completely discontinued if possible.

ii. Intraarticular long-acting glucocorticoid injections are used for reduction of synovitis in inflamed joints.

d. If response is not adequate, biologic agents, tumor necrosis factor (TNF) alpha inhibitors, and other classes of drugs may be instituted. Because of the associated side effects, rheumatology consult is recommended for treatment of these medications.

i. Examples of TNF inhibitors:

1) Infliximab (Remicade) 3 to 10 mg/kg every 4 to 6 weeks; intravenous (IV) infusion.

2) Adalimumab (Humira) 0.8 mL (40 mg) subcutaneous injection into the abdomen or thigh every 2 weeks.

3) Etanercept (Enbrel) 50 mg subcutaneous injection into the abdomen or thigh weekly.

4) Golimumab (Simponi) 50 mg subcutaneous injection monthly.

5) Certolizumab pegol (Cimzia) 200 mg subcutaneous injection every other week.

ii. Janus kinase inhibitor:

1) Tofacitinib citrate (Xeljanz) 5 mg PO BID or 11 mg PO daily.

iii. Interleukin-6 (IL-6) inhibitor:

1) Tocilizumab (Actemra) 162 mg subcutaneous injection given every 1 to 2 weeks or 4 to 8 mg/kg IV infusion every 4 weeks.

iv. T-cell depletion:

1) Abatacept (Orencia) IV infusion given every 4 weeks or subcutaneous injection weekly; dosage is based on weight.

v. B-cell depleting therapy:

1) Rituximab (Rituxan) 1,000 mg IV infusion given at week 0 and week 2, and then repeated approximately every 6 months or as indicated.

e. Clients may also use combination therapy. Examples of this include using MTX plus a TNF inhibitor or sulfasalazine, and so forth.

f. Clients receiving treatment with pharmacologic agents should be evaluated at routine intervals, every 3 months, regarding their functional status to identify whether treatment therapies are improving the symptoms. There are several functional forms that are available for use. The Stanford Health Assessment Questionnaire (HAQ) is a well-known questionnaire that is recommended for use.

g. Drug monitoring, by performing serum blood work, is also recommended at routine intervals when prescribing pharmacologic agents to avoid adverse effects of the medications being prescribed.

FOLLOW-UP

A. Follow-up will be guided by medication therapy. Disease activity and response to therapy should be reassessed every 4 to 6 weeks.

B. Certain medications require frequent monitoring. When prescribing MTX, blood work for liver function (alanine transaminase [ALT], aspartate transaminase [AST]), albumin, CBC with differential, platelets, and urinalysis is initially recommended monthly. ▶

C. Assume all clients with RA are at risk of osteoporosis. Dual-energy x-ray absorptiometry scan is used to evaluate bone loss secondary to glucocorticoid-induced osteopenia. Initiate bisphosphonate therapy as indicated for signs of bone loss. Use a low threshold for starting in postmenopausal females with RA.

D. Anti-TNF agents are contraindicated in clients with an active infection and those who are at high risk of reactivation of tuberculosis (TB). A TB skin test is required before administration. Clients with a positive skin test should be treated with prophylactic anti-TB therapy 1 month before therapy with anti-TNF agents.

E. Clients should receive a baseline ophthalmologic examination before starting antimalarial drugs, such as Plaquenil, and then follow-up examinations every 6 to 12 months while on therapy.

CONSULTATION/REFERRAL

A. Refer all clients with early inflammatory arthritis to a rheumatologist if RA is suspected. Early intervention may prevent bone destruction of the joints and improve the long-term outcome for the client.

B. Clients with a history of chronic swelling and pain of the joints should be referred to a rheumatologist.

C. Refer all clients to a rheumatologist, orthopedist, or ED if a septic joint is suspected.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. RA activity improves substantially in pregnancy.

a. Approximately 70% to 80% of clients will have symptoms improved during pregnancy.

b. Approximately 90% of females will have a flare in the postpartum period. Flares usually occur within the first 3 months postpartum.

2. Leflunomide, etanercept, adalimumab, and infliximab are contraindicated in pregnancy and while breastfeeding.

3. Pregnancy should be avoided with the use of MTX and Arava. Females need to have one normal menstrual cycle following discontinuation of MTX before attempting pregnancy. Males should wait at least 3 months after discontinuing MTX before attempting to conceive.

4. Therapy during pregnancy should be coordinated with the perinatologist and the rheumatologist.

B. Geriatrics:

1. Use NSAIDs with caution. Consider the client's age, weight, and chronic conditions when prescribing NSAIDs. NSAIDs are not recommended in clients with renal and gastrointestinal conditions.

2. Recommendations for DMARDs for geriatric population suffering with RA are first-line MTX with or without **nonbiologics** (leflunomide, hydroxychloroquine, sulfasalazine, and glucocorticoid) or **biologics** (TNF inhibitors, Actemra, Enbrel, Kineret, Rituxan, etc.). Use biologic DMARDs *only* if nonbiologics failed. Collaborate with a rheumatologist when managing clients with these medications.

3. Risks for elder population on MTX (solo/combination) are pneumonitis particularly within the first year and hepatotoxicity. Frequent monitoring of liver enzymes is recommended especially if client has a history of chronic/abusive alcohol use. Instruct geriatric clients (and surrogates) to be seen by their provider for evaluation of infection if cold or cough symptoms begin.

4. Clinical recommendations for geriatrics treated with glucocorticoids: Keep prednisone ≤15 mg/d with synergistic osteoporosis prevention measures and avoid use with clients experiencing delirium.

5. Low-dose prednisone (5–10 mg/d) has been effective as adjunct to MTX in geriatric population.

6. Combination of MTX and TNF increases potential for periodontal disease. Advise geriatrics to be consistent with regular dental checkups.

7. Although neuromuscular electrical stimulation (NES) and high-intensity resistance exercise (HIRE) training have been shown to effectively improve muscle structure and function in adults suffering from RA, thoroughly investigate geriatric comorbidities and their perception of the procedures. Many older adult clients cannot tolerate HIRE or it might be contraindicated. However, NES could be a possible alternative.

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SYSTEMIC LUPUS ERYTHEMATOSUS

DEFINITION

A. Systemic lupus erythematosus (SLE) is a chronic, inflammatory, autoimmune disorder and can affect any organ systems. The body's immune system forms antibodies that attack healthy tissues and organs. The clinical course is marked by spontaneous remission and relapses. Severity varies from a mild episodic disorder to a rapidly fulminating fatal disease. The three types of lupus are as follows:

1. Discoid lupus erythematosus (DLE) affects the skin, causing a rash, lesions, or both.

2. SLE attacks body organs and systems, such as joints, kidneys, brain, heart, and lungs. SLE is usually more severe than DLE and can be life-threatening.

3. Drug-induced lupus symptoms usually disappear when medication is discontinued.

INCIDENCE

A. About 85% of clients with SLE are females; it affects mainly young females after menarche and before menopause. The majority of clients who develop SLE during childhood or after age 50 years are also female. SLE occurs in 1:1,000 Caucasian females and 1:250 African American females. The disorder is concordant in 25% to 75% of identical twins. The risk of developing the disease if a mother has SLE is 1:40 for a daughter and 1:250 for a son. Positive antinuclear antibody (ANA) is seen in asymptomatic family members, and the prevalence of other rheumatic diseases is increased among close relatives of clients. There is a high frequency of specific genes in SLE. Of DLE clients, 10% go on to develop SLE.

PATHOGENESIS

A. The exact cause of SLE is unknown, but clinical manifestations of SLE are secondary to the trapping of antigen–antibody complexes in capillaries of visceral structures, or to autoantibody-mediated destruction of host cells such as thrombocytopenia. SLE is commonly precipitated by recent exposure to illness, infections, UV light, surgery, pregnancy, and stress.

PREDISPOSING FACTORS

A. Female.
B. African American, Asian, Hispanic ancestry.
C. Childbearing age.
D. Positive family history of SLE.
E. Drug use:
  1. Procainamide.
  2. Hydralazine.
  3. Isoniazid.

COMMON COMPLAINTS

A. Joint pain: Joint symptoms occur in 90% of clients.
B. Fever.
C. Loss of appetite.
D. Fatigue.
E. Weight loss.
F. Oral or nasal ulcers.
G. Hair loss.
H. Photosensitive skin rash and skin lesions over areas exposed to sunlight.
I. Relapsing polychondritis.

OTHER SIGNS AND SYMPTOMS

A. DLE:
  1. Rash: erythematosus, round, scaling papules 5 to 10 mm in diameter, appearing as "butterfly" shape across bridge of nose.
  2. Rash, commonly on the trunk, extremities, scalp, external ear, and neck.
  3. Photosensitivity.
B. SLE: The 1997 updated American College of Rheumatology (ACR) criteria for diagnosis must include four or more of the first 11 of the following criteria:
  1. Malar rash.
  2. Discoid rash.
  3. Photosensitivity.
  4. Oral ulcers.
  5. Arthritis (generally bilateral, symmetric, especially in hands and wrists).
  6. Serositis.
  7. Renal disorder: chronic renal problems as indicated by proteinuria (>0.5 g/d of protein or >3+ protein) or cellular casts.
  8. Neurologic disorder (seizures or personality changes, psychosis).
  9. Hematologic disorder.
  10. Immunologic disorder.
  11. ANA; abnormal titer of ANA.
  12. Other signs and symptoms:
    a. Weight loss.
    b. Fatigue.
    c. Acute abdominal pain.
    d. Alopecia.
    e. Tendon involvement.
    f. Urinalysis: active urine sediment (blood or protein without urinary tract infections [UTIs]).
    g. Fever and malaise.
    h. Lymphadenopathy.
  13. Other complications:
    a. Hashimoto thyroiditis.
    b. Hemolytic anemia.
    c. Thrombocytopenia purpura.
    d. Arterial and venous thrombosis in the presence of antiphospholipid antibodies.
    e. Recurrent pleurisy.
    f. Pleural effusion, pneumonitis.
    g. Pulmonary embolism.
    h. Pericarditis, endocarditis, myocarditis.
    i. Hypertension.
    j. Splenomegaly.
    k. Recurrent miscarriages in the presence of antiphospholipid antibodies.
    l. Relapsing polychondritis.

Probable SLE: have two or three of the first 11 ACR guidelines criteria, along with one other feature.

Possible SLE: have one of the first 11 ACR guidelines criteria, along with one other feature.

C. Central nervous system (CNS) problems:
  1. Chronic headaches (migraine).
  2. Seizures or epilepsy.
  3. Personality changes, chronic brain syndrome.
D. Decreased hemoglobin (Hgb), white blood cells (WBCs), and platelets.

SUBJECTIVE DATA

A. Determine systemic features and onset, course, and duration of symptoms (see "Other Signs and Symptoms" section).
B. Obtain medication history (see "Predisposing Factors" section).
C. Determine family history of rheumatoid diseases or other autoimmune disorders.
D. Review the client's recent history for possible allergen exposure.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure (BP).
B. Inspect:
  1. Observe general overall appearance and generalized movement of extremities.
  2. Conduct dermal examination for color, petechiae, rashes, lesions, and hair loss.
  3. Conduct funduscopic examination; note photosensitivity.

Funduscopic examination: Cotton–wool exudates are the most common eye lesion.

  4. Examine mouth and nose for oral lesions/ulcers.
  5. Observe for pericardial lifts and heaves.
  6. Inspect joints for subluxation of the metacarpal phalangeal joints and swan-neck deformities of the hands.
C. Palpate:
  1. Back for tactile fremitus; palpate the heart for lifts, heaves, and thrills.
  2. Neck for thyroid enlargement.
  3. Neck, axilla, and groin for lymphadenopathy.
  4. Abdomen for organomegaly, masses, and tenderness; palpate the suprapubic area for suprapubic tenderness and assess back for costovertebral angle (CVA) tenderness.
D. Percuss:
  1. Chest, anterior and posterior lung fields, for consolidation.
  2. Abdomen for splenomegaly.
E. Auscultate:
  1. Heart.

▶

2. Lungs.
3. Abdomen.

F. Musculoskeletal examination:
1. Examine for bone or joint swelling, tenderness, and increased warmth.
2. Check lower extremities for evidence of phlebitis, asymmetrical swelling, calf tenderness, and palpable cord.

G. Neurologic examination:
1. Complete neurologic examination with mental status examination.

DIAGNOSTIC TESTS

A. Complete blood count (CBC) with differential: leukopenia, anemia, and thrombocytopenia may indicate active disease.

B. Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP): may be elevated with disease activity.

C. ANA: indirect immunofluorescence assay (IFA) positive. In clients with SLE, the ANA will typically be positive with a high-titer ANA. Note pattern associated with positive ANA. A negative ANA by the IFA method dramatically reduces the risk of SLE.

D. Complement levels (decreased C3 and C4).

E. Rheumatoid factor (RF) can be positive in clients with SLE.

F. Thyroid profile.

G. Anti-double-stranded DNA antibodies: seen in approximately 30% of clients with renal disease.

H. Antiphospholipid antibodies: lupus anticoagulant immunoglobulin A (IgA), immunoglobulin G (IgG), and immunoglobulin M (IgM) anticardiolipin antibodies; IgA, IgG, and IgM anti-beta-2-glycoprotein.

I. EKG, lung function tests, imaging studies as indicated.

J. Urinalysis (check for hematuria, proteinuria, and cellular casts) and urine culture for kidney involvement.

K. Collection of 24-hour urine for protein and creatinine clearance.

L. Skin or renal biopsy.

M. Follow-up Coombs test: positive in approximately 10% to 20% of SLE clients.

N. Some tests for syphilis may be falsely elevated in clients with lupus; therefore, false-positive serologic test for syphilis needs follow-up. Microhemagglutination assay for *Treponema pallidum* antibodies (MHA-TP) can be used to confirm reactive syphilis for positive rapid plasma reagin (RPR) as it is highly specific.

O. Chest x-ray film may show changes in SLE clients with pleuritic chest symptoms.

P. X-ray of the joints for nondestructive arthritis.

DIFFERENTIAL DIAGNOSES

A. SLE.

B. DLE.

C. Undifferentiated connective tissue disease.

D. Drug-induced lupus.

E. Rheumatoid arthritis (RA): Lupus can resemble RA, especially early in the course of SLE. Unlike RA, the arthritis is nonerosive: There is no joint destruction.

F. Vasculitis.

G. Scleroderma.

H. Chronic active hepatitis.

I. Acute drug reactions.

J. Polyarteritis.

K. Infection.

L. Influenza.

M. Rosacea.

N. Neoplasm.

PLAN

A. General interventions.
1. After diagnosis, refer the client to a rheumatologist and comanage with a physician.

B. Client teaching: *See Client Teaching Guide for this chapter, "Systemic Lupus Erythematosus."*

C. Pharmaceutical therapy:
1. Nonsteroidal anti-inflammatory drugs (NSAIDs) for arthritis symptoms.
2. Prednisone 40 to 60 mg initially for control of thrombocytopenic purpura, hemolytic anemia, myocarditis, pericarditis, convulsions, and nephritis.
3. Always give the lowest dose that controls the condition.
4. Glucocorticoids ≤7.5 mg/d prednisone (or equivalent) may provide symptom relief. Long-term use of glucocorticoids can have many detrimental effects. Initiation of hydroxychloroquine (HCQ) or immunosuppressive (IS) agents facilitates tapering of glucocorticoids.
5. HCQ sulfate 200 to 400 mg a day or divided twice daily is recommended in all SLE cases. Do not exceed 400 mg/d. *Consult with a rheumatology specialist.*
6. IS agents, such as methotrexate (MTX) or azathioprine, are used in clients who respond poorly to HCQ and glucocorticoids.

FOLLOW-UP

A. Very close follow-up by a physician specialist is needed when immunosuppressants are employed.

B. If fever is present on examination, explore causes of fever to rule out an infection. Monitor the client for infections, especially with opportunistic organisms.

C. Strategies to prevent heart disease are important due to the presence of premature atherosclerosis seen in these clients.

D. Appropriate immunizations need to be up to date.

E. Osteoporosis screening.

Infections are the leading cause of death secondary to immunosuppression, followed by active SLE, and renal or CNS involvement.

CONSULTATION/REFERRAL

A. Refer the client to a rheumatologist at the time of diagnosis.

B. Referral to a specialist may be needed if biopsy is necessary to confirm diagnosis for skin or renal conditions.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Infertility: 25% of clients have difficulties conceiving.
2. Clients with active SLE (with multiorgan involvement) may experience miscarriages and stillbirths.
3. Consultation and management with a perinatologist are needed for high-risk mothers.
4. Approximately 33% of clients have an antibody (anticardiolipin) that is associated with early failure of the placenta.
5. Approximately 10% have a related antibody (lupus anticoagulant) that allows early pregnancy, but compromises fetal growth as the placenta fails.
6. It is estimated that 25% of the remaining pregnancies deliver prematurely.
7. Family planning:
   a. Barrier methods or intrauterine devices (IUDs) are safest.
   b. Clients with active SLE should avoid the use of birth control pills with estrogen. Consider alternate methods of birth control.
8. Pregnancy may exacerbate lupus in some clients.

B. Geriatrics:
1. The average age of onset is about 10 years later in males.
2. Older adults with SLE are at increased risk of developing cognitive impairment. Early detection is necessary to assist the client and the family to adapt and prevent further decline.
3. Thoroughly assess SLE geriatric sleep patterns. Impaired sleep and depression may contribute to cognitive decline. These need to be diagnosed and treated to prevent further deterioration.

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TEMPORAL ARTERITIS/GIANT CELL ARTERITIS

DEFINITION

A. Temporal arteritis, also known as giant cell arteritis (GCA) is a systemic vascular illness affecting primarily the blood vessels. There is inflammation of the arteries that begins in the aortic arch and branches out to the cranial arteries.

INCIDENCE

A. GCA occurs in approximately 1 in 500 individuals older than 50 years. GCA rarely occurs in individuals younger than 50 years and is more commonly seen during later years (older than 70 years). It can also be seen in clients diagnosed with polymyalgia rheumatica (PMR). Approximately 40% to 50% of clients diagnosed with GCA will also have PMR.

PATHOGENESIS

A. The cause of GCA is unknown. The immune system attacks the body and causes inflammation of the medium and large arteries, which causes thickening of the arterial walls and narrowing of the lumen. When these changes occur, the result is a decrease in blood flow that can potentially cause an occlusion in the artery, leading to ischemia. Arteries commonly involved include the temporal artery, the medium and large vessels, and the vessels of the eyes. Blindness is the major acute morbidity with GCA.

PREDISPOSING FACTORS

A. Age (older than 50 years).
B. Ethnicity: (Scandinavian) Northern European descent.
C. Sex (female to male ratio of almost 3:1 in Scandinavian populations).

COMMON COMPLAINTS

A. Abrupt new onset of headaches with tenderness of scalp.
B. Visual impairment.
C. Joint pain (PMR).

OTHER SIGNS AND SYMPTOMS

A. Fever.
B. Normochromic anemia.
C. Jaw or arm claudication.
D. Fatigue.
E. Weight loss.

POTENTIAL COMPLICATIONS

A. Visual loss, blindness.
B. Joint pain.

SUBJECTIVE DATA

A. Have the client describe the presenting complaints. What makes it better or worse?
B. Ask the client when presenting symptoms began. Discuss the course of new-onset symptoms. Note systemic symptoms such as fever, weight loss, fatigue, headache, vision changes, and jaw/arm pain.
C. Ask the client to describe pain, for example, crushing, stabbing, or burning.
D. Have client rate pain on a scale of 1 to 10, with 1 being the least painful.
E. What has the client taken to relieve the pain or symptoms?
F. Has the client noted any visual disturbance or changes? Are visual manifestations persistent or transient with headaches? What makes the headache worse? What makes the headache better?

PHYSICAL EXAMINATION

A. Check vital signs: temperature, pulse, respirations, and blood pressure (BP; take BP in both arms and note discrepancies).
B. Inspect:
1. Inspect overall general appearance. Clients with GCA usually appear chronically ill.
2. Perform funduscopic examination, looking for a pale disc and blurred margins of the disc. Assess pupils equal round reactive to light and accommodation (PERRLA), extraocular muscles (EOMs), and visual acuity.
C. Auscultate:
1. Heart for murmurs.
2. Carotid, brachial, femoral arteries for bruits.
3. Abdomen, noting any bruits.
D. Palpate:
1. Palpate pulses (carotid, brachial, radial, pedal, and femoral pulses).
2. Perform range of motion (ROM) of all joints (shoulders, neck, hips, and extremities), noting limitations due to pain and/or swelling.
3. Assess joints for swelling and pain. (Note symptoms of PMR.)

DIAGNOSTIC TESTS

A. Serum laboratory studies (erythrocyte sedimentation rate [ESR], complete blood count [CBC], C-reactive protein [CRP], and comprehensive metabolic profile [CMP]).
B. Temporal artery biopsy. Any client suspicious of having temporal arteritis should have a temporal artery biopsy performed. This is the gold standard for diagnosing GCA. Recommended sample size of the artery for biopsy should be ≥4 cm. False-negative biopsy results may be up to 20%.

DIFFERENTIAL DIAGNOSES

A. Temporal arteritis/GCA.
B. Vasculitis.
C. PMR.

PLAN

A. General interventions:

1. Educate the client and the family regarding the disease process of GCA.

B. Client teaching:

1. Educate the client and the family that the common symptoms of GCA include headaches, visual change, and jaw/arm joint pain.

2. Teach the client that GCA causes inflammation of the blood vessels in the head and neck, but not the blood vessels in the brain.

3. Advise the client that PMR may also occur in some clients, in which joint pain may be present.

4. Medications commonly used for GCA are steroids. Glucocorticoid therapy will suppress the inflammation, which in return will reduce pain. The steroids are commonly used for the duration of treatment and will be tapered down over several months. Steroids are the only medications that have been proven to prevent blindness. Any side effects or problems should be reported to the primary provider.

5. A low-dose 81-mg aspirin is commonly used daily.

6. Any vision changes should be immediately reported to the primary provider. Vision loss/blindness is one of the greatest risks of GCA and may be irreversible.

C. Pharmaceutical therapy:

1. If GCA is suspected, consult with a collaborating physician and begin steroids immediately: prednisone 40 to 60 mg daily. Do not wait for biopsy results. Steroids are used to suppress the manifestations of symptoms and reduce the risk of blindness. Steroids are tapered slowly when symptoms begin to improve, commonly after 1 month.

2. Aspirin 81 to 325 mg/d is recommended.

FOLLOW-UP

A. Follow up with a physician, rheumatologist, and/or surgeon as recommended.

CONSULTATION/REFERRAL

A. Consultation with the collaborating physician or surgeon should be obtained regarding any client with suspicion of having temporal arteritis.

B. Refer all clients to a rheumatologist within 1 week of diagnosis to manage the client with GCA.

C. All clients suspected of having temporal arteritis should be referred to a general surgeon for a temporal artery biopsy within 2 to 3 days after beginning steroids.

D. Refer the client to an ophthalmologist for further evaluation and treatment.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Most often seen in older clients. Symptoms may occur after the age of 50 years; however, GCA is most commonly seen in clients older than 70 years.

2. Do not delay treatment while waiting for pathology results. Immediately begin prednisone 40 to 60 mg/d and consider adjunct of proton pump inhibitor to prevent gastrointestinal (GI) bleed.

3. Thirty-five percent of geriatrics diagnosed with GCA experience a relapse. Maintain treatment for 1 year to prevent relapse.

4. Geriatrics suffering from GCA have a higher risk of stroke. Consider low-dose (81–100 mg/d) aspirin to reduce risk of visual loss, transient ischemic attack (TIA)/cerebrovascular accident (CVA), and thoracic aortic aneurysm.

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VITAMIN D DEFICIENCY

DEFINITION

A. Vitamin D deficiency is defined as having a serum 25-hydroxyvitamin D (25[OH]D) level lower than 12 ng/mL. Vitamin D insufficiency is defined as having a serum 25-(OH) D level of 12 to 20 ng/mL. Vitamin D sufficiency is defined as a serum 25-(OH)D level greater than 20 ng/mL.

INCIDENCE

A. Vitamin D deficiency is seen in all ages. It is highest in older adults, institutionalized, and/or hospitalized clients. It is found to be more common in those with osteoporosis and malabsorption, including inflammatory bowel disease and celiac disease. Postmenopausal females are at greater risk often due to low dietary intake of vitamin D. It is also more common in the winter months due to less sun exposure during these months.

PATHOGENESIS

A. There are multiple causes of vitamin D deficiency. Deficiency may be due to limited sun exposure, decreased dietary intake, and increased hepatic catabolism. The best source of vitamin D is effective sunlight exposure to the skin. Vitamin D is also absorbed by consuming vitamin D-rich foods. The liver is responsible for metabolizing vitamin D. The liver hydroxylates the vitamin D to storage form, 25[OH]D, which then breaks down into the bioactive form in the kidney, 1,25-dihydroxyvitamin D (1,25[OH]2D). The 1,25(OH)2D is regulated by the parathyroid hormone (PTH) and causes calcium absorption to occur in the intestine, which in return affects bone metabolism and muscle function. When any part of this cascade is interrupted, the cascade is damaged and vitamin D deficiency occurs.

PREDISPOSING FACTORS

A. Darker skin.

B. Age: older adult population at highest risk.

C. Long-term institutionalized individuals (nursing home).

D. Obese individuals.

E. Decreased sun exposure (individuals who spend very little time outdoors in the sun).

F. People with serious nervous or digestive disorders (chronic kidney disease and malabsorption problems).

G. Medications: phenytoin, phenobarbital, and rifampin induce hepatic p450 enzymes and accelerate catabolism of vitamin D.

COMMON COMPLAINTS

A. Asymptomatic to severe clinical manifestations.

B. Chronic muscle aches/pain/fatigue/weakness.

C. Joint pain and bone pain.

OTHER SIGNS AND SYMPTOMS

A. Fracture of bone.

B. Frequent falls and muscle weakness.

C. The most severe form of vitamin D deficiency can cause nutritional rickets.

SUBJECTIVE DATA

A. With clients presenting with complaints, assess onset, duration, and course of complaints.

B. Assess daily nutritional habits. Does the client get enough calcium and vitamin D in current diet?

C. Does the client live in the home or an institution? Is the client allowed to spend time outdoors in sunlight? What time of the season/year is it? Is 20 to 30 minutes in the sun without sunscreen reasonable?

D. Is the client currently taking any vitamin supplements? If so, review the vitamin and its ingredients.

E. Review client history and determine if the client has a chronic medical condition, including malabsorption condition and chronic kidney condition, or takes medications that would interfere with vitamin D absorption. If there is no current diagnosis of a gastrointestinal (GI) problem, inquire regarding food intolerances, stool patterns, constipation, and diarrhea history.

F. Does the client currently take a vitamin D and/or calcium supplement?

G. Has the client had a recent vitamin D level drawn?

H. Inquire regarding the client's fatigue level.

I. Assess for muscle weakness and frequent falls, especially if a pattern of more falls in the winter months is noticed.

J. Has the client been diagnosed with osteoporosis? If so, the client needs to be screened for vitamin D deficiency.

PHYSICAL EXAMINATION

A. Check pulse and blood pressure (BP).

B. Inspect:

1. Observe the client walk in the room and assess for stability.

2. Observe the skin color and overall appearance and type of skin texture.

3. Note any deformities in the spine:

a. Kyphosis.

b. Bowing of the legs.

c. Waddling of gait.

C. Auscultate:

1. Heart.

2. Lungs.

D. Palpate:

1. Affected joints or areas of reported tenderness.

2. Abdomen if intestinal absorption problems are suspected and workup is needed.

DIAGNOSTIC TESTS

A. Serum 25(OH)D: normal 20–40 ng/mL; a minimum of 30 mg/mL in older adults.

B. PTH: normal 14–72 pg/mL.

C. Calcium level: normal 8.5–10.2 mg/dL.

DIFFERENTIAL DIAGNOSES

A. Vitamin D deficiency.

B. Osteoporosis.

C. Rickets.

D. Cystic fibrosis.

E. Malabsorption syndrome.

F. Chronic kidney disease.

G. Celiac disease.

PLAN

A. General interventions:

1. For low-risk adults, routine screening for vitamin D deficiency is not recommended. Suggest 600 to 800 IU of vitamin D_3 daily in adults without regular sun exposure.

2. Educate the client regarding the importance of calcium and vitamin D to the body.

3. High-risk adults with suspicion of vitamin D deficiency should be screened. Measurement of serum (OH)D concentration should be done. Vitamin D levels less than 10 ng/mL are at risk for osteomalacia and should also have serum calcium, phosphorus, alkaline phosphatase, PTH, electrolytes, blood urea nitrogen (BUN), creatinine, and tissue transglutaminase antibodies measured to assess for celiac disease.

4. High-risk clients complaining of bone pain should have an x-ray of the bone where they are having pain.

B. Client teaching:

1. Educate the client about vitamin D deficiency and the importance of receiving adequate dietary sources of vitamin D daily.

2. Suggest eating foods that are higher in vitamin D in the diet. These foods include fish oil, cod liver, salmon, and foods fortified with vitamin D, such as milk and cereals.

3. Discuss how inadequate sun exposure can increase the risk of vitamin D deficiency. Recommend 20 to 30 minutes of effective sunlight exposure on a regular basis.

4. Foods higher in calcium should also be included in the diet daily.

C. Pharmaceutical therapy:

1. Vitamin D level recommendations:

a. For vitamin D levels less than 12 ng/mL: vitamin D_2 or D_3 50,000 IU weekly for 6 to 8 weeks and then 800 IU thereafter.

b. Vitamin D levels between 12 and 20 ng/mL: vitamin D_3 800 to 1,000 IU daily and recheck level in about 3 months.

c. Vitamin D levels between 20 to 30 ng/mL: vitamin D_2 or D_3 600 to 800 units daily.

d. Cholecalciferol (vitamin D_3) is preferred to ergocalciferol (vitamin D_2) for supplementation when available.

2. Calcium levels should be maintained with a recommended dietary intake of 1,000 mg/d for ages 19 to 70 years and of 1,200 mg/d for females 51 to 70 years and all adults over 71 years of age.

3. For malabsorption problems, recommend magnesium 250 mg daily supplement. May also refer to a GI specialist for workup.

FOLLOW-UP

A. Follow-up should be performed according to recommendations based on laboratory results. Initial laboratory results should be repeated in 8 weeks with initial treatment. As soon as vitamin D levels are stable, repeat lab work routinely to confirm that levels remain within normal range. If levels continue to fall, refer to a specialist.

CONSULTATION/REFERRAL

A. Consult with a physician or refer the client to a gastroenterologist for those who consistently have low vitamin D levels or if malabsorption problems are diagnosed.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. No contraindications for treatment during pregnancy. The same prescribing dose is safe for pregnancy and breastfeeding clients.

B. Geriatrics:

1. Vitamin deficiencies are commonly seen in this population. ▶

2. All clients diagnosed with osteoporosis should be assessed for vitamin D deficiency.

3. Clients with a serum 25(OH)D level of less than 10 are at risk of developing osteomalacia.

4. High doses of vitamin D (60,000 IU D_3/mo) increase the risk of falls by 15% and fracture risk by 26% due to the side effect of impaired balance with high doses of vitamin D. The recommendation of total vitamin D intake/mo for clients older than 70 years of age is 24,000 IU/mo.

5. Treatment guidelines for geriatric clients include intake of vitamin D 800 to 1,000 IU/d and calcium 1,000 to 2,000 mg/d in divided doses.

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CLIENT TEACHING GUIDE

FIBROMYALGIA

PROBLEM You have inflammation or pain in the muscles and connective tissues, usually noted in the lower back, shoulders, neck, chest, arms, hips, and thighs. This pain is chronic, and you may also have excessive fatigue and stiffness, difficulty with sleeping, and possibly other symptoms of anxiety, stress, and/or depression.

CAUSE The cause is unknown. Fibromyalgia has been linked to anxiety, depression, and stress.

TREATMENT PLAN/CARE

A. Foundation of care is regular exercise.
B. Achieve adequate amounts of sleep.
C. For symptoms of discomfort, you may try applying heat to the areas of pain, such as hot showers, heating pad, and whirlpools.
D. Gentle massage by a massage therapist may help with comfort.
E. Reducing stress in your life is important for overall well-being and pain reduction.
F. Consider alternatives to relieving stress, such as biofeedback, relaxation techniques, and yoga.
G. Notify your healthcare provider if you are having symptoms of anxiety or depression that need addressing and may possibly be treated with medications.

Activity: Regular exercise is recommended. Exercise at least 30 minutes for three to five times a week if possible. Even 5 minutes is better than no exercise. Establish a regular sleep time. Adequate and sound sleeping may reduce symptoms. You may require naps during the day.

Diet: Eat a nutritious regular diet. Increase your intake of fruits and vegetables. Avoid caffeine and alcohol and foods that may interfere with your normal sleep.

Medications: There are medications to help with your fibromyalgia. You may be prescribed one of these by your healthcare provider. Examples may include duloxetine (Cymbalta), milnacipran (Savella), or pregabalin (Lyrica).

You may also need to treat other symptoms accordingly:
A. Pain can be addressed with nonsteroidal anti-inflammatories such as ibuprofen or naproxen.
B. Sleep disorder: Address this with your healthcare provider for treatment.
C. Anxiety/stress/depression: Address this with your healthcare provider.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. New or unexplained symptoms that have developed.
B. Fever higher than 100.0°F.
C. Other: _____

Phone: _____

RESOURCE The National Fibromyalgia Association: www.fmaware.org

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CLIENT TEACHING GUIDE

GOUT

PROBLEM Gout is caused by high uric acid deposits in the joints, which produce pain and swelling at the joint. Gout commonly occurs in the big toe; however, other joints may be affected. Other signs/symptoms include red, hot, and swollen joints which may be tender to the touch. Gout also produces tophi, which are uric acid crystals that have formed under the skin, such as the edge of the ear, elbow, fingers, and toes, and near the Achilles tendon.

Gout is the result of too much uric acid production or not enough uric acid excretion from the kidney.

PREVENTION/CARE

A. The goal is to prevent recurrences of attacks.
B. Acute attacks: You may apply warm or cool compress to the affected areas for comfort.
C. Avoid weight-bearing objects on the affected joint.
D. Take your medication as prescribed.

Activity: When you have an attack, resting the joint is the best treatment. Drinking large amounts of water will keep your urine diluted to help prevent kidney stones.

Diet:

A. Drink 10 to 12 glasses of water a day. Avoid dehydration.
B. Refrain from alcohol (beer, liquor). These will worsen your symptoms or trigger a new attack.
C. If overweight, you need to begin a weight-loss program appropriate for you.
D. Avoid crash diets. These will also precipitate an acute attack.
E. Foods to avoid: purine-rich foods such as some seafood, sardines, anchovies, red meat and organ meats (liver, kidney), dried beans, shrimp, and sweetbreads, as well as foods with high-fructose corn syrup (soda, cakes, cookies, etc.).
F. Foods to increase: low-fat dairy products, whole grains and vegetables, and coffee.

Medications: Medications to avoid: salicylates (aspirin). They can interfere with the kidney trying to get rid of the high uric acid levels.

You may be prescribed medications for pain, such as anti-inflammatories (ibuprofen or naproxen), steroids, pain relievers (acetaminophen), or colchicine.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. Acute attacks.
B. Fever higher than 101°F.
C. Rash.
D. Swelling of extremities.
E. Vomiting/diarrhea.
F. Signs/symptoms not improving within 3 to 4 days after starting therapy.
G. Any adverse effects from your medication.

Phone: _____

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CLIENT TEACHING GUIDE

OSTEOARTHRITIS

PROBLEM Osteoarthritis (OA) is a very common disorder that affects the weight-bearing and movable joints. OA damages the cartilage tissue in the joint. The cartilage provides the cushion around the joint. If the cartilage becomes damaged, it will become inflamed and irritated and start to thin. When this occurs, there is less cushion between the bones, and you will experience more pain and swelling. The joints most commonly involved are the finger and toe joints, knees, hips, and spine.

CAUSES There are many causes of OA. A previous injury to the joint, repeated stress to the joint (like a bricklayer's hands), genetics, age, obesity and other diseases (such as diabetes), or infections may cause OA.

TREATMENT

A. The goal of treatment is to prevent further joint damage.
B. Learn as much as you can about this disorder and ask your healthcare provider lots of questions.
C. Heat and massage may increase joint movement and reduce pain.
D. Physical or occupational therapy may be needed.
E. Heat or ice packs may be used for localized relief.

Activity:
Exercise:
A. Range-of-motion exercises increase the movement of the joint.
B. Careful exercise may also strengthen the muscles around the joint.
C. Ask your provider what is the best type of exercise for you.
D. Yoga and acupuncture have been shown to help relieve pain and stiffness.

Joint protection:
A. Do not overuse a joint.
B. If you work with your hands and have OA of the fingers, take frequent rest periods. This applies to all the other joints as well.

Diet: A low-fat, low-cholesterol diet may be suggested. Losing weight is recommended if you are overweight and if you have OA of the knees, hips, or spine.

Studies show that for every 1 lb of weight loss there is approximately a 4-lb weight-stress reduction on the knees/joint.

Medications: Your healthcare provider may recommend that you take acetaminophen or an anti-inflammatory drug. This should help relieve the pain and stiffness. Special creams also sometimes help joint pain. In cases of severe OA, your healthcare provider may recommend an injection to the joint. For severe pain, surgery may be required.

***Remember* to take only the medicines prescribed for you.**

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. Increased pain despite medication.
B. Fever higher than 100.4°F.
C. Abdominal pain or discomfort after taking medicine.

Phone: _____

RESOURCE Arthritis Foundation: 800-283-7800, www.arthritis.org

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CLIENT TEACHING GUIDE

OSTEOPOROSIS

PROBLEM Osteoporosis is a condition in which the bone loses its normal density, mass, and strength, making it weak and more vulnerable to fracture (break).

CAUSES The weakening of the bone can be caused by several factors. Some of these risk factors include the following:

A. Inadequate amounts of calcium and vitamin D in the diet.
B. Decreased exercise.
C. Some chronic diseases, such as thyroid disease, diabetes, and heart failure.
D. Cancer.
E. Smoking.
F. Low estrogen levels (menopause).
G. Excessive alcohol intake.
H. Advancing age.
I. Ethnicity: white and Asian females.
J. Long-term use of certain medications, such as steroids, thyroid and seizure medications, and some cancer treatment medications.

PREVENTION

A. Calcium intake: Be sure to get adequate calcium intake.
  1. Diet ary intake of **calcium** is recommended for the following:
    a. Males younger than 50 years: 1,000 mg/d.
    b. Males 50 to 69 years: 1,000 mg/d.
    c. Males older than 70 years: 1,200 mg/d.
    d. Females younger than or equal to 50 years: 1,000 mg/d.
    e. Females older than 50 years: 1,200 mg/d.
  2. Calcium supplements such as calcium carbonate or calcium citrate are recommended. Do not exceed more than 500 mg at one time. If taking two tablets to meet requirements, divided doses are recommended for best absorption.
B. Vitam in D intake: Be sure to get adequate vitamin D daily.
  1. Dietary intake of vitamin D is recommended for the following:
    a. Ages younger than 50 years: 400 to 600 IU/d.
    b. Ages 51 to 70 years: 600 to 1,000 IU/d.
    c. Ages 71 years and older: 800 to 1,000 IU/d.
  2. Food sources of vitamin D include milk fortified with vitamin D, salmon, egg yolks, cod liver oil, tuna, liver, and cereals fortified with vitamin D.
C. Avoid excessive alcohol intake.
D. Avoid smoking and/or encourage smoking cessation.

TREATMENT PLAN

A. If osteoporosis is suspected, your provider may order a bone density study. The goal is to prevent the disease from occurring.
B. If you have been diagnosed with osteoporosis, you need to prevent the disease from progressing and take measures to prevent all bone fractures.
C. Use caution when walking on wet, slippery surfaces.
D. Avoid risk of falls by making your home safe. Avoid throw rugs on floors and remove small items that are easily tripped over. Use caution when pets are around to prevent falling over your pet.

Activity:

A. Physical activity is vital to maintain and prevent further bone loss.
B. Weight-bearing activity is the best activity, such as walking or running for at least 30 minutes a day, 3 days a week.
C. Avoid high-impact sports and activities such as jumping and high-impact aerobics to prevent fracturing the bones.

(*continued*)

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CLIENT TEACHING GUIDE

OSTEOPOROSIS (*CONTINUED*)

D. Avoid any risk of falls.
E. Use walking devices, such as a cane or walker, if needed.
F. Bathtubs should have nonskid protection.

Diet:
A. Eat a well-balanced diet. Increase food sources rich in calcium and vitamin D.
B. Sources of vitamin D include milk, some fish (salmon, tuna), egg yolks, cod liver oil, liver, and drinks and cereals fortified with vitamin D and minerals.
C. You can also get vitamin D from spending 20 to 30 minutes in the summer sunlight, exposing your skin to the sun without wearing sunscreen for this length of time.
D. Sources of calcium include green leafy vegetables (kale and broccoli), almonds, tofu, milk, dairy products (cottage cheese, cheese, yogurt), and drinks fortified with calcium such as orange juice and soy milk.
E. If you are overweight, a low-fat, low-cholesterol diet is suggested to lose weight.

Medications:
A. You may be prescribed nonprescription medications like acetaminophen (Tylenol) as needed for pain.
B. Supplements: calcium, vitamin D, and hormone replacement for females who are menopausal.
C. Bisphosphonates:
1. If prescribed a bisphosphonate, take the medication with a full glass of water.
2. Wait approximately 30 to 60 minutes before reclining or consuming other medications, beverages, or food to lower the risk of regurgitation or a burning sensation.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. Been experiencing any pain; you need to have your provider assess this pain.
B. Other: _____

Phone: _____

RESOURCES National Osteoporosis Foundation: www.nof.org

National Women's Health Information Center: www.4women.gov/FAQ/osteoporosis.cfm

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POLYMYALGIA RHEUMATICA

PROBLEM Polymyalgia rheumatica (PMR) is a common disorder experienced by adults older than the age of 50 years in which complaints involve overall joint stiffness and pain. Common complaints include feeling fine one day and the next day having overall joint pain, with difficulty getting out of bed or standing up from a chair. Symptoms may occur gradually over a period of several days or weeks, or may appear abruptly overnight. Symptoms are usually worse in the morning hours. The joints most commonly affected are the upper arms, thighs, hips, buttocks, and neck.

CAUSES

A. The cause of PMR is unknown.
B. Some theories maintain that PMR may be triggered by an infection; however, research does not identify any specific infection.

PREVENTION There is no way to prevent the onset of PMR.

TREATMENT PLAN

A. You may be treated with low-dose steroids by mouth. Prednisone is a common medication used to treat this disorder. Stiffness and joint pain may quickly improve with use of oral steroids. If improvement with steroids does not occur within 2 weeks of treatment, you should notify your provider.
B. Once improvement is noted, your provider will begin to lower the steroid dose gradually over time. This may take several weeks to months.
C. Do not change or taper your steroid dose on your own. Your provider will advise you on tapering dose when appropriate.

Activity:
A. Once stiffness and pain have improved, you may resume normal activities. Exercise is encouraged on most days of the week.

Diet:
A. There is no special diet to follow for this disorder; however, avoiding sugar and simple carbohydrates should help avoid weight gain while on steroids.
B. Blood sugar: Side effects of prednisone may include increased blood sugar. If you have concerns regarding your blood sugar values or if you are diabetic, you need to discuss proper management and monitoring of your blood sugar while taking steroids.

Medications:
A. Oral steroids (prednisone) are commonly prescribed over a period of time. Discuss proper dosages and tapering of this medication with your provider.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:
A. Any new symptoms, such as headache, jaw pain, or vision changes.
B. Side effects from prednisone or other medications prescribed.
C. Increase in pain or weakness while using prednisone.
D. Any other concerns.

Phone: _____

RESOURCES Arthritis Foundation: www.arthritis.org

National Institute of Arthritis and Musculoskeletal and Skin Diseases Information Clearinghouse: www.niams.nih. gov/Health_info/Polymyalgia/default.asp

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CLIENT TEACHING GUIDE

SYSTEMIC LUPUS ERYTHEMATOSUS

PROBLEM You have been diagnosed with a condition called lupus. Lupus is a chronic, inflammatory, autoimmune disorder. There is currently no cure for lupus; however, it can be managed and can go into remission or a dormant state. Rashes, hair loss, arthritis-like joint pain, and fatigue are very common problems with lupus.

CAUSE Lupus causes your body to attack its own cells. The exact cause is unknown. Lupus tends to run in families, but it is not contagious.

PREVENTION There is no known prevention, only long-term management.

TREATMENT PLAN

A. Avoid sun exposure.
   1. The eyes of many lupus clients are sensitive to light; wear sunglasses and avoid direct exposure.
   2. Light exposure can make your rash worse.
   3. Apply a protective lotion or sunscreen to your skin while outside.
   4. Wear long sleeves and use hats.

B. You may be given steroids for your skin rashes or lesions.

C. Avoid exposure to drugs and chemicals.

D. Review all of your medications and over-the-counter drugs with your healthcare provider.

E. Avoid hairsprays and hair-coloring agents.

F. Mouth ulcers may occur with lupus.
   1. Avoid hot or spicy foods that might cause irritation to ulcers.
   2. Use good oral hygiene.
      a. Get regular dental exams.
      b. Floss your teeth daily.
      c. Brush your teeth at least twice a day.

G. Drugs called nonsteroidal anti-inflammatory drugs (NSAIDs) are used for minor joint pain.

H. If treated with hydroxychloroquine, it is very important for you to have your eyes examined within a year of starting treatment, which includes fundus exam, visual field testing, and spectral domain optical coherence tomography (SD-OCT).

I. Talk to your healthcare provider if you are planning a pregnancy.

Activity: You may get tired more easily; plan rest periods. However, you should still get some exercise as tolerated. Many people gain some weight from steroids.

Diet: Eat a regular diet as tolerated; you do not need any special foods.

Medications: _____

You Have Been Prescribed the Following Steroid: _____

You Need to Take: _____

A. Do not stop taking your steroid abruptly.

B. Steroid medication should be tapered off (decreased until you are off of it).

You Need to Notify the Office If:

A. You are unable to tolerate your medication.

B. You have any sign of infections, sinusitis, bronchitis, flu, urinary tract infections, and so forth.

C. You are getting worse or do not feel better.

D. You are planning a pregnancy or think you are pregnant.

E. Other: _____

Phone: _____

RESOURCES Lupus Foundation of America: 800-558-0121, www.lupus.org

Lupus Research Alliance: www.lupusresearch.org, 212-218-2840, info@lupusresearch.org

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CHAPTER 23

NEUROLOGIC GUIDELINES

Jill C. Cash, Justin Calabrace, and Lauren E. Kimbrell

ALZHEIMER DISEASE

DEFINITION

A. Alzheimer disease (AD) is a permanent, progressive neurocognitive disorder that is characterized by deficits in several dimensions of cognitive functioning, including memory disturbance, that severely interfere with the person's everyday living but produce no decrease in level of consciousness (LOC). Insidious onset, gradually progressive decline in intellectual functioning, and absence of other specific causes of dementia are also present.

B. Alzheimers.net notes that AD has seven distinct stages. See the following website for full descriptions of "What Are the 7 Stages of Alzheimer's Disease?": www.alzheimers.net/stages-of-alzheimers-disease.

1. Stage 1: no impairment.
2. Stage 2: very mild decline (losing keys; forgetting familiar names).
3. Stage 3: mild decline (memory and concentration problems).
4. Stage 4: moderate decline (difficulty planning dinner or paying bills on time).
5. Stage 5: moderately severe decline (unable to recall events or remember important dates like birthdays, anniversaries, etc.).
6. Stage 6: severe decline (needs help getting dressed; personality begins to change).
7. Stage 7: very severe decline (late-stage AD includes trouble eating, swallowing, speaking, and all motor function).

INCIDENCE

A. AD is the most commonly occurring neurocognitive disorder, affecting more that 44 million people throughout the world in 2015 and expected to double by the year 2050. Among the elderly, approximately 40% of those older than 85 years of age are affected. Of all types of dementias, Alzheimer dementia includes 70% of those affected, with the other 30% affected by atypical dementia. AD is the sixth leading cause of death in the United States.

B. Early-onset AD occurs between a person's 30s to mid-60s. Early-onset AD only affects about 10% of all people who have AD.

C. Many people with Down syndrome develop AD as they age. By age 40, almost all people with Down syndrome have plaque from amyloid precursor protein (APP) and protein deposits called tau tangles, which increases the risk of developing AD. People with Down syndrome begin to show symptoms of AD in their 50s or 60s.

PATHOGENESIS

A. The disease is a degenerative process involving cell loss from the basal forebrain, cerebral cortex, and other areas in which plaques and tangles build up and block the cell processes that are needed to survive, thereby destroying the nerve cells. Cortical atrophy is most prominent in the temporal and hippocampal regions of the brain. Death of the nerve cells causes memory loss, changes in personality, and other signs and symptoms of AD. The Apo E epsilon 4 allele of chromosome 19 has been associated with familial and late onset AD.

B. People with Down syndrome are born with an extra copy of chromosome 21, which carries the APP gene. This gene produces a specific protein called APP. APP protein leads to a buildup of protein clumps called beta-amyloid plaques in the brain.

C. Obesity has been identified to increase the risk of AD because it increases the adipokine dysregulation, which causes a release in the proinflammatory adipokines and decreases anti-inflammatory adipokines.

PREDISPOSING FACTORS

A. Definite risks:

1. Advanced age older than 50 years.
2. Atrial fibrillation (AFib).
3. Depression.
4. Abnormal genetic makeup, such as Down syndrome.
5. Positive family history of AD.
6. Sex (females have a higher incidence; Alzheimer is two-thirds more common in females than in males).
7. Obesity.

B. Possible risks:

1. Delirium.
2. Head trauma.
3. Heavy smoking
4. Hypertension (HTN).
5. Hyperlipidemia.
6. Low educational level.
7. Postmenopausal hormone therapy (HT).

COMMON COMPLAINTS

A. Significant memory loss or change in behavior as reported by the client, family, or caregiver. ▶

The contributions of Julie Adkins, Barbara I. Bailes, Cheryl A. Glass, Karen M. Kress, and Kimberly D. Waltrip to this chapter in prior editions are acknowledged here.

B. Typical changes that occur with aging compared with the "10 Alzheimer warning signs" are noted in Table 23.1.

SUBJECTIVE DATA

Family members are a good resource for obtaining an accurate history.

A. Determine onset, course, and duration of symptoms.
B. Note loss of immediate, recent, or remote memory, such as trouble remembering appointments, difficulty recalling recent events, and inability to find personal belongings.
C. Determine the client's ability to make reasonable judgments, such as answering the phone when it rings, and so forth.
D. Is the client able to carry on daily functions of living, including cooking meals and cleaning house?
E. Discuss the client's sleep–wake cycle.
F. Assess the client for decreased appetite, lack of pleasure in usual activities, melancholy mood, or other symptoms associated with depression.
G. Note language difficulties and problems expressing self.
H. Note any recent physical illness.
I. Review the client's medication history, specifically those medications with anticholinergic side effects, including over-the-counter (OTC) products such as diphenhydramine.
J. Discuss alcohol intake and abuse factors.
K. Review medical history of head trauma, HTN, cerebrovascular accident (CVA), cancer, metabolic problems, neurologic disease, infections, gastric surgery (vitamin B_{12} deficiency), and emotional or psychiatric problems.
L. Note any recent major life events such as death of a spouse, a move to a new living environment, or loss of purpose following retirement.

PHYSICAL EXAMINATION

A. Check blood pressure (BP), pulse, respirations, and weight.
B. Inspect:
  1. Inspect overall appearance for hygiene and nutritional status.
C. Auscultate:
  1. Auscultate the heart, lungs, abdomen.
D. Neurologic exam:
  1. Perform a complete neurologic exam.
  2. Note facial asymmetry, distal weakness, and any focal neurologic findings.
E. Perform screening using standardized tools:
  1. Perform the Standardized Mini-Mental State Examination (SMMSE) screening tool available at www2.gov.bc.ca/assets/gov/health/practitioner-pro/bc-guidelines/cogimp-smmse.pdf. ▶

TABLE 23.1 10 ALZHEIMER WARNING SIGNS

Issue	Common Symptoms of Early Stage Alzheimer Disease	Typical for Aging
Memory loss	Forgetting new information and asking for the same information over and over	Forgetting names or appointments occasionally and remembering them later
Difficulty performing familiar task	Difficulty planning or completing everyday tasks Losing track of steps involved in activities, from preparing a meal to making a phone call	Occasionally forgetting why you came into a room or what you were planning to say
New problems with writing or speaking	Forgetting simple words or substituting unusual words, making speech or writing hard to understand	Sometimes having trouble finding the right word
Confusion with time and place	Getting lost in familiar places, forgetting where they are and how they got there, and how to get back home	Forgetting the day of the week or where you are going
Poor or decreased judgment	Dressing inappropriately for the weather, giving away large sums of money to telemarketers	Making a questionable or debatable decision from time to time
Problems with abstract thinking	Unusual difficulty performing complex mental tasks especially relating to numbers	Finding it challenging to balance a checkbook
Misplacing things and losing the ability to retrace steps	Putting things in unusual, inappropriate places	Misplacing keys or a wallet, but being able to retrace steps to find it later
Changes in mood or behavior	Rapid mood swings, extreme confusion, anxiety, suspiciousness, dependent on a family member	Occasionally feeling sad or moody
Trouble understanding visual images and spatial relationships	Change in visual processing; difficulty reading, judging distance, and determining color or contrast, which may cause problems with driving	Vision changes related to cataracts
Withdrawing from social activities	Avoiding being social due to the changes they have experienced Becoming passive, sitting in front of the television for hours, sleeping more than usual, or not wanting to perform activities of daily living	Sometimes feeling weary of work or social obligations

Source: From *a*Place*for*Mom. (2015). *10 Alzheimer's warning signs.* https://www.aplaceformom.com/planning-and-advice/articles/alzheimers-warning-signs. Reprinted with permission from *a*Place*for*Mom: Connecting Families to Senior Living. (Copyright © 2018).

2. The clock-draw test (CDT) may also be administered and used as a screening tool. The CDT is available at www.rehabmeasures.org (see Section II: Procedures, "Clock-Draw Test").

Rule out other specific causes of dementia, including cerebrovascular disease.

F. Complete depression screening with instrument of choice:
1. Beck Depression Scale. Available at www.enhertsccg.nhs.uk/sites/default/files/pathways/Beck%27s%20Depression%20Inventory_0.docx.

G. Assess functional status. May use a functional assessment tool such as the Physical Self-Maintenance Scale, Instrumental Activities of Daily Living (IADL) Scale, or Reisberg Functional Assessment Staging Scale (FAST). Available at www.pbm.va.gov/PBM/clinicalguidance/drugmonitoring/FunctionalAssessmentStagingFAST73108.doc.

H. Palpate:
1. Neck and thyroid for goiter and lymphadenopathy.

DIAGNOSTIC TESTS

A. Complete blood count (CBC).
B. Chemistry profile.
C. Thyroid function studies.
D. Folate and B_{12} levels.
E. Homocysteine.
F. Methylmalonic acid.
G. Vitamin D level.
H. Venereal Disease Research Laboratory (VDRL) for syphilis.
I. CT scan of the brain.

DIFFERENTIAL DIAGNOSES

A. AD.
B. Drug interactions.
C. Delirium.
D. Depression.
E. Cerebrovascular disease.

PLAN

A. General interventions:
1. Identify the stage of impairment.
a. Preclinical: Brain changes, including amyloid buildup and other nerve cell changes, may already be in progress, but significant clinical symptoms are not yet evident.
b. Mild cognitive impairment (MCI): a stage marked by symptoms of memory and/or other thinking problems that are greater than normal for a person's age and education, but that do not interfere with their independence. People with MCI may or may not progress to Alzheimer dementia:
i. Very mild decline: no symptoms of dementia, has memory lapses.
ii. Mild decline: difficulty in memory and concentration.
iii. Moderate decline: impairment with complex tasks, trouble with solving math problems, forgetting personal history.
c. Alzheimer's dementia: the final stage of the disease in which symptoms of Alzheimer, such as memory loss, word-finding difficulties, and visual/spatial problems, are significant enough to impair a person's ability to function independently:
i. Moderately severe decline: unable to remember phone number, address, confusion on the day of the week, difficulty with decisions on dressing self properly.
ii. Severe decline: difficulty with personal history, difficulty with naming family members, spouse, difficulty with dressing self, behavior changes, may wander and get lost.
iii. Very severe decline: unable to communicate appropriately with others, requires assistance with activities of daily living (ADLs), abnormal reflexes, difficulty swallowing.
2. Provide supportive measures for client and family. Explain to family and the client findings of exam and possible treatment options.
3. Treatment may include treating coexisting diseases, thyroid disease, and vitamin B_{12} deficiency.
4. Discuss therapy options such as musical therapy, occupational therapy, and mind-stimulating activities. Cognitive stimulation has been shown to slow down the degenerative process.
5. Environment: Ensure that the client has a safe environment; measures may need to be taken for environmental safety, such as locks and alarms on doors, relinquishing driving privileges, locking firearms, assessing for fire hazards.

B. Client teaching: *See Client Teaching Guide for Chapter 3, "Safety Issues: Fall Prevention."*
1. Educate clients, family, and the community about preventive measures for developing AD. Obesity is a major risk factor; lifestyle changes, such as dietary/caloric restriction, eating a healthy diet, exercise, mental stimulation, and socialization can all help to decrease the risks of developing AD.
a. Exercise: Daily exercise should be encouraged.
b. Recommend medical identification forclients who wander. (www.medicalert.org/product/catalog/medical-ids/bracelets).
2. Support groups are encouraged for the client, spouse, and/or family. Therapy sessions for each may be beneficial.
3. Client support group: Alzheimer's Association, www.alz.org; 24/7 Helpline: 1-800-272-3900.
4. Families should seek out legal and financial advice early and not wait for a crisis. Alzheimer's Disease Family Relief Program: 1-800-437-2423.
5. The National Institute on Aging's Alzheimer's Disease Education and Referral (ADEAR) Center offers information and free print publications about AD and related dementias for families, caregivers, and health professionals. ADEAR Center staff answer telephone, email, and written requests and make referrals to local and national resources.

C. Pharmaceutical therapy:
1. Cholinesterase inhibitors:
a. Rivastigmine (Exelon): 1.5 to 6 mg BID, increasing the dose by 1.5 mg every 4 weeks to a maximum of 6 mg BID. Exelon patch: Start at 4.6 mg/24 hours and then titrate up to 9.5 mg/24 hours. May be increased to the maximum effective dose of 13.3 mg/24 hours as indicated.
b. Donepezil (Aricept): 5 mg at bedtime, then 10 mg/d may be given after 1 month.
c. Galantamine (Razadyne): 4 mg BID with meals for 4 weeks, then may titrate to max of 12 mg BID. For missed doses, will need to retitrate doses. Razadyne ▶

ER: 8 mg once daily. After 4 weeks, may increase to 16 mg once daily, up to maximum of 24 mg daily titration.

2. N-methyl-D-aspartate (NMDA) receptor antagonist.

a. Memantine (Namenda) XR is used for moderate to severe AD: Namenda XR 7 mg daily, titrate up by 7 mg/wk to a recommended dose of 28 mg daily.

3. Assess the client for secondary behaviors of dementia. Psychosis, delusions, hallucinations, anxiety, depression, and insomnia may occur and should be treated accordingly. Cognitive and behavioral interventions should be initiated in assisting the client in controlling these behaviors.

a. The benefits and risks must be weighed, and pharmacologic treatment may be necessary for clients exhibiting extreme, aggressive behaviors with irritability and/or insomnia.

D. Nonpharmacologic treatment for insomnia includes setting a routine schedule, dressing for bed early, not drinking stimulated drinks such as coffee or tea, decreasing fluid intake, decreasing stimulation including television and tablet, adjusting room temperature, dimming lights (should not be in total darkness), toilet prior to getting in bed.

4. Treatment of depression: citalopram (Celexa), sertraline (Zoloft), paroxetine (Paxil), mirtazapine (Remeron), or escitalopram (Lexapro). Monitor selective serotonin reuptake inhibitor (SSRI) for weight loss, decrease in seizure threshold, and tremors. Do not use with monoamine oxidase (MAO)-I. Monitor Coumadin and lithium levels closely.

FOLLOW-UP

A. Follow-up is variable depending on client status and needs of the client and family. Follow-up is recommended every 3 months to follow the progression of disease. If medications are being introduced, monitor the client monthly.

CONSULTATION/REFERRAL

A. Consider using a visiting nurse, social worker, or occupational and/or physical therapist.

B. Consult a physician for medication treatment for secondary behavioral symptoms.

C. Referral to geriatric psychiatry may be appropriate for treatment of behavioral and psychological symptoms.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. AD should be considered at early ages in adults to avoid risk factors.

2. Lifestyle and dietary changes should be encouraged in adults to prevent risks of developing AD. Exercising, eating a healthy diet, and maintaining a healthy weight (body mass index [BMI]) can decrease risks factors for developing AD.

B. Geriatrics:

1. AD is primarily seen in the geriatric population.

2. The use of probiotics in the elderly has been shown to improve the anti-inflammatory properties of the gut. However, behavioral changes should be monitored closely.

3. Long-term prognosis: Average client lives 7 to 10 years after early symptoms, but life span while demented can be 20 years or more.

4. Provide education and early discussions of transitions of care. The ADEAR website provides information to care givers about transitions from home to hospital or assisted living facilities.

5. Educate and connect caregivers about nonpharmacologic approaches to care and need for respite for the caregiver.

6. Establish a relationship to provide a personal, emphatic approach on the discussion on the end of life issues. See Chapter 4, "Caregiver and End-of-Life Issues," for an in-depth discussion.

7. Beers Criteria caution: See Appendix C, "Beers Criteria," on medications that should be avoided due to side effect profiles and increased risk of geriatric syndromes including falls/fractures, an increased risk of cognitive impairment, delirium/behavioral disturbance, and depression.

RESOURCES

Eldercare Locator
1-800-677-1116 (toll-free)
https://eldercare.acl.gov

National Academy of Elder Law Attorneys
1-703-942-5711
naela@naela.org
www.naela.org

National Hospice and Palliative Care Organization
1-703-837-1500
nhpco_info@nhpco.org
www.caringinfo.org

NIA Alzheimer's and Related Dementias Education and Referral (ADEAR) Center
1-800-438-4380 (toll-free)
adear@nia.nih.gov
www.nia.nih.gov/health/about-adear-center

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BELL'S PALSY

DEFINITION

A. Bell's palsy, also known as idiopathic peripheral facial palsy, is characterized by an acute onset of unilateral facial paralysis. The facial nerve affected is the seventh cranial nerve (CN), which travels in the skull beneath the ear to the muscles on each side of the face. Each facial nerve controls the muscles that control eye blinking and closing and facial expressions such as smiling and taste sensations from the tongue. Bell's palsy affects only one of the paired facial nerves, but in rare cases it can affect both sides.

INCIDENCE

A. Bell's palsy has an incidence of 40,000 Americans a year. It accounts for approximately 75% of all cases of facial paralysis. ▶

Approximately 5% of those affected experience recurrence. Both sexes are affected, as well as all ages, but most clients are in their middle years, older than 30 years. Fewer cases are noted prior to the age of 15 years and after the age of 60 years.

PATHOGENESIS

A. The pathogenesis of Bell's palsy is unknown, but some possible etiologies include genetic, metabolic, autoimmune, and vascular causes. An increasing body of evidence reveals that Bell's palsy may be a virally induced neuritis. A triggering event or stressor induces activation of a latent virus, most likely herpes simplex virus or herpes zoster virus, present within the geniculate ganglion of the facial nerve. Viral activation results in reexpression of dormant viral particles and neural inflammation, leading to entrapment, ischemia, and degeneration of the facial nerve.

PREDISPOSING FACTORS

A. Diabetes.
B. Pregnancy.
C. Recent infection.
D. Positive family history.
E. Hypertension (HTN).
F. Hypothyroidism.

COMMON COMPLAINTS

A. Acute onset of unilateral facial weakness with inability to close one eye.
B. Sagging of one eyebrow.
C. Loss of nasolabial fold.
D. Mouth drawn to affected side.

OTHER SIGNS AND SYMPTOMS

A. Ipsilateral retroauricular pain with or preceding paralysis.
B. Hyperacusis or hypersensitivity to sound.
C. Dysgeusia, or perversion of taste, in the anterior two-thirds of the tongue.
D. Facial paresthesia.
E. Drooling.
F. Decreased tearing.

SUBJECTIVE DATA

A. Elicit onset, duration, and course of symptoms.
B. Have the client describe all neurologic symptoms present.
C. Note associated symptoms such as disruption of taste and disturbances in visual function or hearing.
D. Note predisposing factors such as trauma, infection, or pregnancy.
E. Review the client's family history for presence of Bell's palsy.
F. Review the client's medical history; especially note cerebrovascular or cardiac risk factors. A focused history should include contraindications to use of steroids.

PHYSICAL EXAMINATION

A. Check temperature (if indicated), blood pressure, pulse, and respirations.
B. Inspect:
  1. Note facial appearance.
  2. Observe symmetry of eyes. Check corneal reflex (decreased). Assess ears, nose, and throat; assess the skin for lesions. Assess in and behind ears for zosteriform lesions.
  3. Complete ear exam to rule out infection.
  4. Assess paralysis of all the muscles supplied by one facial nerve. Paralysis may be of varying degrees and need not be complete.
C. Auscultate:
  1. Auscultate the heart and lungs.
D. Neurologic exam:
  1. Perform a complete neurologic exam; test all CNs.

Subjective decreased sensation may be present in the trigeminal distribution.

DIAGNOSTIC TESTS

A. Lyme titer: positive in clients with secondary facial weakness from Lyme disease.
B. Skull radiography, CT scan, or MRI: negative in Bell's palsy, but may show evidence of fracture line, bony erosion by infection or neoplasm, stroke, or tumor.
C. Electromyographic (EMG) studies: occasionally performed to predict prognosis and progression. EMG is reserved for severe cases of paralysis lasting longer than 1 week.
D. Lumbar puncture (LP): indicated only when other conditions are suspected. Cerebrospinal fluid (CSF) should be sent for cytology.

DIFFERENTIAL DIAGNOSES

A. Bell's palsy.
B. Sjögren's syndrome.
C. Stroke.
D. Lyme disease.
E. Sarcoidosis.
F. Ramsay Hunt syndrome, herpes zoster oticus.
G. Acoustic neuroma.
H. Middle ear disease such as purulent otitis media or neoplasms.
I. Guillain–Barré syndrome (GBS).
J. Parotid gland tumor.
K. Carcinomatous meningitis.

PLAN

A. General interventions:
  1. The condition is usually short term (3–4 weeks) and may be managed with steroids and acetaminophen as needed for discomfort.
  2. Physical therapy may be beneficial for muscle weakness and strengthening muscles.
  3. Ensure the client gets reassurance and emotional support.
B. Client teaching: *See Client Teaching Guide for this chapter, "Bell's Palsy."*
  1. Provide eye protection by means of the following:
    a. Apply methylcellulose drops as needed and ocular lubricant (Lacri-Lube) at bedtime and as needed.
    b. Tape eye closed, especially at night to avoid drying effect.
    c. Wear dark glasses when outdoors to minimize light exposure. May also be helpful indoors around bright overhead lights or lamps.
  2. Physical therapy may be beneficial.
  3. Ensure that the client gets reassurance and emotional support.

Recovery may take 3 to 6 months or longer and is complete in approximately 80% of the cases.

C. Pharmaceutical therapy.
  1. Prednisone: Adults take 80 mg every day with breakfast for first 3 days, then decrease dosage to 40 mg for 3 days, then to 20 mg daily for 3 days, then stop.

Recent studies suggest that a brief course of prednisone conveys modest benefits with minimal risks.

2. Use of oral acyclovir in conjunction with prednisone, at a dosage of 400 mg five times a day for 10 days. Consider acyclovir for clients without renal insufficiency and with no other contraindications to therapy. Valtrex 1 g BID for 7 to 10 days is also available.

3. Analgesics, such as acetaminophen, for ear pain or face pain.

FOLLOW-UP

A. For clients with severe symptoms, follow up in 3 to 4 days, then again in 2 weeks.

B. If symptoms worsen or do not resolve within weeks, have the client return to the clinic.

CONSULTATION/REFERRAL

A. Consult a neurologist for the following:

1. Failure to resolve significantly after 4 to 6 weeks. Only 5% to 8% report distressing residual signs and symptoms, including contracture of facial muscles at rest and synergistic mass innervation due to defective nerve regeneration, manifested as either crocodile tears secondary to abnormal secretory fibers intended for the salivary glands or ipsilateral eyelid shutting.

2. Other CN involvement or other abnormalities on neurologic exam.

3. Recurrence of facial palsy: About 5% to 7% of clients experience recurrence of symptoms. Known causes of recurrent palsy include sarcoidosis, diabetes, leukemia, and infectious mononucleosis.

4. Bilateral facial palsies.

B. Consult an ophthalmologist for persistent ocular pain or development of a corneal abrasion or ulceration.

C. Consult an otolaryngologist if decompression treatment is considered.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. The incidence of Bell's palsy is increased in pregnancy, with the highest incidence in the third trimester or immediately postpartum. May treat with prednisone during pregnancy.

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CARPAL TUNNEL SYNDROME

DEFINITION

A. Carpal tunnel syndrome (CTS) is a nerve entrapment condition of the median nerve of the wrist.

INCIDENCE

A. CTS occurs in approximately 1% of the general population. It is primarily seen in 30- to 60-year-old adults. Females and older adults are more likely to develop this condition.

PATHOGENESIS

A. CTS occurs from compression of the median nerve in the carpal canal. Compression occurs due to swelling of the flexor tenosynovium; the pressure blocks the nerve fibers, which produces numbness and discomfort in the digits/hands. Repetitive flexion and extension of the wrists create increased pressure in the carpal canal.

B. Potential causes of CTS include blunt trauma or structural changes; tumors; systemic diseases, such as rheumatoid disorders, diabetes mellitus (DM), thyroid disorders, endocrine diseases, and so forth; mechanical overuse syndrome; and infectious diseases, such as tuberculosis (TB) and leprosy. Consider multifactorial causes of CTS. Heredity is likely an important factor as the carpal tunnel may be smaller in some people or anatomic differences change the amount of space of the nerve; therefore, this trait can run in families.

PREDISPOSING FACTORS

A. Female.

B. Hobbies or jobs that require repetitive wrist or hand movement and the use of vibratory tools.

C. Pregnancy.

D. Heredity.

COMMON COMPLAINTS

A. Pain.

B. Tingling/numbness sensation in the wrists, hands, and fingers that radiates up into the forearm.

OTHER SIGNS AND SYMPTOMS

A. Paresthesia in wrists, hands, and fingers.

B. Localized pain of the radial three digits of the hand.

C. Weakness with grasp.

D. Decreased dexterity.

E. Night pain in wrists.

F. Referred pain to elbow and/or shoulder.

G. Long-term pressure in the carpus can produce ischemic changes and may lead to axonal death, muscular atrophy, and pain. Long-term nerve compression may produce irreversible changes.

SUBJECTIVE DATA

A. Determine onset, duration, and course of presenting symptoms.

B. Note the progression of symptoms since the initial occurrence.

C. Assess whether the symptoms increase with hand or wrist activity and decrease with the joint at rest.

D. Identify factors that precipitate symptoms, that make symptoms worse, and that alleviate symptoms.

E. Inquire whether the client awakens at night with numbness and tingling sensations.

F. Have the client describe the pain, and note if radiation is present. Are symptoms bilateral?

G. Note the client's occupation, hobby, and/or daily routines that require hand or wrist use.

H. Identify what treatment and/or relief measures have been used, and note results.

PHYSICAL EXAMINATION

A. Inspect:

1. Inspect the hands for deformities. Note wasting or atrophy.

B. Palpate:

1. Perform sensory motor evaluation of the hand and arm:

a. Perform Tinel test: Tap over the transverse carpal ligament; result is positive if tingling in fingers is noted. ▶

b. Perform Phalen test: Have the client place elbows on flat surface and hold forearms in vertical position, then flex wrists; result is positive if pain, numbness, or tingling is noted within the next 60 seconds.

DIAGNOSTIC TESTS

A. Electromyographic (EMG).
B. Nerve conduction velocity (NCV) studies.
C. If an underlying systemic illness or condition exists, consider the following:
1. Erythrocyte sedimentation rate (ESR).
2. Blood glucose.
3. Thyroid profile.
4. Inflammatory disease studies.

DIFFERENTIAL DIAGNOSES

A. CTS.
B. Peripheral neuropathy.
C. Cervical spondylosis and cervical disc herniation.
D. Brachial plexus lesion.
E. Trauma.
F. Thenar atrophy and neuropathy.
G. Osteoarthritis.
H. Neurologic disorders: polyneuritis, multiple sclerosis (MS), tumors, and so forth.

PLAN

A. General interventions:
1. Help the client identify causative agents, eliminate activity if possible, decrease repetitive use, or use alternative methods to accomplish the same task.
2. Advise resting arms and wrists as much as possible.
3. Encourage performing daily stretching exercises.
4. Give instructions on applying wrist splints, especially at bedtime, while sleeping.
B. Client teaching:
1. Instruct the client on splinting wrists.
2. Demonstrate stretching exercises.
3. Stress the importance of rest and elimination of the causative activity.
C. Pharmaceutical therapy:
1. Nonsteroidal anti-inflammatory drugs (NSAIDs) as needed. Ibuprofen (Motrin) 600 to 800 mg PO TID or naproxen (Naprosyn) 500 mg PO BID unless contraindicated.
2. Vitamin B_6 100 mg/d.
3. Consider corticosteroid injections in carpal canal (40 mg/mL with 1% lidocaine 1 mL).

FOLLOW-UP

A. Depending on treatment, consider follow-up in 1 month to evaluate status.

CONSULTATION/REFERRAL

A. Refer the client to a physician for severe cases requiring evaluation for surgery.
B. Refer the client to a surgeon for severe symptoms that could require carpal tunnel release.
C. Consider occupational therapy consult.
D. Consider physical therapy consult.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. CTS is the most frequent complaint during pregnancy. About 15% of the cases will progress and continue several months postpartum.

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GUILLAIN–BARRÉ SYNDROME

DEFINITION

A. Guillain–Barré syndrome (GBS) is an acute immune-mediated polyneuropathy of the peripheral nervous system. GBS often follows an infection.
B. GBS is not contagious and there is no known cure.
C. GBS usually presents with ascending, progressive, multifocal, or symmetric muscle weakness, as well as paresthesia:
1. The first symptom of GBS is weakness or tingling sensations of the legs. Most people reach the stage of greatest weakness within the first 2 weeks after symptoms appear.
2. By the third week of GBS, 90% of clients are at their weakest.
D. GBS was considered monophasic and remits spontaneously but may also reoccur in 3% of clients. Twenty to thirty percent of clients will have persistent disability measured by tools such as the overall disability sum score (ODSS; Table 23.2). An electronic version of the ODSS is available online at farmacologiaclinica.info/scales/overall-disability-sum-score. This application grades the arm (range 0–5) and the leg (range 0–7) to provide an overall range score. A total score of 0 equals no disability and a total score of 12 equals maximum disability.
E. There are several variants of GBS:
1. Miller Fisher syndrome (MFS) often follows an infection, especially *Campylobacter jejuni* gastroenteritis.
2. Acute inflammatory demyelinating polyradiculoneuropathy (AIDP): Approximately two-thirds occur after an infection, including *C. jejuni*, cytomegalovirus (CMV), *Mycoplasma pneumonia*, influenza virus, or Zika virus.
3. Acute motor axonal neuropathy (AMAN).
4. Acute sensorimotor axonal neuropathy (AMSAN).
5. Acute panautonomic neuropathy (rare).
6. Bickerstaff brainstem encephalitis (BBE).

INCIDENCE

A. The incidence of GBS is 1 to 2 cases per 100,000 adults. A 20% increase is seen in every 10-year rise in age after the first year of life.
B. The lifetime individual incidence is 1:100,000.
C. GBS occurs in all age groups. GBS is more common in older adults, with those older than 50 years of age at greatest risk.
D. 80% to 90% become nonambulatory during the illness.
E. Relapses are not uncommon in adults who have been treated with intravenous immunoglobulin (IVIg) and plasma exchange.
F. Approximately 30% have a residual weakness after 3 years.
G. GBS severe enough to require mechanical ventilation is associated with both incomplete recovery and up to 20% mortality.
H. 5% of clients with GBS die from medical complications such as sepsis, pulmonary emboli, and cardiac arrest related to dysautonomia.
I. GBS is reported throughout the world.

TABLE 23.2 OVERALL DISABILITY SUM SCORE

Area of Body	Activities	Functional Ability Scale for Each Activity	Disability Scale Grade
Arm disability	Dressing upper part of body (excludes buttons/zippers) Washing and brushing hair Turning a key in a lock Using a knife and fork (use of spoon applies if never used a fork/knife) Doing/undoing buttons and zippers	Not affected; affected but does not prevent activity; prevents activity	0 = Normal function for all activities 1 = Minor (S/S) in one or both arms but does not affect the activity 2 = Moderate S/S in one or both arms affecting but not preventing any activity 3 = Severe S/S in one or both arms preventing at least one but not all activities 4 = Severe S/S in both arms preventing all functions, purposeful movements still possible 5 = Severe S/S in both arms preventing all purposeful movements
Leg disability	Do you have any problems walking? Do you walk with a walking aid? How do you get around for 25 feet (10 m)? • Without aid • With one stick or crutch or holding someone's arm • With two sticks or crutches or one stick and a crutch and holding someone's arm • With a wheelchair If you use a wheelchair, can you stand and walk a few steps with help? If you are restricted to bed most of the time, are you able to make some purposeful movements?	No; yes; does not apply	0 = Walking is not affected 1 = Walking is affected but does not look abnormal 2 = Walks independently but gait looks abnormal 3 = Usually uses unilateral support (stick, crutch, one arm) to walk 25 feet (10 m) 4 = Usually uses bilateral support (stick, crutch, two arms) to walk 25 feet (10 m) 5 = Usually uses a wheelchair to travel 25 feet (10 m) 6 = Restricted to wheelchair, unable to stand and walk a few steps with help but able to make some purposeful leg movements 7 = Restricted to wheelchair or bed most of the day, preventing all purposeful movements of the legs (e.g., unable to reposition legs in bed)

Source: Adapted from Merkies, I. S. J., Schmitz, P. I. M., van der Meche, F. G. A. Samihn, J., & van Doorn, P. A. (2002). Clinimetric evaluation of a new overally disability scale in immune mediated polyneuropathies. *Journal of Neurology and Neurosurgery Psychiatry, 72*, 596–601.

PATHOGENESIS

A. GBS is believed to be an immune-mediated response linked to an antecedent infection wherein a patchy demyelination of the motor component of multiple peripheral nerves occurs. This causes a failure of neuromuscular transmission and leads to abrupt, distal weakness, and symmetrical onset of paresthesias. The sensory disturbance is quickly followed by a rapid progressive limb weakness and sometimes paralysis. Most clients are able to identify a specific date of onset of sensory and motor symptoms.

PREDISPOSING FACTORS

A. Up to two-thirds of clients with GBS have experienced a viral upper respiratory infection (URI) or gastroenteritis 10 to 14 days before onset:

1. *C. jejuni* gastroenteritis (30% of infections).
2. CMV (the second most common reported infection preceding GBS [10%]).
3. Epstein–Barr virus (EBV).
4. *Mycoplasma pneumonia.*
5. HIV.
6. *Haemophilus influenza.*
7. Enteroviruses.
8. Hepatitis A and B.
9. Herpes simplex.
10. Varicella zoster virus.
11. *Chlamydophila* (formerly *Chlamydia*) pneumonia.
12. Zika polyneuropathy.

B. Trauma.
C. Surgery.
D. Parturition.
E. Immunization:

1. In 1976, there was a small increase in GBS following the flu vaccine formulated to protect against swine flu.
2. Rabies vaccine is prepared from infected brain tissue.

F. There is no genetic factor for GBS.

COMMON COMPLAINTS

A. The classic clinical features:

1. Progressive, fairly symmetric muscle weakness (symmetric):
a. Difficulty walking.
b. Nearly complete paralysis, including the following:
i. All extremities, generally starting in the proximal legs.
ii. Facial muscles/oropharyngeal weakness.
iii. Respiratory muscles (dyspnea on exertion and shortness of breath [SOB]).
iv. Bulbar (ocular) muscles.

2. Accompanying absent or depressed deep tendon reflexes (DTRs). ▶

B. Acute weakness in hands, dropping things, trouble picking up small objects or buttoning buttons, inability to feel textures.
C. Acute onset of persistent tingling or pins and needles, "crawling-skin" sensation in feet, possibly in hands, and inability to feel pain.
D. Pain most severe in lower back pain, buttocks, thighs, and shoulder girdle.

OTHER SIGNS AND SYMPTOMS

A. Sinus tachycardia or other arrhythmias.
B. Bilateral, generally symmetric muscle weakness not improved by rest.
C. Urinary retention.
D. Ileus-gastric motility disorders.
E. Severe residual fatigue that may persist for years.
F. Loss of sweating.
G. Facial or pharyngeal weakness.

SUBJECTIVE DATA

A. Ask the client about any dyspnea. If there is SOB, assess the need to go immediately by ambulance to a hospital.
B. Elicit information regarding the onset and duration of symptoms.
C. Question the client regarding change or progression of symptoms.
D. Ascertain if the client has had recent URI, flu, gastroenteritis, other infections, recent trauma, or surgery: Determine if there was an associated fever.
E. Look for paresthesias preceding weakness by approximately 24 to 48 hours.
F. Look for recent exposure to environmental hazards: lead, pesticides, volatile solvents, or ticks.

PHYSICAL EXAMINATION

A. Check temperature (if indicated), pulse, respirations, and blood pressure (BP; persistent hypertension [HTN] alternating with hypotension, or orthostatic hypotension). HTN is seen in about one-third of clients with GBS and can be labile or followed by hypotension.
B. Inspect:
  1. Observe overall appearance.
  2. Look for gait disturbance
  3. Assess for respiratory distress.
C. Auscultate:
  1. Note heart rate and rhythm: Tachycardia is common; bradycardia and other arrhythmias may be noted.
  2. Auscultate the lung fields.
  3. Abdomen assessment for bowel sounds/dysfunction. Gastrointestinal motility disorders occur in 15% of severely affected persons with GBS.
D. Palpate:
  1. Palpate the extremities.
  2. Note muscle tone, normal muscle bulk.
  3. Assess DTRs, muscle strength, areflexia (lack of reflexes), or hyporeflexia (diminished), although one-third of clients may have hyperreflexia.
  4. Look for symmetric weakness. Incidence of weakness is greater in the ankle and knee than in biceps and triceps.
  5. Palpate the abdomen: Assess urinary retention.
D. Neurologic exam:
  1. Perform complete neurologic exam: Generally no sensory deficits to touch or pinprick are noted. Decreased proprioception or vibration may be seen. In approximately 50% of clients, GBS may progress rapidly, sometimes within hours, to severe respiratory muscle weakness and respiratory failure.

DIAGNOSTIC TESTS

A. Prompt treatment mandates that the clinician make the diagnosis of GBS solely on history and examination. The presence of distal paresthesia increases the likelihood that the diagnosis is GBS. Testing done in the inpatient setting includes the following:
  1. Lumbar puncture (LP):
    a. LP primarily rules out other infectious disease.
    b. Most spinal fluid results for clients with GBS have elevated cerebrospinal fluid (CSF) protein levels with normal CSF cell counts.
  2. Needle electromyogram.
  3. Nerve conduction studies used to confirm the presence, pattern, and severity of neuropathy.
  4. Antibody testing.
  5. Serum testing of electrolytes, liver function tests, creatine phosphokinase (CPK), and erythrocyte sedimentation rate (ESR).

DIFFERENTIAL DIAGNOSES

A. GBS.
B. Myasthenia gravis (MG).
C. Poliomyelitis.
D. Acute intermittent porphyria.
E. West Nile encephalomyelitis.
F. Tick paralysis: Lyme neuroborreliosis.
G. Diphtheria.
H. HIV.
I. Cervical myelopathy.
J. Sarcoidosis.
K. Poisoning:
  1. Heavy metal poisoning (arsenic, lead, thallium).
  2. Hexacarbon abuse (glue sniffing neuropathy).
  3. Organophosphate poisoning.
  4. Botulism.

PLAN

A. General interventions:
  1. Early diagnosis is crucial to appropriate management. A possible diagnosis of GBS requires *immediate* hospitalization at a facility with ICU capabilities and consultation with a neurologist who has experience managing GBS.
B. Hospital medical management:
  1. Plasmapheresis (plasma exchange).
  2. IVIg administration.
  3. Steroids have not been shown to be helpful and may be detrimental.
  4. Due to the associated autonomic instability, HTN should be treated with short-acting intravenous (IV) agents.
  5. The presence of at least four of the six predictors indicates the need for support/mechanical ventilation:
    a. Onset of symptoms less than 7 days.
    b. Inability to cough.
    c. Inability to stand.
    d. Inability to lift the elbows.
    e. Inability to lift the head.
    f. An increase in liver enzymes.
  6. Heparin and support/pressure stockings are used for nonambulatory clients due to the risk of deep vein thrombosis (DVT) and pulmonary embolus. ▶

C. Pharmaceutical therapy:
1. Pain therapy:
   a. Gabapentin (Neurontin) 15 mg/kg/d.
   b. Carbamazepine (Tegretol) 300 mg/d.
   c. Narcotics may be necessary.
   d. Tricyclic antidepressants (TCAs).
2. Immunizations:
   a. Immunizations are not recommended during the acute phase and up to approximately 1 year after the onset of GBS.
   b. Influenza, tetanus, and typhoid immunizations have been most commonly associated with relapse of GBS symptoms.

FOLLOW-UP

A. Clients generally follow up with a neurologist once they have been discharged from the hospital or rehabilitation facility.
B. Full recovery can take up to 3 years with severe cases, and a small percentage of clients experience recurrence. Severe fatigue is a sequel of GBS in two-thirds of adults.
C. The most critical part of treatment consists of keeping the client's body functioning during recovery of the nervous system. Physical rehabilitation with a multidisciplinary team including occupational, speech, and physical therapists focuses on proper limb positioning, posture, orthotics, exercise, and strengthening swallowing muscles:
1. Foot and wrist drop is not uncommon and may require orthotics.
2. Joint contracture requires active and passive range of motion (ROM).

D. Psychological counseling may be required to help with adaptation.
E. The Centers for Disease Control and Prevention (CDC) has set up the Vaccine Adverse Event Reporting System (VAERS) to monitor vaccine safety. The CDC and the U.S. Food and Drug Administration (FDA) comanage the VAERS as an early warning system about possible side effects noted following immunization.

CONSULTATION/REFERRAL

A. Consult with a physician if GBS is suspected.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. Age older than 55 years is a poor prognostic factor.

RESOURCES

CDC Vaccine Adverse Event Reporting System (VAERS): http://vaers.hhs.gov
GBS/CIDP Foundation International: www.gbs-cidp.org

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HEADACHE

DEFINITION

A. Headache is a discomfort of the head that is produced from inflammation and/or tightness of the arteries, nerves, and/or muscles of the cranium. Headaches are a major cause for missed school and work, loss of productivity at work (presenteeism), and disability in adults.
B. There are multiple types of headaches with many subcategories defined by the International Classification of Headache Disorders (ICHD). Refer to the ICHD-3 website at www.ichd-3.org for a full review of the description of each headache criteria and sub category definitions:
1. Primary headaches include migraines, tension-type headaches (TTH), trigeminal autonomic cephalalgias (TACs), and other primary headache disorders. Other primary headache disorders encompass those induced by coughing, exercise, sexual activity, cold stimulus, and external pressure, as well as primary stabbing headache, thunderclap headache, nummular headache, hypnic headache, and new daily persistent headache (NDPH).
2. Secondary headaches include those attributed to trauma or injury to the head and/or neck, cranial or cervical vascular disorder, nonvascular intracranial disorder, at substance withdrawal, infection, disorder of homeostasis, or psychiatric disorder, as well as headache or facial pain attributed to disorders of the cranium, neck, eyes, ears, nose, sinuses, teeth, mouth, or other facial or cervical structure.
3. Painful cranial neuropathies, other facial pains and other headaches, and other headache disorders.

INCIDENCE

A. Headaches are very common, and their incidence depends on age, gender, and type of headache. Headaches may be self-limiting or refractory to aggressive treatment programs.
1. TTHs, the most common primary headaches, affect 31% to 74% of the population.
2. The prevalence of cluster headaches is less than 1% of the population, with males affected more than females.
3. Chronic daily headaches are more common in females than in males.
4. Medication overuse headaches (MOHs) are reported in 20% to 36% of adolescents with daily headaches.

PATHOGENESIS

A. Because there are different types of headaches, the origin of each type differs. Many people have a combination of the different types of headaches. Headache causes range from systemic illness, such as infections to medical disorders, tumors or hemorrhage, medications, drug use, and/or stress. Tension headaches occur because of contracted muscles of the scalp and neck. Cluster headaches have an uncertain etiology; however, they appear to be caused by extracerebral vasodilation.
B. Review environmental/seasonal factors. Headaches may be cyclic in the spring and summer months with allergic rhinitis and in the fall and winter with carbon monoxide poisoning from gas heaters.
C. Medications are associated with headaches; examples include the following:
1. Nitroglycerine.
2. Nifedipine.
3. Dipyridamole.
4. Selective serotonin reuptake inhibitor (SSRIs).

PREDISPOSING FACTORS

A. Tension, stress.
B. Cervical, or back, disorders.
C. Medications (e.g., nitroglycerine).
D. Bruxism.
E. Sleep disorders (e.g., sleep apnea, snoring, insomnia). ▶

F. Foods/caffeine/alcohol.
G. Hormonal changes.
H. Family history of headaches.
I. Sexual activity.
J. Cough.
K. Exertion/exercise.
L. Viral/infectious etiologies.
M. Poor-fitting dentures.
N. Faulty/inefficient gas heating.
O. Trigeminal neuralgia (TN).
P. Valsalva maneuvers.
Q. Head trauma.

COMMON COMPLAINTS

A. Pain and location depend on the type of headache.
B. Characteristics of the headache depend on the type of headache.
C. Depending on the type of headache, other symptoms may coexist, such as lacrimation, nasal congestion, restlessness, and visual changes.

SUBJECTIVE DATA

A. Use the acronym PQRST for subjective information:
1. P: Provocation, or worsening of factors stimulating headaches.
2. Q: Quality of pain, severity of pain.
3. R: Region of headache.
4. S: Strength of pain; evaluate pain on scale of 0 to 10.
5. T: Time, including onset, frequency, and duration of headaches.
B. Assess whether the client has migraine headaches frequently. Is this the first or worst headache ever experienced by the client?
C. If recurrent headaches exist, note frequency and patterns of similar headaches.
D. Note whether the client has ever identified potential triggers of recurring headaches such as dietary, stressors, and odors (e.g., perfumes, cigarette smoke).
E. Identify the location of pain, along with radiation if present.
F. Describe the type of pain: throbbing, constant, or burning.
G. Assess the presence of associated symptoms: nausea or vomiting, photophobia, noise sensitivity, or the presence of halos around lights.
H. Determine whether the client experiences any neurologic symptoms and/or prodromal symptoms prior to a headache.
I. Review the methods used in the past to abort and/or prevent headaches and the results.
J. Inquire about past diagnostic evaluations for headaches.
K. Note a family history of headaches.
L. List current medications, including over-the-counter (OTC) medications and herbals.
M. Review the client's medical history for head trauma, allergies, presence of a ventriculoperitoneal (VP) shunt, or other neurologic diagnoses.
N. Is the client in the second or third trimester of pregnancy?
O. Does the client present with a fever or have a recent history of infection?
P. Rule out gas exposure.

PHYSICAL EXAMINATION

A. Physical exam may be normal unless client presents with a headache.
B. Check blood pressure (BP) pulse, and respirations (temperature if meningeal signs are present).
C. Inspect:
1. Observe overall appearance for the presence of discomfort, photosensitivity (use of sunglasses indoors), and level of consciousness (LOC).
2. Examine the eyes; perform funduscopic exam.
3. Inspect the ears, nose, and throat.
D. Auscultate:
1. Listen for bruit at neck, eyes, and head for clinical signs of arteriovenous malformation (AVM).
E. Palpate:
1. Palpate the head, eyes, ears, temporomandibular joint (TMJ) syndrome, sinus cavities, temporal and neck arteries.
2. Palpate cervical vertebrae, cervical muscles, and shoulder regions. Identify potential trigger areas: Occipital nerves leave halfway between the middle of the neck at the back of the neck and lateral to this area. When palpating this trigger area, pain may be reproduced with palpation.
3. Examine the spine and neck muscles.
4. Assess cervical range of motion (ROM).
F. Neurologic exam.
1. Extraocular movements (EOMs).
2. Pupil response.
3. Getting up from a seated position without any support.
4. Walking on tiptoes and heels.
5. Tandem gait.
6. Romberg test.
7. Symmetry on motor, sensory, deep tendon reflexes (DTRs), and coordination tests.
8. Perform neck flexion for nuchal rigidity.

DIAGNOSTIC TESTS

A. Tests are selected based on history and physical exam:
1. Sinus films to rule out sinusitis or a lesion.
2. Sleep studies for obstructive sleep apnea.
3. CT scan or MRI: needed if headache is severe, no results are achieved with drug therapy, and/or aura is present.
4. People with any positive neurologic signs of an intracranial process should have neuroimaging.
5. Lab tests are rarely needed for headaches, unless an infectious process is suspected.

DIFFERENTIAL DIAGNOSES

A. Headache:
1. Tension.
2. Cluster.
3. MOH.
4. Migraine.
5. Combination headache.
6. NDPH.
7. Hypnic headache (occurs only in the elderly).
B. Sinusitis.
C. Meningitis: meningism, acute headache with fever, lethargy, nausea or vomiting, irritability, photophobia, and systemic infection.
D. Space-occupying lesion: subacute and progressive pain, new onset for adults older than 40 years.
E. Temporal arteritis: new-onset progressive headache for adults older than 50 years, with presenting symptoms of temporal artery swelling, pain, pulselessness, visual changes, mental sluggishness, systemic symptoms (fever, anorexia, malaise), and erythrocyte sedimentation rate (ESR) greater than 50 mm/hr. ▶

F. Carotid dissection: sudden headache with neck pain, radiating to the face, ear, or eye; onset related to neck movement or trauma, Horner syndrome, tinnitus, ipsilateral tongue weakness, cervical bruit or tenderness, diplopia, and syncope.
G. TMJ syndrome: jaw claudication, clicking, and locking sensation, ill-fitting dentures.
H. Carbon monoxide poisoning.
I. Temporal arteritis.
J. Trigeminal neural.
K. Pregnancy-induced hypertension (PIH).
L. Medication-induced headache: Review side effects of current medications (individual and/or combination of drugs).

PLAN

A. General interventions:
1. Encourage the client to restrict associated triggers, such as food, alcohol, and exposure to odors.
2. Encourage the client to exercise daily.
3. Have the client begin a stress management routine, including yoga, meditation, and massage.
4. Tell the client to take medications as prescribed.
5. Cluster headaches can be exacerbated by alcohol.
6. Use ice/heat for muscular tension.
7. Individual and/or family psychotherapy should be considered.
8. Complementary and alternative medicine (CAM):
a. Nutraceutical options:
i. Magnesium 400 to 600 mg/d.
ii. Riboflavin 400 mg/d.
iii. Coenzyme Q10.
iv. Alpha lipoic acid.
b. Herbal preparations:
i. Feverfew 50 to 82 mg/d.
ii. *Petasites hybridus* (butterbur).
iii. *Cannabis* (marijuana).
c. Acupuncture.
d. Oxygen/hyperbaric oxygen therapy.
e. Transcutaneous electrical nerve stimulation (TENS) unit.
f. Chiropractic manipulation.
g. Physical therapy.
h. Continuous positive airway pressure (CPAP).
i. Biofeedback.
j. Relaxation training.

B. Client teaching:
1. Encourage the client to keep a diary of headaches and associated factors to try to pinpoint headache triggers.
2. Teach clients who have menstrual headaches to avoid precipitating factors, such as alcohol, tyramine, or phenylethylamine foods; missed meals; and sleeping late.
3. Discuss sleep hygiene guidelines (see section "Sleep Disorders" of Chapter 25, "Psychiatric Guidelines").
4. For muscular headaches that are nonmenstrual, biofeedback, breathing exercises, and visualization are helpful. Prevention must be stressed. Encourage lifestyle changes and daily exercise.
5. When clients overuse various analgesics for headaches, paroxysmal migraines can convert into chronic daily headaches. Caution clients regarding this effect.
6. MOHs occur with the highest incidence involving opioids, butalbital-containing combinations, and acetylsalicylic acid (aspirin)/acetaminophen/caffeine combinations, as well as triptans. Withdrawal of the overused medication is the treatment of choice for MOHs.

C. Pharmaceutical therapy: Therapy should be started at the lowest dosage and titrated up as tolerated, avoiding overuse. Examples of common medications for treatment of headaches include the following:
1. Nonsteroidal anti-inflammatory drugs (NSAIDs):
a. Aspirin (ASA) 650 to 1,000 mg orally; maximal dose 4 g/d.
b. Ibuprofen (Advil) 400 to 800 mg Q8H; maximum dose 2.4 g/d.
2. Combination medications:
a. Acetaminophen, butalbital, and caffeine (Fioricet) with or without codeine one to two tablets Q4H as needed. Maximum dose is six tablets per day.
b. ASA, butalbital, and caffeine (Fiorinal) one or two tablets Q4H as needed. Caution the client regarding dependency patterns.
c. Isometheptene mucate, dichloralphenazone, and acetaminophen (Epidrin or Migratine) one to two capsules Q4H if needed, up to eight capsules in a 24-hour period for tension headaches in adults.
d. Acetaminophen, ASA, and caffeine (Excedrin) two tablets Q6H. Maximal dose of 4 g of aspirin or acetaminophen.
3. Cluster or vascular headaches:
a. Verapamil is the agent of choice for prevention therapy with cluster headaches.
b. Propranolol (Inderal) or carbamazepine 80 mg daily in divided doses. Maximum range 160 to 240 mg daily.
c. Oxygen 100% therapy has been effective for cluster headaches.
4. Menstrual headaches:
a. Estrogen supplements or continuous cycling is used to decrease headaches. Have the client start taking estrogen 2 days before expected migraine and use for 7 days.
b. Naproxen 275 to 550 mg every 2 to 6 hours (maximum dose 1.5 mg/d) starting 7 days as needed before menses.
c. Ibuprofen 600 mg TID.
d. Fluoxetine (Prozac) 10 to 20 mg daily is also used for clients with premenstrual syndrome with luteal phase defect.
5. Antidepressants may also be utilized for preventive treatment.
6. Pharmaceutical therapies for chronic daily and NDPHs combine therapies that are used for tension-type and migraine headaches.

FOLLOW-UP

A. See the client in 2 weeks to evaluate how therapies have worked.
B. Evaluate the client's "headache log" to assist him or her in identifying headache triggers, if present.

CONSULTATION/REFERRAL

A. Consult a physician if headaches are caused by acute problems other than migraine and/or tension headaches.
B. Severe episodes need to be evaluated by a physician for opioid agonists and antagonists, narcotics, and neuroleptics.
C. If medications do not help with headaches, refer the client to a neurologist.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. PIH often presents with a headache. ▶

B. Adults:

1. Clients who are premenopausal may see improvement when their estrogen levels are constant, rather than cyclic.

C. Geriatrics:

1. The most common primary headache types in the elderly are tension, migraine, late-life migraine, and cluster.

2. Hypnic headache (alarm clock headache), a rare primary headache, occurs in elderly persons. Hypnic headaches occur exclusively at night, awaking the client with dull, throbbing, pulsating, sharp, or stabbing pain lasting approximately 3 hours. Symptoms include nausea, vomiting, phonophotophobia, lacrimation, ptosis, rhinorrhea, or nasal congestion.

3. Headaches generally decrease with age; however, hypnic headaches only occur in the elderly. Hypnic headaches awaken clients from sleep and are short-lived.

4. Serious secondary causes of headaches that increase with age include those associated with the following:

a. Temporal arteritis.
b. TN.
c. Sleep apnea.
d. Postherpetic neuralgia.
e. Cervical spondylosis.
f. Subarachnoid hemorrhage (SAH).
g. Intracerebral hemorrhage.
h. Intracranial neoplasm.
i. Postconcussive syndrome.

5. Consider imaging studies in the elderly with unusual presentations of headaches.

6. Consider chronic subdural hematomas (SDHs) with clients who have frequent falls; perform CT scan or MRI for evaluation.

7. Elderly clients may not exhibit any symptoms other than a headache.

8. When medicating the elderly, start dosages low and increase as tolerated:

a. Naproxen and hydroxyzine are commonly used oral rescue therapies for older adults with migraine or tension headaches.

9. Oral agents for prevention of hypnic headaches include caffeine and lithium.

10. Cough headaches respond to indomethacin or acetazolamide.

11. Beers medication cautions:

a. Consider all contraindications when prescribing medications for headaches in the elderly.
b. In the presence of cardiovascular disease, ergot derivatives are contraindicated.
c. Amitriptyline and doxepin are not recommended for older individuals due to the risk of cognitive impairment, urinary retention, and cardiac arrhythmia.
d. Consider renal function prior to prescribing NSAIDs.

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MIGRAINE HEADACHE

DEFINITION

A. Migraine headaches are a common medical complaint responsible for a significant disability and loss of quality of life (QOL). The economic impact involves loss of workdays, school, social interaction, and productivity while at work (presenteeism). Migraines are ranked the third highest cause of disability worldwide in both males and females under 50 year of age.

B. Migraines have two major types, migraine without aura and migraine with aura:

1. Migraine without aura is a clinical syndrome characterized by headache with specific features and associated symptoms:

a. Recurrent headache disorder manifesting in attacks lasting 4 to 72 hours. Typical characteristics of the headache are unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity, and association with nausea and/or photophobia and phonophobia.

2. Migraine with aura is primarily characterized by the transient focal neurologic symptoms that usually precede or sometimes accompany the headache. Some clients also experience a prodromal phase, occurring hours or days before the headache, and/or a postdromal phase following headache resolution. Prodromal and postdromal symptoms include hyperactivity, hypoactivity, depression, cravings for particular foods, repetitive yawning, fatigue, and neck stiffness and/or pain.

C. There are multiple subtypes of migraines described by the International Headache Society (IHS) by type and diagnostic criteria.

D. Migraine headaches have been associated with increased risk of cerebral ischemia and an increased risk of cardiac ischemia.

E. Migraine and epilepsy are prototypical examples of paroxysmal brain disorders. Although migraine-like headaches are quite frequently seen in the epileptic postictal period, sometimes a seizure occurs during or following a migraine attack. This phenomenon, sometimes referred to as "migralepsy," is a rare event.

INCIDENCE

A. Headaches are one of the most common medical complaints. The exact incidence of migraines is unknown because clients self-treat, are underdiagnosed, and are commonly misdiagnosed.

B. It is estimated that more than 30 million people have one or more migraine headaches per year.

C. Sex: 64% of severe headaches occur in females and 43% of severe headaches in males. Approximately 75% of all persons who experience migraines are female.

D. Ethnicity: The prevalence of migraines is lowest among African Americans and Asian Americans.

E. Onset of migraines after age 50 is rare.

F. Migraines and ischemic strokes reportedly occur in 1.4 to 3.3 per 100,000 and account for 0.8% of total strokes.

G. Migraineurs, especially those with migraine with aura, have a higher incidence of adverse cardiovascular profiles.

PATHOGENESIS

A. Migraines have broad sensory processing dysfunction, with a prominent perception of pain in the dense somatosensory innervation of intracranial vessels. Current pathophysiologic ▶

concepts of migraine and migraine aura include a possible dysfunction of neuromodulatory structures in the brainstem and cortical spreading depression (CSD). Different receptors, including calcitonin gene-related peptide (CGRP), transient receptor potential cation channel subfamily V member (TrpVI [also known as the capsaicin receptor]), and glutamate receptors, are currently being targeted for migraine therapeutics.

PREDISPOSING FACTORS

A. Family history of migraines.
B. Chronic use of over-the-counter (OTC) analgesics (rebound).
C. Posthead trauma.
D. Food, odor, light, sound, sleep, weather changes, hormonal changes, and stress triggers.
E. Menstruation.
F. Obesity.
G. Daily habitual snoring is a modest risk factor.
H. Estrogen use.

COMMON COMPLAINTS

A. Unilateral headache.
B. Frontotemporal area.
C. Photophobia, or sensitivity to light.
D. Phonophobia, or sensitivity to sound.
E. Osmophobia, or hypersensitivity/aversion to odors.
F. Nausea with/without vomiting.
G. Prodrome phase: fatigue, reduced concentration, agitation, craving, irritability, depression, frequent yawning, or hyperexcitability hours to days before the onset of aura and headache.

OTHER SIGNS AND SYMPTOMS

A. Muscle tension and neck pain.
B. Cutaneous allodynia (pain from stimulus to normal skin or scalp).
C. Sinus congestion.
D. Prodrome phase can last 25 hours accompanied by fatigue and a "hangover" headache.

SUBJECTIVE DATA

A. Use the acronym PQRST for subjective information:
  1. P: Provocation, or worsening of factors stimulating headaches.
  2. Q: Quality of pain, severity of pain.
  3. R: Region of headache.
  4. S: Strength of pain; evaluate pain on a scale of 0 to 10.
  5. T: Time, including onset, frequency, and duration of headaches.
B. Assess whether the client has migraine headaches frequently. Is this the first or worst headache ever experienced by the client?
C. If recurrent headaches exist, note frequency and patterns of similar headaches.
D. Note whether the client has ever identified potential triggers of recurring headaches such as menstruation, diet, stressors, and odors (e.g., perfumes and cigarette smoke).
E. Identify the location of the pain, along with radiation if present.
F. Describe the type of pain: throbbing, constant, or burning.
G. Assess the presence of associated symptoms: nausea or vomiting, photophobia, and noise sensitivity.
H. Determine whether the client experiences any neurologic symptoms and/or prodromal symptoms prior to headache.
I. Review the methods used in the past to abort and/or prevent headaches and the results.
J. Inquire about past diagnostic evaluations for headaches.
K. Note a family history of headaches.
L. List current medications, including OTC medications and herbal products.
M. Review the client's medical history for head trauma, infection, allergies, presence of a ventriculoperitoneal (VP) shunt, or other neurologic diagnoses.

PHYSICAL EXAMINATION

A. Physical exam may be normal unless the client presents with a headache.
B. Check blood pressure (BP), pulse, and respirations (temperature if meningeal signs are present).
C. Inspect:
  1. Observe overall appearance for the presence of discomfort, photosensitivity (use of sunglasses indoors), and level of consciousness (LOC).
  2. Examine the eyes; perform funduscopic exam.
  3. Inspect the ears, nose, and throat.
D. Auscultate:
  1. Listen for bruit at neck, eyes, and head for clinical signs of atrioventricular (AV) malformation.
E. Palpate:
  1. Palpate the head, eyes, ears, temporomandibular joint (TMJ), sinus cavities, temporal and neck arteries.
  2. Palpate cervical vertebrae, cervical muscles, and shoulder regions. Identify potential trigger areas: Occipital nerves leave halfway between the middle of the neck at the back of the neck and lateral to this area. When palpating this trigger area, pain may be reproduced with palpation.
  3. Examine the spine and neck muscles.
  4. Assess the cervical range of motion (ROM).
F. Neurologic exam:
  1. Extraocular muscles (EOM).
  2. Pupil response.
  3. Getting up from a seated position without any support.
  4. Walking on tiptoes and heels.
  5. Tandem gait.
  6. Romberg test.
  7. Symmetry on motor, sensory, deep tendon reflexes (DTRs), and coordination tests.

DIAGNOSTIC TESTS

A. Neuroimaging, MRI and CT, is based on client history and physical examination:
  1. Adults with stable headaches, a normal examination, and absence of seizures do not require neuroimaging.
  2. An emergent noncontrast CT should be obtained in an emergency setting when the client complains of "the worst headache ever" or when focal neurologic findings, nuchal rigidity, or altered mental status exist.
  3. The presence of personality changes, depression, and a migraine may indicate a temporal lobe tumor.
  4. The presence of orbital bruit requires neuroimaging.
  5. Neuroimaging is recommended for adults with onset of headache after age 40.
  6. Onset of headache with exertion, cough, or sexual activity should be considered for neuroimaging.
B. A lumbar puncture (LP) may be indicated with altered mental status or focal findings.
C. Order sinus films to rule out sinusitis or a lesion. ▶

D. Laboratory tests are not required for most clients with typical symptoms and a negative physical examination.
1. Drug screen may be indicated.
2. Complete metabolic panel (CMP).
3. Complete blood count (CBC).
4. Thyroid-stimulating hormone (TSH).
5. Sedimentation rate.

DIFFERENTIAL DIAGNOSES

A. Migraine:
1. Migraine with aura.
2. Migraine without aura.

B. Other types of headaches:
1. Medication overuse headache (MOH).
2. Common headache.
3. Cluster headache.
4. Combination headache.
5. Chronic daily headache.
6. Tension headache.
7. Hypnic headaches (geriatrics).

C. Sinusitis.

D. Space-occupying lesion: subacute and progressive pain, new onset for adults older than 40 years.

E. Temporal arteritis: new-onset progressive headache for adults older than 50 years.

F. Carotid dissection: sudden headache with neck pain radiating to the face, ear, or eye.

G. TMJ syndrome.

H. Meningitis.

I. Brain abscess.

J. Encephalitis.

K. Idiopathic intracranial hypertension (HTN).

PLAN

A. General interventions: There are four main approaches to migraine therapy:
1. Nonpharmacologic interventions:
   a. Adjust habits to maintain a routine pattern of sleeping. This is especially important to maintain on weekends and vacations.
   b. Do not skip breakfast. Eat regular meals with one or two snacks.
   c. Avoid food triggers identified by the client's migraine diary.
   d. Encourage drinking no more than two caffeinated beverages a day.
   e. Hydration is important.
   f. Encourage at least 30 minutes of exercise 3 to 7 days a week.
   g. Cold compress.
2. Behavioral interventions:
   a. Use relaxation techniques such as yoga, deep breathing, meditation, and guided imagery.
   b. Biofeedback is an adjunct to relaxation training.
   c. Cognitive behavioral therapy (CBT).
   d. Psychiatric therapy.
3. Complementary and alternative interventions:
   a. Acupuncture.
   b. Nutraceuticals, including magnesium and coenzyme Q10.
   c. Vitamins: riboflavin (B_2).
   d. Herbal (nonregulated by the Food and Drug Administration [FDA]), including feverfew, *Petasites* (purified extract of butterbur root extract), and MIG-99 (extract of *Tanacetum parthenium* [feverfew]).
   e. Physical therapy.
   f. Hypnosis.
   g. Cerena Transcranial Magnetic Stimulator (Cerena TMS) is used to relieve pain caused by migraine headache with aura for individuals 18 years and older. Users hold the device with both hands to the back of the head and press a button to release a pulse of magnetic energy that stimulates the occipital cortex. The recommended daily usage of the device is not to exceed one treatment in 24 hours.
   h. In January 2018, the FDA approved a vagus nerve stimulator (VNS) for treatment of migraine pain in adults. The handheld, noninvasive device was previously approved for treating episodic cluster headache pain. The prescription-only device is placed over the vagus nerve in the neck and releases a mild electrical stimulation to the nerve's afferent fibers.
   i. Chiropractic manipulation and occlusal adjustment are also noted in the literature.

B. Client teaching: *See Client Teaching Guide for this chapter, "Migraine Headaches."*
1. Encourage the client to keep a headache diary; an example of a migraine diary is at www.webmd.com/migraines-headaches/guide/headache-diary.
   a. Examples of food triggers are aspartame, saccharin, red wine, alcohol, chocolate, aged cheese, oranges, tomatoes, avocado, nuts, onions, tyramine, phenylethylamine, monosodium glutamate (MSG), and nitrates as well as nitrites found in hot dogs, luncheon meat, and sausage.
   b. Examples of odor triggers are tobacco smoke, perfumes, and strong odors.
   c. Examples of visual triggers include strobe lights, bright lights/sunlight, fluorescent lights, and glare.
   d. Other triggers are medications, barometric weather changes, too much/too little sleep, and high altitude.
   e. The 2014 American Heart Association (AHA)/American Stroke Association (ASA) guidelines for prevention of strokes in females state that due to the increased stroke risk seen in females with migraine headaches with aura and smoking, it is reasonable to strongly recommend smoking cessation.

C. Pharmaceutical therapy (Table 23.3): The choice of drug therapy prophylactic agents depends on the client's comorbid conditions such as cardiac, respiratory, psychiatric, sleep, and gastrointestinal (GI) disorders:
1. Abortive medications:
   a. Selective serotonin receptor (5-hydroxytryptamine-1, or 5-HT1) agonists (triptans):
      i. Sumatriptan (Imitrex, Sumavel DosePro, Alsuma, Sumavel, Zembrace).
      ii. Sumatriptan and naproxen (Treximet).
      iii. Sumatriptan intranasal (Imitrex Intranasal, Onzetra Xsail).
      iv. Sumatriptan transdermal (Zecuity).
      v. Naratriptan (Amerge).
      vi. Zolmitriptan (Zomig, Zomig-ZMT).
      vii. Rizatriptan (Maxalt, Maxalt-MLT).
      viii. Almotriptan (Axert).
      ix. Frovatriptan (Frova).
      x. Eletriptan (Relpax).
   b. Ergot alkaloids:
      i. Ergotamine tartrate (Ergomar).
      ii. Dihydroergotamine (DHE-45, Migranal).

c. Analgesics:
   i. Acetaminophen.
   ii. Aspirin-Free Anacin Extra Strength.
d. Nonsteroidal anti-inflammatory drugs (NSAIDs):
   i. Aspirin (Bayer, Bufferin, Ecotrin, Ascriptin).
   ii. Ketorolac intranasal (Sprix).
   iii. Ibuprofen (Motrin, Advil, NeoProfen, Caldolor).
   iv. Naproxen (Naprosyn, Anaprox, Aleve, Midol Extended Relief).
   v. Ketoprofen (Active-Ketoprofen, Ketophene RapidPaq).
e. Combination products:
   i. Excedrin (acetaminophen, aspirin, caffeine).
   ii. Codeine/acetaminophen (Tylenol #3, Tylenol #4).
   iii. Isometheptene, dichloraphenazone, and acetaminophen (Midrin, Epidrin).
   iv. Butalbital, aspirin, and caffeine (Fiorinal).
   v. Butalbital, acetaminophen, and caffeine (Fioricet).
f. Antiemetics:
   i. Promethazine (Phenergan).
   ii. Prochlorperazine (Compro, Compazine). ▶

TABLE 23.3 MEDICATIONS FOR MIGRAINE HEADACHES

Abortive Medications	Prophylactic Medicines
Selective serotonin receptor (5-HT1) agonists (triptans) • Sumatriptan (Imitrex, Sumavel DosePro, Alsuma, Sumavel, Zembrace) • Sumatriptan and naproxen (Treximet) • Sumatriptan intranasal (Imitrex Intranasal, Onzetra Xsail) • Sumatriptan transdermal (Zecuity) • Naratriptan (Amerge) • Zolmitriptan (Zomig, Zomig-ZMT) • Rizatriptan (Maxalt, Maxalt-MLT) • Almotriptan (Axert) • Frovatriptan (Frova) • Eletriptan (Relpax) Ergot alkaloids • Ergotomine tartrate (Ergomar) • DHE-45 (Migranal) Analgesics • Acetaminophen • Aspirin-Free Anacin Extra Strength NSAIDs • Aspirin (Bayer, Bufferin, Ecotrin, Ascriptin) • Ketorolac intranasal (Sprix) • Ibuprofen (Motrin, Advil, NeoProfen, Caldolor) • Naproxen (Naprosyn, Anaprox, Aleve, Midol Extended Relief) • Ketoprofen (Active-Ketoprofen, Ketophene RapidPaq) Combination products • Excedrin (acetaminophen, aspirin, caffeine) • Codeine/acetaminophen (Tylenol #3, Tylenol #4) • Isometheptene, dichloralphenazone, and acetaminophen (Midrin, Epidrin) • Butalbital, aspirin, and caffeine (Fiorinal) • Butalbital, acetaminophen, and caffeine (Filoricet) Antiemetics • Promethazine (Phenergan) • Prochlorperazine (Compro, Compazine) Opioids (not first-line treatment—prescribe sparingly to clients who do not respond to routine abortive treatment) • Morphine (drug of choice; Arymo ER, Astramorph, DepoDur, MS Contin, Duramorph, Avinza, Infumorph, Kadian, MorphaBond) • Oxycodone (OxyContin, Roxicodone, Oxecta, Oxaydo, Xtampza ER) • Meperidine (Demerol) • Hydromorphone (Dilaudid, Dilaudid-HR, Exalgo) Antipsychotic. • Chlorpromazine (Thorazine)	Antiepileptic drugs • Valproic acid (Depakote, Depacon, Depakene, Stavzor) • Topiramate (Topamax, Qudexy XR, Topiragen, Trokendi XR) Beta-blockers beta-1 selective (not a first-line agent) • Propranolol (Inderal, Inderal LA, Hemangeol, InnoPran XL) • Timolol (Apo-Timol, Nu-Timolol, Teva-Timolol) Tricyclic antidepressants • Amitriptyline (Elavil) • Nortriptyline (Pamelor) • Doxepin (Silenor) • Protriptyline (Vivactil) Calcium channel blockers • Verapamil (Calan, Isoptin SR, Verelan) SSRIs • Paroxetine (Paxil, Paxil CR, Pexeva, Brisdelle) • Fluoxetine (Prozac, Prozac Weekly, Sarafem) • Sertraline (Zoloft) NSAIDs (as previously noted) Serotonin antagonists Botulinum toxin • CGRP inhibitors • Erenumab (Aimovig) • Fremanezumab (Ajovy) • Galcanezumab (Emgality)

5-HT1, 5-hydroxytryptamine-1; CGRP, calcitonin gene-related peptide; DHE-45, dihydroergotamine; NSAIDs, nonsteroidal anti-inflammatory drugs; SSRIs, selective serotonin reuptake inhibitors.

g. **Opioids (not first-line treatment—prescribe sparingly to clients who do not respond to routine abortive treatment):**
   i. Morphine (drug of choice; Arymo ER, Astramorph, DepoDur, MS Contin, Duramorph, Avinza, Infumorph, Kadian, MorphaBond).
   ii. Oxycodone (OxyContin, Roxicodone, Oxecta, Oxaydo, Xtampza ER).
   iii. Meperidine (Demerol).
   iv. Hydromorphone (Dilaudid, Dilaudid-HR, Exalgo).
h. Antipsychotic.
i. Chlorpromazine (Thorazine).

2. Prophylactic medications:
a. Antiepileptic drugs (AEDs):
   i. Valproic acid (Depakote, Depacon, Depakene, Stavzor).
   ii. Topiramate (Topamax, Qudexy XR, Topiragen, Trokendi XR).
b. Beta-blockers beta-1 selective (not a first-line agent):
   i. Propranolol (Inderal, Inderal LA, Hemangeol, InnoPran XL).
   ii. Timolol (Apo-Timol, Nu-Timolol, Teva-Timolol).
c. Tricyclic antidepressants (TCAs):
   i. Amitriptyline (Elavil).
   ii. Nortriptyline (Pamelor).
   iii. Doxepin (Silenor).
   iv. Protriptyline (Vivactil).
d. Calcium channel blockers.
e. Verapamil (Calan, Isoptin SR, Verelan).
f. Selective serotonin reuptake inhibitors (SSRIs).
   i. Paroxetine (Paxil, Paxil CR, Pexeva, Brisdelle).
   ii. Fluoxetine (Prozac, Prozac Weekly, Sarafem).
   iii. Sertraline (Zoloft).
g. NSAIDs (as previously noted).
h. Serotonin antagonists.
i. Botulinum toxin.
j. CGRP inhibitors: erenumab (Aimovig), galcanezumab (Emgality), and atogepant (Qulipta).

3. Clients should be counseled to take medications as prescribed. When clients overuse various analgesics for headaches, paroxysmal migraines can convert into chronic daily headaches. Acute overuse of symptomatic medication is considered one of the most important risk factors for migraine progression. MOH can occur with any analgesic, including acetaminophen or NSAIDs, such as ibuprofen, naproxen, and aspirin.

4. Opioid or butalbital-containing medication should not be prescribed as first-line treatment for recurrent headache disorders. Analgesics like butalbital and narcotics are associated with rebound headaches. Increasing the use of combination preparations may fail to provide pain relief and may worsen headache symptoms.

5. NSAIDs are generally used as abortive therapy in mild to moderately severe migraine headaches. However, these agents, especially ketorolac, may also be effective for severe headaches.
a. NSAIDs are also used as prophylactic agents, but they are associated with a higher risk of adverse effects, particularly gastropathy or nephropathy, than when they are used as abortive medications.

6. Triptans are used as abortive medications for moderately severe to severe migraine headaches. Triptans should be used cautiously in clients with cardiovascular comorbidities. Drug interactions occur with potent CYP450 3A4 inhibitors (e.g., ketoconazole, itraconazole, nefazodone, troleandomycin, clarithromycin, ritonavir, nelfinavir), which may increase toxicity, and with concurrent administration of ergot-containing drugs, which may increase vasospastic reactions.

7. Use ergot derivatives selectively. These medications are not to be used on a long-term basis or more than three times per week. There is an associated risk of strokes when using these medications due to the vasoconstrictive mechanisms on peripheral and cranial blood vessels

8. Ergots and triptans should not be given within 14 days of an monoamine oxidase (MAO) inhibitor.

9. Calcium channel blockers are particularly useful in clients with comorbid hypertension and in those with contraindications to beta-blockers, such as asthma and Raynaud disease. Calcium channel blockers may have particular advantage in clients with prolonged aura, basilar-type migraine, or hemiplegic migraine. Verapamil is frequently the first choice for prophylactic therapy due to ease of use and a favorable side effect profile.

10. Beta-blockers are approved formigraine prophylaxis but are not considered a first-line therapy; however, they must be used with caution in clients with comorbidities such as asthma, depression, diabetes, and thyroid disease. Compared with other antihypertensive medications, beta-blockers pose a higher risk of cardiovascular events.

11. Antiemetic agents are used to treat migraine and the emesis associated with acute attacks. Drugs in this category are commonly combined with diphenhydramine to minimize the risk of akathisia.

12. Topiramate is indicated for migraine headache prophylaxis in adults and adolescents aged 12 years or older. Topiramate should not be prescribed or should be discontinued for a history of kidney stones.

13. As a rule, dopamine antagonists are avoided in clients with traumatic brain injury.

14. OnabotulinumtoxinA (BOTOX) is Food and Drug Administaration (FDA)-approved for chronic migraines in adults who are 18 years or over with headaches 15 or more days a month lasting 4 hours or more each day. Safety and effectiveness have not been established for the prophylaxis of episodic migraine (14 headache days for fewer per month).

15. In 2018 the FDA approved new injectable drugs to prevent migraine treatment that work by blocking the activity of calcitonin gene-related peptide (CGRP):
a. Erenumab (Aimovig).
b. Fremanezumab (Ajovy).
c. Galcanezumab (Emgality).
d. Atogepant (Qulipta).

16. Dihydroergotamine mesylate (Migranal) is not to be used in clients during pregnancy or with heart disease, or for use with ischemic or vasospastic circulatory disease.

FOLLOW-UP

A. See the client in 2 weeks to evaluate how therapies have worked. ▶

B. Evaluate the client's headache diary to assist them in identifying headache triggers or patterns, such as prior to menstruation. Utilize the information documented in the diary as a tool for reevaluating the need for other tests and consultations.

C. Monitor liver enzymes and CBC periodically with antiseizure medication prophylaxis.

CONSULTATION/REFERRAL

A. Consult a physician if headaches are caused by acute problems other than migraine and/or tension headaches.

B. If medications do not help with headaches, refer the client to a neurologist.

EMERGENT ISSUES/INSTRUCTIONS

A. Send the client to the ED for any neurologic, life-threatening signs.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Many medications are contraindicated in pregnancy.

B. Geriatrics:

1. Headaches decrease after age 50. Headache onset after age 50 is associated with epilepsy, essential tremor, ischemic stroke, mood disorders, asthma, and patent foramen ovale.

2. Consider imaging studies for elderly clients who present with unusual headaches.

3. Consider chronic subdural hematomas (SDHs) with clients who have frequent falls; perform CT scan or MRI for evaluation. Elderly clients may not exhibit any symptoms other than a headache.

4. Consider all contraindications when prescribing medications; many elderly clients have cardiovascular disease, which is contraindicated with ergot derivatives.

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MILD TRAUMATIC BRAIN INJURY

DEFINITION

A. Head injury is defined as any external structural damage (e.g., a blow, a jolt, a bump) or functional impairment of cranial content, including the scalp, skull, meninges, blood vessels, or brain. Any of the following may occur immediately after the initial injury: loss of consciousness or decreased awareness, memory loss specific to events immediately pre- or postinjury, altered mental status, and neuro deficits involving motor strength, balance, vision, sensation, and speech. Mild traumatic brain injury (mTBI) results in a disruption of brain function (altered mental status), indicating severity of the initial injury. mTBI-related deficits are often mild in nature without overt symptoms. Radiographic testing is negative for anatomic abnormality (e.g., cerebral edema, hemorrhage). *Concussion* is a commonly used term to describe mTBI, where a loss of consciousness may have occurred; confusion is also associated. Ninety percent of clients with concussive injuries do not experience decreased level of consciousness (LOC). Serious complications of mTBI include asymptomatic extradural hematomas, fatal thrombosis of the basilar artery, and hemorrhage from existing conditions such as fibrous dysplasia or essential thrombocytopenia.

B. The American Academy of Neurology offers the following guidelines for grading the severity of concussions:

1. Grade 1: transient confusion; no loss of consciousness; resolution of mental status abnormalities in less than 15 minutes.

2. Grade 2: transient confusion; no loss of consciousness; concussion symptoms or mental status abnormalities with amnesia for longer than 15 minutes.

3. Grade 3: loss of consciousness (IIIa, unresponsive period lasts seconds; IIIb, unresponsive period lasts minutes).

C. The Glasgow Coma Scale (GCS) can also be used to grade the severity of concussions. A GCS score of 13 to 15 that is measured approximately 30 minutes after the injury would be classified as a mild traumatic brain injury. The GCS can be found at www.mdcalc.com/glasgow-coma-scale-score-gcs.

INCIDENCE

A. The full incidence is unknown; many individuals with mTBI do not seek medical attention, affecting the actual number of reported cases.

B. Traumatic brain injury (TBI) is a major cause of death and disability in the United States. In 2019, there were 60,611 TBI-related deaths in 2019, which averaged to 166 deaths per day.

C. Elderly clients (>65 years) represent 10% of those with an mTBI; however, this population represents approximately 50% of TBI-related deaths.

PREDISPOSING FACTORS

A. Motor vehicle accidents (MVAs).

B. Assaults.

C. Sports- and recreation-related trauma.

D. Male sex.

E. Ages of increased incidence:

1. 0 to 4 years.

2. 15 to 19 years.

3. 65 years and older.

F. Military occupation (e.g., exposure to blasts).

G. Falls.

PATHOGENESIS

A. Head-injured clients can potentially sustain two different types of injuries: *primary* (impact) injury and *secondary* injury:

1. A primary injury is a direct result from the injury and occurs at the time of initial insult. This type of injury is purely mechanical and may be focal (contusion or laceration, bone fragmentation), or diffuse, as in concussion or diffuse axonal injury (DAI). These injuries do not require surgical intervention.

2. Secondary injury is caused by a flow–metabolism mismatch. It is a complication of primary brain damage. This includes ischemic and hypoxic damage, cerebral edema, intracranial hemorrhage (ICH), and the impact of prolonged increased intracranial pressure (ICP), hydrocephalus, and infection. Secondary injury has delayed onset, occurring in a matter of seconds, minutes, hours, or days.

COMMON COMPLAINTS

A. Headaches that are often constant, generalized, or frontal and may last days or weeks are the most common posttraumatic symptom. Headaches usually occur within the first 14 days of the mTBI. ▶

B. Brief amnestic epoch surrounding impact.
C. Faintness.
D. Nausea/vomiting.
E. Changes in vision, often slight blurring.
F. Drowsiness.
G. Loss of consciousness.
H. Confusion.

OTHER SIGNS AND SYMPTOMS

A. Other presenting symptoms and complaints are identified in four categories: physical, emotional, cognitive, and sleep-cycle disturbances:

1. Physical:
   a. Reported or observed injury to the head.
   b. Dizziness.
   c. Fatigue.
   d. Decrease/change in balance.
   e. Photophobia.
   f. Sensitivity to noise.
   g. Numbness/tingling.
   h. Seizures, delayed-onset status postinjury.
2. Emotional:
   a. Irritability.
   b. Nervousness.
   c. Depression.
3. Cognitive:
   a. Difficulty concentrating.
   b. Memory impairment:
      i. Short-term memory loss.
      ii. Repetition.
   c. Confusion.
   d. Slow responses/difficulty processing.
   e. Changes in reaction time.
   f. Changes in speech.
   g. Disorientation.
   h. Fatigue.
   i. Altered taste and smell.
4. Sleep cycle disturbance:
   a. Feeling "drowsy."
   b. Difficulty falling asleep.
   c. Sleeping more or less than usual.

SUBJECTIVE DATA

A. Obtain a description of injury from the client or witness of the traumatic event, if possible. Identify the cause of the head injury, how it occurred (direct or indirect injury), and what type of force was exerted.

B. Confirm the client's level of consciousness (LOC) at the time of injury and after injury. Inquire about amnesia (retrograde and anterograde), which might predict increased severity of the injury. Ask about the occurrence, whether it was observed by others, and duration.

C. Review initial and current symptoms (see "Common Complaints" and "Other Signs and Symptoms"). Include description, location, severity, and onset of symptoms. It is important to note what has happened since the actual injury. It is not uncommon for clients to report symptoms that reemerge or worsen with exertion.

D. Obtain the client's medical history, especially of previous head injuries. Learning disabilities (e.g., attention deficit hyperactivity disorder [ADHD]), developmental disorders, depression, anxiety, sleep disorders, and mood disorders should also be noted because these can affect recovery.

E. Review current medications. Evaluate any recent change in the dosage and polypharmacy issues (number of prescriptions [>4 medications]). Certain medications like warfarin (Coumadin) can be a predisposing factor that can lead to complications.

F. Document any drug and alcohol history.

G. Ask significant others if they have noticed any additional signs or symptoms, behavioral changes, or evidence of seizure activity.

PHYSICAL EXAMINATION

A. Check pulse, respirations, and blood pressure (BP).

B. Inspect:

1. Observe overall appearance. Note level of awareness.
2. Inspect the skin and head for obvious injury. Periorbital ecchymosis ("raccoon's eyes"), postauricular/ mastoid ecchymosis (Battle sign), or evidence of a cerebrospinal (CSF) leak (otorrhea, rhinorrhea) suggests a basilar skull fracture.
3. Examine the eyes for presence of papilledema (indicates increased ICP), proptosis, and periorbital edema.
4. Examine the ears (hemotympanum or possible laceration to the external canal), nose, and throat.
5. Examine for facial fractures.
6. Examine for any trauma (e.g., malalignment, abnormal curvature) to the cervical spine.

C. Auscultate:

1. Auscultate over the globes of the eyes if warranted (bruit may indicate traumatic carotid-cavernous fistula).
2. Auscultate carotid arteries bilaterally if warranted (bruit may indicate carotid dissection).
3. Auscultate the heart and lungs if cardiovascular etiology is suspected.
4. Auscultate the abdomen if other injuries have occurred from incidental injury such as a contact sport or motor vehicle accident (MVA).

D. Palpate:

1. Palpate for instability of the facial bones, including the zygomatic arch: can have a palpable step-off with orbital rim fractures.
2. If appropriate, palpate the abdomen and the entire posterior spine to rule out any other incidental injury.

E. Neurologic examination:

1. Assess mental status and memory. Determine whether the client is awake, alert, cooperative, and oriented (to person, place, time, and situation). Temporary impairment of memory is one of the most common deficits after a head injury.
2. Assess cranial nerve (CN) function:
   a. Olfactory (CN I).
   b. Ophthalmoscopic/visual exam (CN II).
   c. Pupillary response (CN III).
   d. Extraocular movements (CNs III, IV, VI).
   e. Facial sensation and muscles of mastication (CN V).
   f. Facial expression and taste (CN VII).
3. Perform a motor examination on all four extremities.
4. Perform a sensory exam on all four extremities.

DIAGNOSTIC TESTS

A. A plain film of the skull is usually not obtained for a minor traumatic injury. If the client has suspected skull fracture or clinical indications for imaging, a CT scan is preferred. This type of imaging will usually reveal any linear or basilar skull fractures.

B. CT scan is indicated in clients with the following:

1. Decreasing consciousness during or after the injury.
2. Focal neurologic deficits.

▶

3. Potential, penetrating, or depressed skull fractures.
4. Increasing or persistent severe headache with nausea or vomiting.
5. Seizures postinjury.
6. Alcohol or substance intoxication.
7. Amnesia status postinjury.
8. Unreliable or questionable accuracy regarding the history of the injury.

C. Consider anteroposterior (AP) and lateral spine films for suspected soft tissue injury or vertebral fracture, especially if the client has experienced an amnestic episode or cannot recall the accident.
D. Drug screen.
E. Blood alcohol level.

DIFFERENTIAL DIAGNOSES

A. Concussion (mTBI).
B. Contusion.
C. ICH.
D. Shearing injury.
E. Skull fracture.
F. Subarachnoid hemorrhage (SAH), traumatic.
G. Subdural hematoma (SDH).
H. Epidural hematoma (EDH).
I. Vascular occlusion or dissection.

PLAN

A. General interventions:
1. Admit to the hospital for decreased LOC, seizure activity, focal deficits, penetrating or depressed skull fracture, vomiting, serious facial injuries, and positive head CT findings.
2. Hospitalization may be required if the client has an injured middle meningeal artery or if venous sinus or fractures posteriorly in the skull are suspected. Posterior fossa hematomas may present suddenly (will see a wide pulse pressure).
3. Consider possible secondary injuries including cerebral edema, cerebral infarction, cerebral hemorrhage, hydrocephalus, and infection.
4. The client should not be impaired from alcohol or other drugs when leaving the clinic, which can impact and potentially mask neurologic function or emerging deficits.
5. Hospital admission should be considered for clients without home observation/supervision.
6. Discuss suspected abuse with the client in a private setting.

B. Client teaching: *See Client Teaching Guide for this chapter, "Mild Head Injury."*
1. Provide instruction regarding safety and accident prevention. *See Client Teaching Guide for Chapter 3,"Safety Issues: Fall Prevention."* Instructions include the following:
a. Use safety helmets.
b. Use safety belts.
c. Never drive or operate machinery under the influence.
d. Remove scatter rugs and other objects that would increase fall risk.
e. Use nonslip mats in the shower/bathtub.
f. Install grab bars in the shower/bathtub.
g. Always keep stairs, floors, and hallways clean and clear from clutter.
h. Install handrails in stairways.
i. Wear adequate, correct protective gear related to athletic games and work.
j. Use helmets for biking and other sports.
2. Recommend regular physical activity on most days of the week for elderly clients. Regular exercise will increase upper and lower body strength and balance. Improved strength and balance will help to prevent unintentional falls.

C. Pharmaceutical therapy:
1. For pain relief, the most effective drugs include nonsteroidal anti-inflammatory drugs (NSAIDs) and antidepressants such as tricyclic antidepressants (TCAs).
2. Anxiety and depression occur in varying levels of intensity following mTBI. Anxiety may respond to serotonin enhancers. Depression may respond to selective serotonin reuptake inhibitor (SSRIs).
3. Chronic fatigue with related decrease in attention, concentration, and memory may respond to a moderate use of caffeine or short-term trials of methylphenidate.
4. Poor emotional control or lability may response to SSRIs or anticonvulsants such as carbamazepine or valproate.

FOLLOW-UP

A. Clients with injuries mild enough to be discharged may be observed. Clients with normal examinations in the outpatient setting generally do not require routine follow-up.
B. Assessment of driving ability of the elderly should be done after an mTBI from an MVA.
C. Postconcussion symptoms may continue for some period of time. The use of the Rivermead Post-Concussion Symptoms Questionnaire may be helpful for serial evaluation. The Rivermead tool is available at www.tbi -impact.org/cde/mod_templates/12_F_06_Rivermead.pdf.
1. The most common cause of dizziness following mTBI is related to posttrauma vestibular system dysfunction (benign positional vertigo).
2. Cervical vertigo is related to musculoskeletal neck injury from whiplash. Symptoms often respond to multiple therapeutic interventions provided by a physical therapist.
3. Vision disturbances are frequently unrecognized after mTBI, even with persistent complaints for the person; proper assessment is often not pursued by clinicians.

EMERGENT ISSUES/INSTRUCTIONS

A. Refer all clients to the ED for the following:
1. Focal neurologic deficit(s).
2. Decreasing LOC.
3. Persistent headaches, nausea, and vomiting.
4. Seizures, any other evidence of skull fractures.
5. Neuropsychological dysfunction.

CONSULTATION/REFERRAL

A. Refer to a neurologist to evaluate postconcussive syndrome for continued complaints (e.g., irritability, fatigue, headaches, difficulty concentrating, dizziness, and memory problems). Further evaluation including MRI and EEG testing may be needed.
B. Refer to a psychologist trained in neuropsychological testing (indicated for clients with mild head injury). The assessment tools evaluate brain function in the areas of attention/concentration, initiation/planning, motor/sensory skills, visual perception, learning and memory, language, speed of processing information/reaction time, and complex problem-solving. ▶

C. Other consultations include the following:
1. Complete evaluation by a physical therapist.
2. Need for an occupational therapy evaluation.
3. Speech language pathologist (SLP).

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. The second highest risk factor for adults older than 65 years of age is MVAs due to slower vision problems, slower reflexes, alcohol, and medication.
2. Medications such as aspirin and anticoagulant therapies increase the complication of a head injury.
3. Limited studies exist on the elderly and mTBI. However, findings suggest that elderly clients who experience an mTBI have a higher mortality rate and increased frequency of intracerebral hematomas, and show an increased risk of cognitive deficits after an mTBI.
4. Mortality is greatest among the elderly following an mTBI. mTBI increases their risk of premature death.
5. Studies showed that 2-week post-mTBI elderly clients had posttraumatic complaints/episodes (81%) and displayed emotional distress (30%). Frailty, rather than age, was a significant predictor of earlier complaints and worsening long-term outcomes. These findings could assist providers in recognizing geriatrics at risk for poor recovery and strategize an interprofessional collaborative care plan of psychosocial services and nutritional and physical/occupational therapies, which would promote the possibility of an efficient rehabilitation outcome.
6. For older adults, NPs should practice special consideration of the following geriatric syndromes:
   a. Falls: The number one risk factor in older adults for a head injury is unintentional falls. This is three times greater than in other age groups.
   b. Cognition: Decreased attention/concentration results from disturbance in the speed of neural processing:
      i. Cognitive deficits include attention/concentration, perception, memory, speech/language, or executive functions. The person with frontal lobe damage will have difficulties with decision-making, planning, and initiating and completing tasks. Conversation may be rambling or tangential.
      ii. Reading and auditory comprehension problems may cause the person to decrease or avoid reading and/or listening to conversations. Oculomotor or vision disturbances, including an impaired ability to visually scan reading material or follow a moving object, should be ruled out as causes:
   c. Depression: There is a tendency to isolate and significantly limit or avoid activities. Suicidal ideations and attempts may occur especially if mTBI is underdiagnosed, misdiagnosed, or ignored.
   d. Pain: Pain must be carefully evaluated for its underlying cause before medications for relief are carefully prescribed. Appropriate physical therapy treatment should be used in conjunction with medications.
7. Beers Criteria precaution: Medications for headaches, musculoskeletal pain, or depression/anxiety must be carefully evaluated and prescribed in the elderly to avoid the sedating properties, which can have an impact on a person's attention, cognition, and motor performance.

RESOURCES

Brain Injury Resource Foundation, P.O. Box 84151, Seattle, WA98124-5451, 206-621-8558, www.headinjury.org

The National Collegiate Athletic Association (NCAA) has concussion fact sheets for student athletes and for coaches: http://fs.ncaa.org/Docs/health_safety/ConFactSheetcoaches.pdf

TBI Resource Center: www.braininjuryresources.org

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MULTIPLE SCLEROSIS

DEFINITION

A. Multiple sclerosis (MS) is an autoimmune degenerative disease that damages neuronal axons and breaks down myelin sheaths. The process of inflammatory demyelination varies in progression with recurrent relapse and remission of symptoms over time. The most common symptoms include visual disturbances, spastic paraparesis, and bladder dysfunction. The course of MS is typically intermittent with periodic exacerbations occurring in various areas of the central nervous system (CNS).

B. MS can also present more acutely in severity, progression, and variety of symptoms. Early diagnosis is difficult, but crucial for effective treatment. An MS attack or *exacerbation* lasts at least 24 hours, occurring without fever or any infectious process. Complete recovery after the first exacerbation is common, presenting as a clinically isolated syndrome, but typically converting to MS within 5 years. Subsequent progression of the disease with recurrent exacerbations diminishes physical function over time.

C. The loss of myelin leads to neurologic deficits in vision, speech, gait, writing, and memory, and can affect the swallow or cough reflex. Individuals typically present to the ED upon relapse, with 80% experiencing exacerbations of previous deficits. Clinical diagnosis is supported when at least one reported exacerbation correlates with MS-related findings obtained during the neurologic exam, with MRI, or with visual evoked potential (VEP) studies if visual disturbances are reported.

D. MS is classified as relapsing–remitting (RR) or progressive, and further described as *active* versus *not active, with* or *without progression*. Progressive MS is further differentiated with the following three subtypes:
1. *RR* affects approximately 85% of MS clients; exacerbations of symptoms and periodic remission occur.
2. *Primary progressive (PP)* is a less common form of MS, accounting for around 10% of MS cases; primary progressive multiple sclerosis (PPMS) progresses slowly without periods of relapse or remission.
3. *Secondary progressive (SP)*: It is not unusual for clients with relapsing–remitting multiple sclerosis (RRMS) to progress to secondary progressive multiple sclerosis (SPMS) over time. Progression of the disease process continues with or without periods of remission. Symptoms do not necessarily decrease or stabilize in terms of severity.

INCIDENCE

A. Currently, the Multiple Sclerosis Foundation estimates 400,000 individuals in the United States have been diagnosed with MS and that there are a total of 2.5 million cases worldwide (as reported by other international organizations). It is one of the leading causes of disability in young adults. These statistics are challenged when U.S. physicians are not required to report new cases of MS and when diagnosis is questionably accurate. The signs and symptoms of MS may not be recognized or reported initially, nor when persisting over time; they may also be defined as other disease processes.

B. MS is typically diagnosed in adults between 20 and 50 years of age. Between 4% and 9% of individuals with MS experience onset of symptoms after age 50. However, "late-onset multiple sclerosis (LOMS)" has been diagnosed in adults older than age 55. The occurrence of MS is even less prevalent in those older than age 60. This may be associated with improved healthcare and longevity.

PATHOGENESIS

A. The cause of MS is unknown. A suspected combination of genetic predisposition and a trigger (chronic viral infections, environmental factors, metabolic issues) may create an autoimmune disorder that facilitates the degenerative disease process.

B. Autoimmune attacks on the myelin sheaths of neurons initiate an inflammatory response, followed by eventual plaque formation and scarring, the hallmark characteristic of the disease.

C. A loss of saltatory conduction is noted. Axonal death occurs during this acute inflammatory response, explaining any permanent disability. The associated inflammation and edema around an MS lesion, along with myelin and axonal loss, contribute to the associated neurologic deficit. A limited amount of remyelination and the eventual resolution of inflammation will allow a certain degree of recovery with remission. With each exacerbation, there is a lesser degree of recovery and subsequent decrease in function.

PREDISPOSING FACTORS

A. Family history of MS.

B. Female sex (two times more likely than males to develop MS).

C. Age 20 to 50 (onset can vary from age 10 to 70).

D. Caucasian race (Northern European ancestry).

E. Environment (living in temperate zones; e.g., Canada, northern areas of the United States, Europe). This is associated with prevalence, but no direct link has been established at this time; it is suspected that this distance from the equator causes a vitamin D deficiency secondary to a lack of direct sunlight, contributing to the development of MS.

F. Previous chronic viral infection *may* increase susceptibility.

G. Smoking.

COMMON COMPLAINTS

A. Sensory loss (paresthesia) is often reported early in the course of the disease.

B. Visual disturbances: diplopia upon lateral gaze (occurs in 33% of individuals with MS), blurred vision, loss of vision, and ocular pain.

C. Urinary incontinence, frequency, hesitancy, or urgency (>90% of individuals with MS report bladder dysfunction).

D. Fatigue (in up to 90% of individuals with MS).

E. Weakness in one or more extremities.

F. Gait imbalance (within 15 years of onset, 50% will require assistance during ambulation).

OTHER SIGNS AND SYMPTOMS

A. Babinski response.

B. Spasticity (usually in lower extremities).

C. Depression (nearly 50% of cases; also reported effects on memory, attention, and concentration).

D. Hyperreflexia.

E. Loss of proprioception.

F. Impotence (males).

G. Impaired cognition: subjective difficulties with attention, concentration, short-term memory, planning, and judgment; dementia reported in 3% of individuals with late-stage MS.

H. Dysarthria.

I. Reduced libido (in both males and females).

J. Constipation.

K. Pain.

L. Trigeminal neuralgia (TN; rare).

M. Dysphagia (may also have recurrent respiratory infections secondary to aspiration).

N. Vertigo (in 30%–50% of individuals with MS).

SUBJECTIVE DATA

A. Establish location and onset of symptoms. Is this the first time the client has experienced the symptom in question? Was the onset acute or insidious?

B. Ask the client to describe the quality and severity of the symptom and how it has evolved over time, including duration. Is there a relapse–remitting pattern? Is the symptom progressing in severity?

C. Ask the client if any associated factors aggravate or alleviate the symptom. Does hot weather (as well as hot tubs, saunas, and overexertion) aggravate the condition? Does rest help? Did the symptom resolve spontaneously?

D. Establish if any viral or bacterial infections occurred prior to onset of symptoms.

E. Evaluate visual complaints, including the presence of scotoma, decreased color perception, diplopia, decreased acuity, or painful extraocular eye movements. Is visual deterioration induced by exercise, a hot meal, or a hot bath (the Uhthoff phenomenon)?

PHYSICAL EXAMINATION

A. Check blood pressure (BP), pulse, and respirations.

B. Inspect:

1. Perform a complete eye exam (use Snellen eye chart; test cranial nerve [CN] II, III, IV, and VI):

a. 50% of individuals with MS present with retrobulbar involvement but have no abnormal findings upon funduscopic exam.

b. Anterior involvement causes papillitis; look for presence of macular star.

c. Assess pupillary response bilaterally; look for pendular nystagmus or sinusoidal involuntary oscillations in one or both eyes, and/or loss of smooth eye pursuit.

C. Neurologic and a musculoskeletal exam:

1. Sensory: Test for perception of sharp versus dull stimulus, heat versus cold stimulus, ability to localize pain, proprioception, and vibratory sense, utilizing the tuning fork to evaluate.

2. Motor strength (test all extremities); also observe for increased tone (spasticity), clonus, and tremors. ▶

3. Romberg test and heel-to-toe tandem gait testing (evaluate for ataxia, cerebellar involvement).
4. Finger-to-nose testing and heel-to-shin testing (rule out dystaxia).
5. Check deep tendon reflexes (DTRs) to assess for hyperreflexia; Babinski response may also be present in individuals with MS.
6. Assess mental status (orientation to person, place, time, and situation); test short-term memory and the ability to plan—impaired planning is a cognitive deficit associated with MS.
7. Assess for pain: Include location, descriptive characteristics, onset, duration, timing and setting, aggravating factors, alleviating factors, and any other associated information.
8. Assess for depression, especially with progression of symptoms.

DIAGNOSTIC TESTS

A. There is no specific confirmatory test for MS.

B. MRI is the test of choice supporting clinical diagnosis of MS. An MRI of the brain will reveal the associated plaques when present, but it cannot determine whether these particular lesions are specific to MS (similar radiographic findings are noted with other diseases):

1. Transverse myelitis lesions identified with MRI may convert to MS over time.

C. Cerebrospinal fluid (CSF) analysis: Characteristics of MS include the presence of oligoclonal bands (85%–90% of MS cases), increased immunoglobulin G (IgG; >12%), and increased white blood cells (WBCs; >5%).

D. Evoked response (ER) or evoked potential (EP): Several different tests evaluate brain function and nerve conduction velocity (NCV):

1. Brain auditory evoked response (BAER) detecting subtle changes in brainstem function with hearing.
2. Visual interpretation and perception (VEP), evoked visual response to flash and pattern reversal (VER).
3. Somatosensory evoked potential (SSEP) studies evaluate nerve conduction from the extremities via the spinal cord to the brain.

E. Complete blood count (CBC) with differential.

F. Serum glucose to rule out hypoglycemia and chronic hyperglycemia as potential causes of neurologic findings.

DIFFERENTIAL DIAGNOSES

A. MS.
B. CNS lymphoma.
C. CNS infection.
D. Acute disseminated encephalomyelitis (ADEM).
E. Tumor: brainstem, cerebellar, or spinal cord.
F. Amyotrophic lateral sclerosis (ALS).
G. Systemic lupus erythematosus (SLE).
H. Syringomyelia.
I. Progressive multifocal leukoencephalopathy (PML).
J. Sarcoidosis.
K. Sjogren's syndrome.
L. Acute transverse myelitis.
M. Myasthenia gravis (MG).
N. Guillain–Barré syndrome (GBS).
O. Cerebrovascular accident (CVA).
P. Diabetes mellitus (DM).
Q. Spinal cord compression (stenosis, ruptured disc).
R. Behcet disease.
S. Neuromyelitis optica (NMO).

PLAN

A. General interventions.

1. Discuss the purpose and availability of local MS support groups.
2. Discuss counseling services for development of adaptive coping techniques, improved family dynamics and relationships with significant others, adjustment to physical disability, and any anticipatory grief issues.
3. Educate on all medications, including side effects and associated laboratory tests with follow-up.
4. Suggest strategies regarding short-term memory and planning ability: Individuals may choose to write things down, develop schedules and calendars, make lists, draw pictures, and take more time when devising plans.

B. Client teaching:

1. Educate on heat sensitivity and how it can aggravate symptoms: Avoid hot tubs, saunas, prolonged exposure in hot/humid weather; choose appropriate clothing for the season. Around 60% of individuals with MS experience heat sensitivity, facilitating a *pseudoexacerbation* where MS symptoms may worsen, but do not necessarily indicate additional axon or myelin degeneration.
2. For visual disturbances, offer advice on resting the eyes at various times during the day; for diplopia, an eye patch can be used temporarily.
3. Stress the importance of exercise and its effect on MS-related fatigue and spasticity; overactivity/overexertion is another issue to address at this time. Rest periods are needed during exacerbations.
4. Provide education on Kegel exercises and timed voiding to improve bladder function; also advise to avoid alcohol and caffeinated beverages.
5. Promote increased water intake, dietary fiber intake, and physical activity for increased bowel motility.
6. Educate regarding the signs and symptoms of infection, especially urinary tract infections (UTIs), and how infection can trigger an exacerbation.
7. Instruct safe performance of self-intermittent catheterization (SIC) for urinary retention and emphasize the importance of sterile technique.

C. Pharmaceutical therapy: Medications are prescribed specifically for the type of MS and may include a combination of the following:

1. Disease-modifying drugs suppress the immune system to slow the progression of MS (lessen frequency and severity of exacerbations, reduce MS plaques):
   a. Injectable medications (recommended for first-line therapy):
      i. Interferon beta-1a: Avonex, Betaseron, Extavia, Rebif.
      ii. Glatiramer acetate: Copaxone (40 mg), Glatopa (a generic equivalent of Copaxone, 20 mg).
      iii. Peginterferon beta-1a: Plegridy.
   b. Oral medications:
      i. Teriflunomide (Aubagio): recommended for first-line therapy.
      ii. Fingolimod (Gilenya): recommended for second-line therapy.
      iii. Dimethyl fumarate (Tecfidera): recommended for first-line therapy. ▶

c. Intravenous (IV) infusions:

Natalizumab (NTZ; Tysabri) therapy includes a risk-minimization action plan utilized by neurologists who specialize in MS treatment under the TOUCH™ Prescribing Program. It is administered at infusion centers registered and authorized under TOUCH, a distribution program designed to assess the risk of PML associated with Tysabri administration, minimize this risk, reduce PML-associated deaths and disability, and promote informed decision-making regarding the risks and benefits of Tysabri use. Additional information is available on the Food and Drug Administaration (FDA) website at www.fda.gov/downloads/drugs/drug safety/postmarketdrugsafetyinformationforpatientsandproviders/ucm107197.pdf.

i. Alemtuzumab (Lemtrada): recommended for second-line therapy.
ii. Mitoxantrone (Novantrone): recommended for second-line therapy.
iii. Rituximab (Rituxan): Off-label use for RRMS when second-line medications have been ineffective.
iv. Natalizumab (Tysabri): recommended for second-line therapy.

2. Short-term steroid use (3- to 5-day course of methylprednisolone or dexamethasone) may shorten an exacerbation. Long-term use is not recommended. IV steroids may be prescribed initially, then transitioned to oral steroids (prednisone, dexamethasone) that are tapered off over time:
a. H.P. Acthar Gel (adrenocorticotropic hormone [ACTH]) is an option when individuals cannot tolerate the side effects of high-dose corticosteroids, do not respond to corticosteroid therapy, have no available IV infusion services, or when individuals have difficult/inadequate peripheral IV access.

3. Dalfampridine is an FDA-approved drug that has been shown to improve ambulation in the MS population. It is a potassium channel blocker that targets channels located on the outside of nerve fibers, possibly improving nerve conduction when the myelin sheaths are damaged.

4. Meclizine (Antivert) may be prescribed for dizziness or vertigo.

5. Various medications (antiepileptics, antidepressants) may be considered for treatment of neuropathic or spasticity-related pain:
a. Phenytoin (Dilantin).
b. Amitriptyline (Elavil).
c. Clonazepam (Klonopin).
d. Gabapentin (Neurontin).
e. Nortriptyline (Pamelor, Aventyl).
f. Carbamazepine (Tegretol).

6. MS-related spasticity may be treated with the following drugs:
a. Baclofen.
b. Zanaflex.
c. Clonazepam, diazepam (watch for sedation, dependence).
d. Dantrium (use only when other drugs are ineffective as it can cause liver damage).
e. Botulinum toxin injections.
f. Intrathecal (IT) pump placement is another option for continuous IT administration of baclofen or clonidine for spasticity.

7. Stool softeners (Colace, Peri-Colace), bulk-forming agents (Metamucil), and/or laxatives (milk of magnesia, MiraLAX) may be prescribed for complaints of constipation, depending on the severity of symptoms.

8. Flomax or Hytrin can be used to improve urinary flow.

9. Ditropan, Tofranil, and Detrol are several examples of medications commonly used to treat bladder spasms.

10. Options for treatment of depression include the following:
a. Selective serotonin-norepinephrine reuptake inhibitors (SSNRIs): duloxetine hydrochloride (Cymbalta), venlafaxine (Effexor).
b. Selective serotonin reuptake inhibitor (SSRIs): fluoxetine (Prozac), sertraline (Zoloft), paroxetine (Paxil).
i. Individuals with protracted, painful, and/or progressive medical conditions are at risk for suicide.

11. Provigil, Symmetrel, or Prozac can be used to treat fatigue symptoms.

12. Stem cell transplantation for RRMS is emerging in the literature. Clinical trials present encouraging results that indicate reversal of symptoms while offering a way to "reset" the immune system. Stem cell transplantation shows promise as a future treatment option for RRMS.

FOLLOW-UP

A. A neurologist who specializes in MS management should coordinate and prescribe all necessary treatment and therapy for this population. Nurse practitioner comanagement with a primary care physician for follow-up care depends on the clinical presentation, type of diagnosis, and treatment involved.

CONSULTATION AND REFERRALS

A. Neurology referral and consultation for management of MS.

B. Ophthalmology for visual disturbances and optic neuritis.

C. Urology for genitourinary (GU) disturbances (impotence in males; urinary hesitancy, frequency, incontinence, and urgency; UTIs).

D. Occupational therapy can address any issues with fine motor skills (coordination, strength) and performing activities of daily living (ADLs), and can prescribe the necessary adaptive equipment.

E. Speech therapy can address any issues with language, cognition, or swallowing (including evaluations for feeding tube placement and diet recommendations).

F. Physical therapy can address gait imbalance, motor weakness, spasticity, and range of motion (ROM), as well as prescribe assistive devices and equipment for impaired physical mobility.

G. Psychiatry referral and consultation for pharmacologic management of depression and dementia (if indicated); also consider psychology or licensed professional counselor referrals for counseling.

H. Social work referral for assistance with insurance issues, locating community resources, applying for disability, arranging home healthcare, obtaining placement in a skilled nursing facility (SNF), and providing counseling for the individual and the family.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Symptoms of MS may stabilize or remit during pregnancy, but 20% to 40% of clients have a relapse within 3 months after delivery.
2. No evidence suggests that pregnancy affects the long-term course of MS. There is no acceleration in the rate of disability or disease progression postpartum. ▶

3. Neither epidural anesthesia nor breastfeeding has an adverse effect on the rate of relapse or progression of disability.
4. There are no accepted guidelines for recommending for or against pregnancy in individuals with MS. MS history and current neurologic deficits should be considered independently per client.
5. Pregnancy may affect the treatment regimen; some of the drugs used to treat MS are known teratogens. Glucocorticoids may cause neonatal adrenal suppression and maternal glucose intolerance:
   a. Intravenous immunoglobulin (IVIg) is an alternative to consider during pregnancy or while breastfeeding, after weighing the risks and benefits carefully before administration. The adverse effects are few, but may present complications that can be severe (e.g., aseptic meningitis, thromboembolism).

B. Geriatrics:
1. The occurrence of MS older than age 60 is not typical. Spinal infarcts are more commonly diagnosed when evaluating specific inflammatory lesions in this population.
2. Clinical diagnosis of LOMS clients can be challenging due to the number of other age-associated diagnoses, atypical presentation of signs and symptoms, and lack of information on MS in older individuals. Hepatic and renal functions decline with age as other comorbidities may increase, which also complicates diagnoses and treatment.

RESOURCES
Multiple Sclerosis Association of America: www.mymsaa.org
Multiple Sclerosis Foundation: www.msfocus.org.
National Multiple Sclerosis Society: www.nationalms society.org

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MYASTHENIA GRAVIS

DEFINITION
A. Myasthenia gravis (MG) is a chronic autoimmune disorder that affects the neuromuscular junction and is characterized by fatigability and weakness of voluntary muscles.

INCIDENCE
A. The prevalence is 0.5 to 11.5 cases per 1 million people. There are two peaks in MG incidence that are age- and sex-related: One is females in their 20s and 30s, and the other is males in their 60s and 70s.

PATHOPHYSIOLOGY
A. MG is believed to be an antibody-mediated autoimmune attack that destroys variable numbers of acetylcholine receptors (AChR) at the postsynaptic junction. The decrease in AChRs results in weakness with repeated activities and recovery after rest. MG is often associated with thymic hyperplasia or tumors; the thymus plays an unclear role in the autoimmune process of MG.

PREDISPOSING FACTORS
A. No predisposing factors have been identified.

COMMON COMPLAINTS
A. Classic triad: ptosis, diplopia, and dysphagia:
1. Fluctuating symptoms such as droopy eyelid(s).
2. Blurry or double vision.
3. Sense of choking.

B. Difficulty chewing.
C. Slurring of speech.
D. Easy fatigability.
E. Symptoms are more pronounced with fatigue or in the evening.

OTHER SIGNS AND SYMPTOMS
A. Selected voluntary muscles that fatigue with activity.
B. Motor function that improves with rest but then decreases with use.
C. Signs of impending MG crisis:
1. Sudden onset of inspiratory distress.
2. Difficulty swallowing.
3. Visual difficulty.
4. Tachycardia.
5. Rapid onset of weakness.

SUBJECTIVE DATA
A. Establish onset of symptoms and possible progression.
B. Ask the client what makes the symptoms better or worse: Does rest help?
C. Ask if the client feels better in the morning or in the afternoon or evening.
D. Look for difficulties with chewing or swallowing.
E. Investigate medications the client is currently taking or has recently taken, such as antibiotics.

PHYSICAL EXAMINATION

The presentation and course of MG are both highly variable, and MG can therefore be very difficult to diagnose.

A. Check pulse, respirations, and blood pressure (BP).
B. Inspect:
1. Observe overall appearance.
2. Perform complete eye exam. Subtleties of eye movement dysfunction are often key in differentiating MG from other disorders.

C. Palpate:
1. Assess deep tendon reflexes (DTRs); note normal to increased reflexes.

D. Neurologic exam:
1. Perform complete neurologic exam.
2. Test the following:
   a. Muscle strength: Weakness is increased with repetition or sustained activity; arm raise cannot be sustained. ▶

b. Eyes:
i. Upward or lateral gaze cannot be maintained for longer than 30 seconds.
ii. Eyes: Ptosis occurs with repetitive lid closure.
iii. Ice pack test: Fill a plastic bag or glove with ice and place over closed eyelid for 2 minutes. Remove ice and evaluate the degree of ptosis. Noted to be very sensitive with prominent ptosis.
c. Voice: Voice quality or speech changes when counting out loud to 100.

Normal coordination, normal sensory perception, and normal pupillary response are noted in MG.

DIAGNOSTIC TESTS

A. Antibody titer for acetylcholine receptor (AChR-Ab) is positive in 90% of clients with MG. May also perform muscle specific kinase (MuSK) antibody titer. Approximately 6% to 12% of clients will have negative antibody titers for both titers.
B. Cholinesterase-inhibiting drug test: improvement in strength following injection of edrophonium (Tensilon).
C. Repetitive muscle stimulation test: decremental response.
D. Single-fiber electromyographic (EMG) and/or repetitive nerve stimulation (RNS) studies are diagnostic studies performed for diagnosis.

Initial diagnostic tests may be equivocal with some negative test results, but this does not absolutely rule out MG.

DIFFERENTIAL DIAGNOSES

A. MG: The most common disorder of neuromuscular transmission. **It is important to maintain a high index of suspicion and include MG in the differential diagnosis of any client presenting with variable muscle weakness even without eye signs.**
B. Incomplete extraocular nerve palsy.
C. Polymyositis.
D. Brainstem transient ischemic attack (TIA).
E. Amyotrophic lateral sclerosis (ALS).
F. Brainstem vascular accident: "Dizziness" is a symptom rarely seen with MG but often associated with brainstem ischemia.
G. Guillain–Barré syndrome (GBS).
H. Brainstem tumor.
I. Hyperthyroidism or hypothyroidism.
J. Cholinergic crisis: Although it is useful to distinguish myasthenic crisis (weakness from MG exacerbation) from cholinergic crisis (weakness from too much medication), both can rapidly lead to respiratory failure. Transportation to an ED for evaluation should not be delayed by attempts to differentiate the two.
K. Eaton–Lambert myasthenic syndrome: Often associated with bronchogenic carcinoma but may precede detection of the carcinoma by as many as 2 years.

PLAN

A. General interventions:
1. MG is primarily managed by a neurologist, given the difficulty in diagnosing it, the variable course of the disease, and the highly individualized medication regimen required.
2. The course of MG fluctuates most during the first 3 to 5 years after diagnosis.
3. Autoimmune disorders, such as thyroid disease, rheumatoid arthritis, and systemic lupus erythematosus (SLE), should also be screened for in clients diagnosed with MG.
B. Client teaching: *See Client Teaching Guide for this chapter, "Myasthenia Gravis."*
1. Clients should have a MedicAlert tag and always carry a list of their medications and dosing schedules in case of an emergency.
C. Medical and surgical management.
1. Thymectomy is an early consideration; an MRI of the chest is obtained once the diagnosis is made to assess for thymic enlargement.

Thymectomy lessens the severity of MG but rarely results in complete elimination of the need for medication.

2. Plasmapheresis, or plasma exchange to remove antibodies, is used emergently for management of myasthenic crisis. Opinion is mixed regarding its use in the long-term management of myasthenia.
3. A myasthenic crisis occurs when the muscles that control breathing are weakened to the point of requiring ventilation. This usually is triggered by an infection, fever, or adverse reaction to a medication.
D. Pharmaceutical therapy:
1. Many medications can worsen myasthenic symptoms, so changes and additions of any medication require consultation with the client's neurologist. Cholinesterase-inhibiting medications:
a. Pyridostigmine (Mestinon) 60 mg and 180 mg sustained-release (SR), titrate as needed, with usual dose up to 600 mg/d.
b. Neostigmine methylsulfate (Prostigmin) 0.25 to 0.5 and 1.0 mg/mL concentrations, titrated with starting dose 0.5 mg subcutaneously or intramuscularly (IM) every 3 to 4 hours.
2. Steroids may be used when inpatient and then tapered on an outpatient basis, tapering every 3 days.
3. Effectiveness of medication regimen is gauged by changes in ptosis, diplopia, dysphagia, chewing ability, and muscle fatigue.
4. Care should be used with use of medications that may worsen symptoms of weakness.

FOLLOW-UP

A. Individuals with MG require lifelong management by a neurologist, given the variable course of both the disease and the client's response to treatment.
B. The client is initially followed every 1 to 2 months, then every 3 to 4 months.

CONSULTATION/REFERRAL

A. If MG is suspected, consult with a physician and consider neurologic referral.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. MG is considered high risk for both the pregnant person and the fetus.
a. Pregnancy requires management by the client's neurologist and a perinatologist.
2. MG frequently manifests for the first time during pregnancy. Refer to a perinatologist.

B. Adults:
1. Oral contraceptives may worsen myasthenic symptoms.

RESOURCE
Myasthenia Gravis Foundation of America: https://myasthenia.org

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NEUROCOGNITIVE DISORDERS (DEMENTIA)

DEFINITION
A. Neurocognitive disorders are characterized by deficits in cognitive function, with a significant decline from a previous level of function. Decline may be evident in one or more areas of function, including attention, language, memory, visuospatial skills, or executive function (complex tasks such as organizing, sequencing, judgment, and reasoning).
B. Neurocognitive disorders include Alzheimer disease (AD), vascular dementia, Lewy body dementia, and other dementias (frontotemporal dementia, Parkinson dementia, HIV dementia, neurosyphilis, and Korsakoff dementia).
C. Clinical features that differentiate these disorders:
1. AD:
a. Gradual onset and a course of progressive decline.
b. Memory, language, and visuospatial deficits.
c. Depressive symptoms, which may precede diagnosis.
d. Delusions, hallucinations, agitation, and apathy.
2. Vascular dementia:
a. Abrupt onset and a stepwise course of progression.
b. Aphasia.
3. Lewy body dementia:
a. Visual hallucinations and delusions.
b. Extrapyramidal symptoms (muscle rigidity, parkinsonism).
c. Fluctuating mental status.
d. Increased sensitivity to antipsychotic medications.
4. Frontotemporal dementia:
a. Change in personality.
b. Hyperorality.
c. Impairment in executive function, with relatively well-retained visuospatial skills.
d. Loss of social awareness.
D. The diagnosis of dementia must be differentiated from delirium, a disturbance in cognition that develops over a short period of time and is characterized by an alteration in attention that fluctuates in severity during the course of the day. Delirium may be the consequence of an acute medical condition, hospitalization, or medication-/substance-induced. Delirium typically may last weeks to months, with gradual improvement in cognition.

INCIDENCE
A. Dementia is present in 1% to 2% of persons aged 65 years, and up to 30% of those 85 years and older have a neurocognitive disorder. AD is the most commonly occurring dementia (60%–70%), with the other cases due to mixed causes (30%), such as multi-infarct or vascular dementia. Alzheimer accounts for more than 50% of nursing home admissions and is the condition most feared by aging adults. The Alzheimer's Association reports more than 5 million Americans have AD. One in three senior adults will die with a diagnosis of AD or another form of dementia. The incidence is higher in females, with almost two-thirds of persons with AD being female.

PATHOGENESIS
A. The following includes the pathogenesis of each type of dementia:
1. AD:
a. Cortical atrophy is most prominent in the temporal and hippocampal regions of the brain.
b. There is extracellular deposition of beta-amyloid protein.
c. Intracellular neurofibrillary tangles contribute to neuronal loss; tau proteins are a major component.
d. Decreased levels of acetylcholine.
e. The Apo E epsilon 4 allele of chromosome 19 has been associated with familial and late-onset AD.
2. Vascular dementia:
a. Microvascular changes of the brain.
b. Cortical and subcortical infarctions.
3. Lewy body dementia:
a. Extensive cortical neuritic plaques.
b. Presence of Lewy bodies (intraplasmic spherical neuronal inclusion bodies) in the brainstem, cortex, and neocortex.
4. Frontotemporal dementia:
a. Atrophy of the frontal and temporal regions of the brain.
5. A number of diseases alter cerebral metabolism resulting in dementia, such as Huntington chorea and Parkinson disease (PD). A variety of diseases that can produce or mimic dementia may be arrested or reversed. These are classified as pseudodementia, such as hypothyroidism or depression.

PREDISPOSING FACTORS
A. Definite risks:
1. Advanced age.
2. Atrial fibrillation (AFib).
3. Depression.
4. Family history.
5. Down syndrome.
B. Possible risks:
1. Delirium.
2. Head trauma.
3. Heavy smoking.

COMMON COMPLAINTS
A. Interview the client, family members, and/or friends/caretakers who spend quality time with the client to assess for social, neurologic, and cognitive changes experienced by the client.
1. Memory impairment.
2. Change in behavior and inability to perform normal activities of self-care.

OTHER SIGNS AND SYMPTOMS
A. Disoriented to date and/or place.
B. Naming difficulties (anomia). ▶

C. Impaired recent recall.
D. Decreased insight.
E. Impaired judgment.
F. Social withdrawal.
G. Problems managing finances; inability to pay bills and to balance checkbook; spending money in unusual ways.
H. Getting lost in familiar environments.
I. Lack of safety awareness; leaving the stove on, taking medications incorrectly, increased vulnerability to strangers.

SUBJECTIVE DATA

A. Elicit onset and duration of symptoms; commonly, this information comes from family members.
B. Question the family members and/or caregivers regarding personality changes in the client or any changes in personal hygiene.
C. Review the client's history, including history for sexually transmitted infections.
D. Review medications, specifically those medications with anticholinergic side effects, including over-the-counter (OTC) products such as diphenhydramine.
E. Is there a loss of interest in things the client used to find important?
F. Evaluate the client's history for any recent major life events such as death of a spouse, a move to a new living environment, or loss of purpose following retirement.

EXAMINATION

A. Evaluate blood pressure (BP), pulse, respirations, and weight.
B. Inspect:
1. Observe general appearance; note grooming, interest in conversation, and apathy.
2. Note the presence of slurred speech and slowed body movements.
3. Inspect the nail beds and mucous membranes for anemia.
C. Auscultate:
1. Auscultate heart, lungs, and abdomen.
D. Palpate:
1. Palpate the thyroid.
E. Neurologic exam:
1. Perform a complete neurologic exam, including cranial nerves (CNs), gait, motor function, and cerebellar function.
2. Note facial asymmetry, distal weakness, and any focal neurologic findings.
3. Complete mental status exam with instrument of choice: See section "Diagnostic Tests."
4. Assess functional status. May use a functional assessment tool such as the Physical Self-Maintenance Scale, Instrumental Activities of Daily Living (IADL) Scale, or Reisberg Functional Assessment Staging (FAST) Scale. Available at www.pbm.va.gov/PBM/clinicalguidance/drugmonitoring/FunctionalAssessmentStagingFAST73108.doc.
5. Complete depression screening with instrument of choice:
a. Geriatric Depression Scale. Available at www.consultgeri.org/try-this/general-assessment/issue-4.pdf.
b. Patient Health Questionnaire (PHQ-9). Available at www.phqscreeners.com.
c. Beck Depression Scale. Available at www.bmc.org/Documents/Beck-Depression-Inventory-BDI.pdf.

DIAGNOSTIC TESTS

A. Mini-Mental State Examination (MMSE). Available at www.uml.edu/docs/Mini%20Mental%20State%20Exam_tcm18-169319.pdf.
B. Pfeiffer's Short, Portable, Mental Status Questionnaire or other mental examination of choice. Pfeiffer's mental status questionnaire is available at geriatrics.stanford.edu/culturemed/overview/assessment/assessment _toolkit/spmsq.html.
C. The clock-draw test (CDT) may also be administered and used as a screening tool. The CDT) is available at www.ohsu.edu/xd/outreach/oregon-rural-health/about/rural-health-conference/upload/clock_drawing _test-mini-cog.pdf (see Section II: Procedures, "Clock-Draw Test").
D. Rule out possible reversible causes of dementia; not all are required, so use discretion:
1. Thyroid function tests, to rule out either hypothyroidism or hyperthyroidism.
2. Complete blood count (CBC).
3. Vitamin B_{12} level: anemia or B_{12} deficiency.
4. Serum chemistry profile: hyponatremia.
5. Toxicology screen or serum drug screen: toxicity or intoxication.
6. Venereal Disease Research Laboratory (VDRL), fluorescent treponemal antibody absorption (FTA-ABS), or microhemagglutination assay for antibody to *Treponema pallidum* (MHA-TP; cerebrospinal fluid [CSF]) to confirm syphilis.
7. HIV-1 antibody titer: AIDS dementia complex.
8. Liver function tests: liver disease.
9. CT scan or MRI: vascular dementia, tumor, chronic subdural hematomas (SDHs), normal pressure hydrocephalus (NPH), and AIDS dementia complex.
10. EKG: Creutzfeldt–Jakob disease.
11. Neuropsychological evaluation.

The history is the key to diagnosis of dementia. The physical exam may be normal. Dementia is not a normal part of aging; normal aging intelligence scores decrease by only about 10% by age 80. A thorough search for a potentially reversible cause is required.

DIFFERENTIAL DIAGNOSES

A. Completely reversible dementia, rarely.
B. Depression and adverse reactions to medications are the most common reversible causes of dementia. Use the DEMENTIA pneumonic:
1. D: Drugs or depression.
2. E: Emotional upset.
3. M: Metabolic, for example, vitamin B_{12} deficiency or hypothyroidism.
4. E: Ear or eye impairment or sensory impairment.
5. N: NPH.
6. T: Tumors or masses, for example, subdural hematomas (SDHs).
7. I: Infection or sepsis.
8. A: Anemia.
C. AD.
D. Dementia with Lewy bodies.
E. PD with dementia.
F. Vascular dementia.
G. Frontotemporal dementia.

PLAN

A. General interventions:
1. The goal is to treat identifiable abnormalities. ▶

2. Educate family and client regarding the diagnosis, disease process, and progression.
3. Encourage healthy behaviors including regular exercise, healthy diet, and stress management.
4. Maintain brain function through involvement in stimulating social activities.
5. Consider driving evaluation if the client is still driving.
6. Recommend the use of a safe-return bracelet.
7. Evaluate the home for safety features.
8. Arrange for supportive care for the family and client.
9. Information on support groups for the caregiver and family is very helpful.
10. See section "Alzheimer Disease" for treatment and care for families needing legal and/or financial assistance. Discuss advance directives and planning for future care needs.

B. Client teaching: *See Client Teaching Guide for this chapter, "Dementia."*
1. Educate regarding the diagnosis, disease process, and progression.
2. Discuss advance directives and planning for future care needs.
3. Encourage client and family caregivers to become involved in dementia support groups.
4. Consider driving evaluation, if client is still driving.
5. Recommend the use of a safe-return bracelet.
6. Evaluate the home for safety features.
7. Avoid medications such as anticholinergic medications, including diphenhydramine, hydroxyzine, tricyclic antidepressants (TCAs), and oxybutynin.
8. Recommended handbooks for the family include the following:
a. *The 36-Hour Day: A Family Guide to Caring for People Who Have Alzheimer's Disease, Related Dementias and Memory Loss.*
b. *Guidelines for Dignity* and *Family Guide*.
c. Both books can be ordered through the Alzheimer's Disease and Related Disorders Association or its local chapters. The national headquarters can be contacted at the following address:
Alzheimer's Disease and Related Disorders Association
225 North Michigan Avenue
Floor 17
Chicago, IL 60601
800-272-3900

C. Pharmaceutical therapy:
1. There are not any medications that have been found to decrease the rate of decline in cognitive behavior. However, studies have shown that clients can be treated with medications for agitation, hallucinations, and depression and to improve mental alertness. Studies indicate that clients with a diagnosis of mild to moderate AD, vascular dementia, Lewy body dementia, and Parkinson dementia may trial and benefit from a cholinesterase inhibitor:
a. Donepezil (Aricept): Start 5 mg daily at bedtime for 4 weeks, then 10 mg at bedtime.
b. Rivastigmine (Exelon): Start 1.5 mg BID, then increase by 1.5 mg every 4 weeks to maximum dose of 6 mg BID.
c. Also Exelon patch: Start 4.6 mg topically daily; increase to 9.5 mg topically daily after 4 weeks.
d. Galantamine (Razadyne): Start 4 mg BID for 4 weeks, then 8 mg BID. Maximum dose 12 mg BID.
2. Clients with moderate-to-severe AD:
a. Memantine (Namenda): Namenda XR 7 mg daily, titrate by 7 mg per week to recommended dose of 28 mg daily.

FOLLOW-UP

A. Follow-up visit at 1 month to evaluate client's status, response to medication, and side effects.
B. Clients and families have an ongoing need for education and support in learning to deal with the diagnosis.
C. Subsequent follow-up visits can be scheduled every 3 to 6 months.

CONSULTATION/REFERRAL

A. Refer the client to a neurocognitive specialist if the diagnosis is unclear.
B. Refer clients and families to support groups in the community.
C. Geriatric psychiatry may be appropriate for management and treatment of behavioral and psychological symptoms of dementia.
D. Refer to social services or geriatric case management for assistance with respite care and placement options.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. Irreversible dementia or AD begins in the fourth to fifth decade of life and is characterized by loss of recent memory, inability to learn new information, language problems, mood swings, and personality changes.

RESOURCES

Alzheimer's Association: www.alz.org
Alzheimer's Disease Education and Referral Center: www.nia.nih.gov/alzheimers
Frontotemporal dementia: National Institute of Neurological Disorders and Stroke: www.ninds.nih.gov
Lewy Body Dementia Association: www.lbda.org

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NORMAL PRESSURE HYDROCEPHALUS

A. Idiopathic normal pressure hydrocephalus (NPH) is characterized as a communicating hydrocephalus with ventricular enlargement and a normal opening pressure on lumbar puncture (LP). Idiopathic NPH, a slowly progressive clinical complex, results in a triad of symptoms: gait disorder, urinary incontinence, and dementia.

B. Secondary NPH is caused by an obstruction or noncommunication within the ventricles that prevents the circulation of cerebral spinal fluid and results in hydrocephalus.

INCIDENCE

A. Idiopathic NPH is considered a rare condition when compared with other causes of dementia. Different studies report the incidence from 2 to 20 million per year. Misdiagnosis or delayed diagnosis is common. Idiopathic NPH is more common between the ages of 60 and 70 years. Approximately 50% of NPH have secondary causes. Both genders are affected equally.

PATHOGENESIS

A. Cerebrospinal fluid (CSF) is produced by the choroid plexus and flows from the third and fourth ventricles into the subarachnoid spaces and into the venous circulation. Impaired absorption of CSF results in ventricular enlargement. Several mechanisms of idiopathic NPH have been suggested, including congenital, compensated hydrocephalus present since birth and becoming symptomatic later in life, normal CSF production but decreased CSF absorption, retrograde flow into the internal jugular veins increasing central venous pressure, and vascular risks such as hypertension (HTN) and peripheral vascular disorders. The major causes of secondary NPH are subarachnoid hemorrhage (SAH), head trauma, tumor, and meningitis.

PREDISPOSING FACTORS

A. Most common:
- **1.** Age: most common between ages 60 and 70.
- **2.** Systemic HTN.
- **3.** Peripheral vascular disease.
- **4.** Arteriosclerotic cardiac disease.

B. Probable:
- **1.** Diabetes mellitus.

COMMON COMPLAINTS

A. The three classic features are:
- **1.** Gait: slow, cautious, shuffling, with small steps and a wide base with increased truncal swaying and unsteadiness, unstable turning, and difficulty initiating steps (magnetic gait). **This feature must be present for diagnosis of NPH.**
- **2.** Urinary incontinence: urgency may occur at onset progressing into incontinence.
- **3.** Cognitive impairment: slowly progressive; becomes evident following gait dysfunction. Includes psychomotor slowing, apathy, forgetfulness, decreased attention and bradyphrenia (slowness of thought).

B. Symptoms and symptoms that are prominently absent include the following:
- **1.** Elevated opening pressure on LP.
- **2.** Complaints of headache.
- **3.** Nausea or vomiting.
- **4.** Papilledema.

C. Comorbidities commonly present in older persons that impact on clinical presentation and outcomes:
- **1.** Musculoskeletal conditions such as osteoarthritis of the knees, hips, or spine.
- **2.** Urinary disorders: previous urinary tract problems including frequency, nocturia, infection, and large postvoid residual.
- **3.** Cerebrovascular disorders including Binswanger disease, a form of vascular dementia.
- **4.** Vascular disorders including hyperlipidemia, diabetes, obesity, and physical inactivity.
- **5.** Alzheimer disease (AD).
- **6.** Parkinson disease (PD).

SUBJECTIVE DATA

A. Family members or significant others are a good resource for obtaining an accurate history.

B. Determine the onset and presentation of gait disorder.

C. Note characterization of gait and its similarity and difference from Parkinson disorder.

D. Note onset, course, and characterization of urinary incontinence.

E. Review medical history of comorbid disorders and treatment protocols, recent acute physical illness, head trauma, hemorrhage, or meningitis.

F. Note evidence of difficulty with attention and concentration (digit span and arithmetic progression). General orientation and general memory spared when compared with Alzheimer dementia.

G. Note evidence of forgetfulness, decreased attention span, inertia, and bradyphrenia.

H. Note if the client is able to carry on activities of daily living (ADLs) and instrumental activities of daily living (IADLs), such as using the telephone, shopping, preparing meals, taking medications, and handling finances.

I. Review the client's medication history and compliance, especially those with anticholinergic side effects, herbal remedies, and over-the-counter (OTC) medications.

J. Assess the client for nutritional status and dietary intake.

K. Assess depressive symptoms such as insomnia, anhedonia, and sense of worthlessness and hopelessness.

PHYSICAL EXAMINATION

A. Check blood pressure (BP), pulse, respirations, height, and weight.

B. Inspect:
- **1.** Skin for overall status for hygiene, nutritional status.

C. Palpate:
- **1.** Chest, abdomen, and joints.

D. Auscultate:
- **1.** Lungs, heart, and abdomen.

E. Neurologic exam:
- **1.** Perform complete neuro exam. Note facial asymmetry, gait, and balance.
- **2.** Check deep tendon reflexes (DTRs); assess for hyperreflexia.
- **3.** Assess cranial nerves (CNs) II to XII.
- **4.** Assess muscle strength/tone, testing upper and lower extremities for increased tone (spasticity), tremors, and/or decreased tone.
- **5.** Assess sensory response, testing perception for sharp versus dull stimulus, heat versus cold stimulus, local pain ▶

perception, and proprioception, and evaluate sense of vibration with tuning fork.

6. Note gait, posture, coordination, and balance.

F. Perform Standardized Mini-Mental State Examination (SMMSE) screening test:

1. Mini-Mental State Examination (MMSE). Available at www.uml.edu/docs/Mini%20Mental%20State%20Exam_tcm18-169319.pdf.
2. Montreal Cognitive Assessment (MOCA). Available at www.Mocatest.org.
3. Saint Louis University Mental Status (SLUMS) Examination. Available at medschool.slu.edu/ agingsuccessfully/pdfsurveys/slumsexam_05.pdf.
4. Mini-Cog. Available at www.mini-cog.com/mini-cog-intrument/standardized-mini-cog-instrument.
5. Hachinski Ischemia Score. Available at www.strokecenter.org/wp-content/uploads/2011/08/hachinski.pdf.
6. *Rule out other specific causes of dementia, including cerebrovascular disease.*

G. Complete depression screening:

1. Geriatric Depression Scale (GDS). Available at https://consultgeri.org/try-this/general-assessment/issue–4.
2. Beck Depression Inventory. Available at www.enhertsccg.nhs.uk/sites/default/files/pathways/Beck%27s%20Depression%20Inventory_0.docx.
3. Katz Index of Independence in ADLs. Available at https://www.alz.org/careplanning/downloads/katz -adl.pdf.
4. The Lawton Instrumental Activities of Daily Living (IADL). Available at https://consultgeri.org/try-this/general-assessment/issue-23.pdf.

DIAGNOSTIC TESTS

A. Diagnostic cerebral spinal fluid removal: either large volume LP (30–50 mL of removal) and/or external lumbar drainage. Documentation of the client's gait function before and within an hour of CSF removal is useful.

B. Complete blood count (CBC) with differentials.

C. Chemistry profile.

D. Thyroid function tests.

E. B_{12} and folate levels.

F. Vitamin D levels.

G. Venereal Disease Research Laboratory (VDRL) syphilis.

H. Ceruloplasmin: copper test.

I. A*B*42, tau, phospho-tau.

J. MRI preferred; or CT.

K. Cisternography: Nuclear medicine test demonstrates impaired draining of CSF from lateral ventricles in 48-hour period. NPH is suggested by the lack of isotope appearance over the brain convexities at 72 hours. Ventriculomegaly: Hallmark finding on either MRI or CT scan is the absence of out-of-proportion enlargement of the sulci and with no evidence of obstruction at the level of the third or fourth ventricles. While not considered specific to NPH, the degree of cortical atrophy helps to distinguish NPH from age-related ventricular enlargement.

DIFFERENTIAL DIAGNOSES

A. NPH.

B. AD.

C. PD.

D. Parkinson plus disorders (progressive supranuclear palsy, multiple system atrophy).

E. Chronic alcoholism.

F. Multi-infarct dementia.

G. Subdural hematoma.

H. Tumor.

I. Hypothyroidism.

PLAN

A. General interventions:

1. Levodopa challenge may be given to rule out idiopathic PD (no significant response with NPH).
2. Preparations for shunt procedure or LP drainage as determined by a neurologist and/or neurosurgeon.
3. Discuss therapy options, the benefits, risk factors, and possible outcomes.
4. Provide supportive measures as required based on the client's ability to provide self-care based on results of ADL and IADL evaluation.
5. Ensure that the client is in a safe environment and has appropriate assistive devices for ambulation and protective padding on knees and/or hips to reduce injury with fall incidences.
6. Encourage daily exercise. Physical therapy may be beneficial.
7. If shunt procedure done, appropriate follow-up care of shunt and monitoring for shunt complications including overdrainage, infection, and shunt failure.

B. Client Teaching**:**

1. The benefits and risks associated with the shunt procedure.
2. Review signs and symptoms that alert the client and significant others of shunt complications.
3. Stress that improvement of symptoms does not always occur with shunting.
4. Point out that improvements occur over several months and benefits of shunt may last for several years.
5. More improvement occurs with gait than with cognition.
6. Encourage support groups for the client and significant others.

C. Pharmaceutical therapy:

1. No specific medications are helpful for NPH.
2. Medications that are prescribed for established comorbid disorders.
3. Medications usually prescribed for clients with established Alzheimer disorder may be prescribed.
4. Medication for depression if screening tests for depression are positive.
   a. Escitalopram (Lexapro), sertraline (Zoloft), or paroxetine (Paxil) may be prescribed. Monitor selective serotonin reuptake inhibitor (SSRI) for weight loss and do not use with monoamine oxidase (MAO)-I.

FOLLOW-UP

A. Follow up on improvement of gait, urinary incontinence, and cognition.

B. Monitor shunt function and changes that may indicate that shunt is not functioning properly or is infected. Symptoms to monitor include new-onset headaches, altered mental status, and fever/chills.

C. Follow up mental status tests to determine cognitive improvements.

CONSULTATION/REFERRAL

A. Consider referral to a home health nurse, physical therapy, and social work to develop a working plan for home care to optimize function and reduce caregiver stress. ▶

B. Consider a referral to an access living expert that can evaluate and make recommendations about changes/ improvements in the residence to prevent injuries and maintain a degree of independence.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. NPH is a condition of older persons, primarily ages 60 to 70.

2. Persons afflicted with NPH will have normal age-related changes as well as other comorbidities, especially arteriosclerotic vascular disorders, that make a diagnosis of NPH difficult to isolate, diagnose, and treat.

3. Even if idiopathic NPH is the established diagnosis without other comorbidities, treatment and full resolution of symptoms, especially cognition, are not possible:

a. Current research indicates evidence that ventriculoperitoneal (VP) shunting is effective, safe, and recommended for geriatric clients diagnosed with NPH. One to three months post-VP surgery, the following beneficial outcomes were reported:

i. CSF protein concentration associated with antioxidation and antiaging increased, which suggested the brain might be less predisposed to neurodegeneration.

ii. CSF proteins associated with inflammation and toxins decreased.

iii. Gait analysis revealed lower cadence, longer step length, and increased single-limb support.

iv. Overall balance improvement and stable ambulation.

4. Brain comorbidities such as AD will affect the outcomes of NPH treatment. The gait may not improve, stride time variability could continue to deteriorate, and apathy might not show signs of improvement. However, consider the benefits of decreasing/resolution of other symptoms such as headache, blurry vision, bladder control, nausea, lethargy, irritability and imbalance, which all would improve quality of life (QOL).

5. The Hydrocephalus Association has a support website and mobile app for NPH clients and caregivers. It is found at www.hydroassoc.org.

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PARKINSON DISEASE

DEFINITION

A. Parkinson disease (PD) is an idiopathic, progressive, chronic neurologic syndrome characterized by a combination of akinesia or bradykinesia, or reduction of spontaneous activity and movement; rigidity, or increase in spontaneous muscle tone and involuntary movements; tremor; and postural instability.

INCIDENCE

A. PD affects between 100 and 200 per 100,000 people over 40, and over 1 million people in North America alone. It is uncommon in those younger than 40. The incidence of PD increases over 60 years, with a mean age at diagnosis of 70.5 years. It is suspected that males may have a higher risk of developing PD than females.

PATHOPHYSIOLOGY

A. For reasons that are unclear, degenerative changes occur in the basal ganglia and deplete the dopaminergic neurons in the substantia nigra, resulting in dopamine reduction in the striatum. This interrupts neuronal circuits and produces akinesia and rigidity. The pathophysiology of tremor is less clear, but thalamic involvement is implicated. Symptoms are caused by loss of neurons that produce dopamine. Certain nerve cells (neurons) in the brain break down or die. The severity of motor symptoms appears to be an independent predictor of mortality.

PREDISPOSING FACTORS

A. Antecedent encephalitis.

B. Arteriosclerosis.

C. Trauma.

D. Toxins.

E. Drugs, particularly phenothiazines.

F. Familial neurodegenerative diseases in which parkinsonism is a prominent feature.

G. Presence of Lewy bodies.

COMMON COMPLAINTS

A. Cardinal symptoms:

1. Tremor at rest. It is typically described as "pill rolling" and may be intermittent but progresses over time.

2. Rigidity: Joints are more rigid. Increased resistance to passive movement. Appears unilaterally and then progresses to the opposite side. Usually asymmetrical. Appears as decreased movement in arm swing when ambulating, stooped posture, cogwheel rigidity—resistance with tremor.

3. Bradykinesia, or slow voluntary movement, especially with daily activities such as cutting food, dressing self, and so forth. When walking, shorter steps are taken, shuffle-step, feeling of unsteadiness with walking. It is present at onset of PD in close to 80% of clients.

4. Postural instability is an impairment of centrally mediated postural reflexes that causes a feeling of imbalance. Those affected are at a high risk of falls. It usually does not appear until later in the course of PD.

OTHER SIGNS AND SYMPTOMS

A. Micrographia (handwriting that decreases in size when writing out name).

B. Voice changes: fading, softness, hoarseness, and mumbling.

C. Saliva escaping mouth, especially at night.

D. Dysphagia.

E. Neuropsychiatric changes: cognitive impairment/dementia/memory loss, sleep disturbance, fatigue, anxiety, depression, pain, and sensory changes.

F. Oily, greasy skin.

G. Excessive perspiration.

▶

H. Constipation.
I. Urinary hesitancy or frequency.
J. Visual loss: impaired vision, reflex, upward gaze, and convergence.
K. Impaired posture and balance.

SUBJECTIVE DATA

A. Elicit information regarding onset of symptoms. Note changes in progression of symptoms.
B. Talk with the client and family to establish if there have been behavioral changes, problems with activities such as eating or getting out of chairs, or personality changes.
C. Determine if other family members have had similar symptoms.
D. Ascertain the client's medical history, including current medications, both prescription and over-the-counter (OTC).
E. Particularly in clients younger than 55 years, investigate substance abuse and exposure to herbicides or pesticides.

PHYSICAL EXAMINATION

A. Check pulse, respirations, blood pressure (BP), and weight: Note orthostatic hypotension.
B. Inspect:
1. Observe overall appearance.
2. Note asymmetric tremor at rest.
3. Note subtle facial masking, increased drooling, decreased frequency, and amplitude of eye blinks.
4. Note posture and gait disturbances: festination, or shuffling, increasingly tiny steps; usually walking with arms down to side; difficulty turning; freezing, or inability to continue to move.
C. Palpate:
1. Palpate extremities, noting increased tone in resting muscles.
D. Neurologic exam:
1. Perform complete neurologic exam. Assess all cranial nerves (CNs). Assess deep tendon reflexes (DTRs).
2. Assess rapid alternating movements. Note difficulty with rapid alternating movements such as tapping fingers or turning palm alternately up and down.
3. Check cogwheel phenomenon, which is stepwise rigidity of movement with passive range of motion (ROM) rather than anticipated smooth movement through ROM. Best tested in wrists.
4. Perform mental status exam.
5. Assess the progression of the disease state with the scale of choice. Scales commonly used include:
a. Unified PD Rating Scale. Available at https://img.medscape.com/fullsize/701/816/58977_UPDRS.pdf.
b. International Parkinson and Movement Disorder Society. This site hosts a list of rating scales and questionnaires: www.movementdisorders.org.
c. Hoehn and Yahr Scale. Available at https://emedicine.medscape.com/article/2172546-overview.
d. Scales for Outcomes in PD—Psychiatric Complications (nonmotor evaluation) and Nonmotor Symptom Screening Questionnaire. Available at https://eprovide.mapi-trust.org/instruments/non-motor-symptoms-questionnaire.

DIAGNOSTIC TESTS

A. There is no definitive diagnostic test for PD.
B. Keystroke dynamic features are now being used to focus on the pattern and cadence of a person's typing. This in conjunction with other tools has led to increasing accuracy in early detection of PD, allowing for an easier more accurate and earlier diagnosis of PD that is both objective and noninvasive.
C. Urinalysis to rule out urinary tract infection (UTI) with any urinary symptoms.
D. Speech therapy evaluation of dysarthria and dysphagia to assess aspiration risk.
E. Brain CT scan or MRI to exclude mass lesion, acute infarct, or normal pressure hydrocephalus (NPH).
F. MRI of cervical spine if there is increased gait disturbance after a fall.

DIFFERENTIAL DIAGNOSES

A. PD.
B. Essential tremor.
C. Multiinfarct dementia.
D. Alzheimer disease (AD).
E. Brain tumor.
F. Progressive supranuclear palsy.
G. NPH.
H. Shy–Drager syndrome.
I. Hypothyroidism.
J. Hereditary disease such as Huntington chorea or Wilson disease.
K. Chorea: not generally seen in PD; its development in a client with PD is generally a medication side effect and should be discussed with the client's neurologist.

PLAN

A. General interventions:
1. Encourage regular exercise to maintain or improve flexibility.
2. The client should follow a diet that is high in fiber and calcium, with adequate fluid intake, to limit complications due to constipation and osteoporosis. In some clients, protein intake may need to be timed to limit interactions with levodopa.
3. Emphasize the importance of the nonmotor symptoms being addressed and adequately treated. Encourage the family to notify the provider if these symptoms are not being controlled. Anxiety, depression, fatigue, mood, and behavioral issues need to be addressed and controlled for quality of life (QOL) for the client and the family.
4. Home safety evaluations are recommended because the symptoms of PD place clients at high risk for falls and accidental injury.
5. Surgery, such as pallidotomy or thalamotomy, is an option for severe PD in which tremor is poorly controlled with medications.
B. Client teaching: *See Client Teaching Guide for this chapter, "Managing Your Parkinson Disease."*
C. Pharmaceutical therapy:
1. Polypharmacy is the hallmark rather than the exception with PD. Always comanage with a neurologist.
2. Lower doses of several medications, rather than high doses of a single agent, aid in maximizing function while minimizing side effects.
3. Drug dosages are always tapered, not stopped abruptly.
4. First-line drug:
a. Levodopa, combined with a decarboxylase inhibitor, is the mainstay of treatment. Sinemet is the levodopa and carbidopa combination drug most often used:
i. The dose and dosing frequency are very individualized.
ii. Clients are often on a combination of sustained-release and short-acting preparations. ▶

iii. See literature for individual dosing. Titrate dose up every 3 days for adjustments.

iv. Long-term use is often associated with adverse effects requiring careful medication dosage.

5. Second-line therapy:

a. Dopamine agonists (DAs): Generally given in conjunction with levodopa, these allow use of lower doses of levodopa that can delay or reduce levodopa-associated problems. Examples include pramipexole (Mirapex) and ropinirole (Requip).

6. Ergot derivatives were once used in clients with PD; however, they have largely been replaced by non-ergot DAs in PD due to rare, but potentially severe complications associated with long term use.

a. Pergolide was withdrawn from the U.S. market in 2007.

b. Nonergot drugs are preferred due to fewer side effects:

i. Pramipexole (Mirapex) 0.125 mg TID up to 4.5 mg/d maximum is useful for tremors, or ropinirole (Requip) 0.25 mg TID, maximum 24-hour period. Avoid combining sedating medications while taking Mirapex.

7. Neuroprotective agents:

a. Selegiline (Eldepryl), monoamine oxidase (MAO)-B inhibitor 5 mg tablet at breakfast and at lunch, may have neuroprotective effects and slow progression of symptoms. Selegiline is often an initial treatment and is usually continued throughout the course of the disease. Maximum dose is 10 mg/d.

b. Amantadine (Symmetrel) is used as short-term monotherapy in clients younger than 60 years with mild to moderate PD in which akinesia and rigidity are more prominent than tremor:

i. Its effects tend to wane, and it should be tapered once other antiparkinsonian drugs are started.

ii. The usual dose is 100 to 300 mg BID. Adjust the dose gradually.

iii. Caution should be used with these medications due to the interactions with other medications and foods that can precipitate high BP to dangerous levels. Advise to avoid foods high in tyramine, such as some cheeses, tofu, and yeast extracts.

8. Anticholinergics: These are useful for treating resting tremor but not akinesia or impaired postural reflexes:

a. The centrally acting drug trihexyphenidyl HCl (Artane) is the most common anticholinergic used:

i. It is usually started at 1 mg daily with food and slowly increased if needed per manufacturer's guidelines. Consider dosage reduction when used in conjunction with levodopa.

ii. Dosage should always be tapered; never stopped abruptly.

iii. Use is not recommended in individuals older than 60 or those with dementia.

b. Benztropine mesylate (Cogentin) 0.5 to 1 mg at bedtime may also be used in PD. Maximum is 6 mg/d. Increase every 5 to 6 days.

9. Sleep disorders are common in PD and respond well to tricyclic antidepressants (TCAs), benzodiazepines, and diphenhydramine.

10. Excessive daytime sleepiness should first be evaluated as a symptom of depression before it is attributed to medications or effects of PD.

As PD progresses, clients often develop clear "on" and "off" times of medication effectiveness and functional ability, so medication schedules are very carefully customized to maximize "on" times.

FOLLOW-UP

A. PD requires lifelong management by a neurologist.

B. Frequency of appointments depends on severity of disease and response to medication. Depression and neuropsychiatric side effects of medications are often seen in clients with PD, so any office visit should involve screening for these. Inquire particularly about memory loss, vivid dreams or nightmares, hallucinations, symptoms of depression or anxiety, and occurrence of panic attacks. Discuss findings with the client's neurologist, as medication adjustments could be required.

CONSULTATION/REFERRAL

A. Managing PD requires referral to a neurologist to initiate and adjust medications.

B. If a PD client requires the addition of medication for other conditions, consult the client's neurologist to evaluate for possible serious adverse effects.

C. Involvement with a support group can be helpful for the client and family. Information about PD and local support groups can be obtained from the following:

1. The American Parkinson's Disease Association, Inc.
135 Parkinson Avenue
Staten Island, NY 10305
www.apdaparkinson.org
800-223-2732 Fax 718-981-4399

2. National Parkinson Foundation
1501 N.W 9th Ave./Bob Hope Road
Miami, FL 33136-1494
www.parkinson.org
800-473-4636

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. The prevalence of PD increases with age.

2. Management of PD in this population is challenging due to the possibility of existing comorbidities.

3. Medication compliance is also an important consideration. Multidisciplinary approach to management of PD should be emphasized.

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RESTLESS LEGS SYNDROME

DEFINITION

A. Restless legs syndrome (RLS), also known as Willis–Ekbom disease (WED), is a neurologic disorder usually ▶

involving throbbing, pulling, creeping, or other unpleasant sensations of the legs with sometimes an overwhelming urge to move them. Symptoms occur usually at night when in a relaxing, resting position, but can increase in severity throughout the night. Moving the legs usually relieves the discomfort causing disorders of sleep. Left untreated, RLS can cause exhaustion and fatigue, which are often associated with daytime concentration and memory, and can cause depression.

INCIDENCE

A. Between 5% andto 15% of the population may have RLS. Familial RLS tends to occur at ages younger than 45 years. Ages can be from childhood to older than 90 years.
B. RLS occurs in both males and females, but the incidence is about twice as high in females.
C. African Americans are less frequently affected as compared with Whites. Of pregnant individuals, 11% to 29% are affected. It is three times more common in those who are pregnant than in nonpregnant individuals, with a higher rate in the third trimester. As many as 25% to 50% of clients with end-stage renal disease (ESRD) have RLS.

PATHOGENESIS

A. RLS is classified as a movement disorder as people are required to move their legs to get any relief. More than 80% of people with RLS may also experience a condition, periodic limb movement of sleep (PLMS), which involves leg twitching or jerking movements during sleep occurring every 15 to 40 seconds. Although many clients with RLS develop PLMS, most people with PLMS do not have RLS or any other cause of PLMS. Periodic limb movement disorder (PLMD) may be a variant of RLS and responds to similar treatments. The cause of RLS is unknown; however, it may have a genetic component. Nearly half of people with RLS also have a family member with the condition. Evidence indicates that low levels of iron in the brain may be responsible for RLS.
B. Considerable evidence suggests that RLS is a dysfunction in the brain's basal ganglia that uses the neurotransmitter dopamine. Disruption of this pathway frequently results in involuntary movements.
C. Alcohol and sleep deprivation may aggravate or trigger RLS symptoms.

PREDISPOSING FACTORS

A. Alcohol use.
B. Sleep deprivation.
C. Chronic diseases such as kidney failure, diabetes, anemia, and peripheral neuropathy.
D. Certain medications such as antiemetics, antipsychotic drugs, antidepressants, and some cold and allergy medications.
E. Pregnancy, especially during the third trimester.
F. Family history of RLS.
G. Varicose veins.

COMMON COMPLAINTS

A. A classic feature of RLS is that the symptoms are worse at night, with a distinct symptom-free period in the early morning.
B. Uncomfortable sensations in the legs with an irresistible urge to move the limb. Sensations may occur on only one side of the body, but most often affect both sides.
C. Need to keep legs in motion, such as pacing the floor, moving legs while sitting, and tossing and turning in bed.
D. Clients describe the symptoms as crawling, creeping, pulling, itching, drawing, or stretching, all localized to deep structures rather than superficial. Pain/tingling type of paresthesia of the type that occurs with peripheral neuropathy is usually absent. No sensitivity in touching the skin.

OTHER SIGNS AND SYMPTOMS

A. May vary day to day, in severity and frequency, and from person to person.
B. Triggering factors may include long car trips; sitting for long periods of time, such as at a movie theater or long-distance flights; immobilization of a cast; or relaxation exercises.
C. Worsening of symptoms occurs with sleep deprivation.
D. In severe cases, interruption and impairment of daytime function occur.

SUBJECTIVE DATA

A. Ask the client to describe the sensations (urge to move legs) of which they are complaining. Do sensations occur in one or both legs?
B. What makes symptoms worse? What makes symptoms better?
C. What does the client do to make the sensations better?
D. Do symptoms occur during rest or activity? Is there a particular activity that usually makes the symptoms worse or better? Do they improve with movement?
E. How often are symptoms present? Do the symptoms occur during the daytime or nighttime? Daily, nightly, several times a day, once a week, and so forth?
F. Does the client have other medical or behavioral conditions that can be attributed to the symptoms that occur?
G. Are symptoms triggered by rest, relaxation, or sleep?
H. Determine sleep patterns and disturbances.
I. Review current medications being taken and discuss chronic conditions.
J. Inquire regarding the amount of alcohol ingested each day.
K. Inquire regarding the use of tobacco.
L. If pregnant, are symptoms new during the pregnancy or have they always had the symptoms?

PHYSICAL EXAMINATION

A. Check blood pressure (BP), pulse, respirations, and weight.
B. Inspect:
    1. Overall appearance and hygiene.
C. Auscultate:
    1. Heart.
    2. Lungs.
D. Neurologic examination:
    1. Perform full neurologic examination.

DIAGNOSTIC TESTS

A. Laboratory tests may be performed to rule out other conditions, which include the following:
    1. Iron studies: serum ferritin level.
    2. Complete blood count (CBC).
    3. Vitamin B_{12} and folic acid.
    4. Complete metabolic panel (CMP).
    5. Hemoglobin A1C (Hgb A1C).
    6. Thyroid studies.
B. Sleep study.
C. If radiculopathy or neuropathy is suspected, an electromyography (EMG) or nerve conduction study can be performed.

DIFFERENTIAL DIAGNOSES

A. RLS: There is no specific test for RLS. The basic criteria for diagnosing the disorder are the following:

1. Symptoms are worse at night and are absent or negligible in the morning.

2. A strong and often overwhelming need or urge to move the affected limb(s), often associated with paresthesias or dysesthesias.

3. Sensory symptoms that are triggered by rest, relaxation, or sleep.

4. Sensory symptoms that are relieved with movement, in which the relief persists as long as the movement continues.

5. None of the previously noted symptoms are caused by another medical condition.

B. Sleep apnea.

C. Alcoholism.

D. Specific vitamin deficiencies.

E. Pregnancy (third trimester).

F. Parkinson disease (PD).

G. Radiculopathy.

H. Neuropathy.

PLAN

A. General interventions:

1. Activities that worsen symptoms should be discontinued.

2. Medications can be prescribed and are effective for many clients.

3. Some chronic conditions may contribute to RLS, such as diabetes or peripheral neuropathy, or vitamin deficiency, and should be evaluated.

B. Client teaching:

1. Advise client to discontinue activities that worsen symptoms.

2. Lifestyle changes may be suggested, such as discontinuing the use of alcohol, tobacco, and caffeine.

3. Bedtime rituals and regular sleep patterns should be encouraged. Avoid exercise at least 1 to 2 hours before bedtime. Encourage relaxation techniques before going to bed.

4. The use of relieving techniques, such as massage, warm bath, or warm/cold compress, is encouraged.

C. Pharmaceutical therapy:

1. Dopaminergic agents (increase dopamine) are recommended first-line treatment for frequent or nightly symptoms. However, caution should be used. Long-term use can lead to worsening symptoms as early as a few months after initiation of drugs. Augmentation manifests as earlier onset of RLS symptoms during the evening, shorter latency to onset after assuming a restful position, increased intensity, or extension of the symptoms to the upper body. Rebound refers to an increase in severity of symptoms occurring in the morning. This is reversible with withdrawal of the medication. Medications are best initiated at low doses and taken 1 to 2 hours before bedtime to allow for sufficient absorption.

a. Food and Drug Administration (FDA)-approved nonergotamine dopamine agonists for moderate to severe RLS include the following:

i. Ropinirole (Requip): 0.25 mg orally once 1 to 3 hours before bedtime. May gradually increase dose based on response per manufacturer's guidelines. Maximum is 3mg/d.

ii. Pramipexole (Mirapex): initial dose: 0.125 mg orally once 2 to 3 hours before bedtime. Dose may be increased gradually per manufacturer's guidelines based on response. Maximum dose is 0.75 mg/d. Requires dosage adjustment in renal impairment.

iii. Rotigotine (Neupro) transdermal patch: 1 mg/24 hours patch; apply new patch nightly at alternating sites. May increase patch weekly to a maximum dose of 3 mg/24 hours.

b. Gabapentin enacarbil (Horizant) is also FDA-approved for RLS treatment:

i. 600 mg once daily with food is recommended to take about 5p.m. A lower starting dose (300 mg) is recommended for older adults.

ii. Should not be cut, crushed, or chewed.

iii. **Not recommended for clients with a creatinine clearance <30 mL/min or on hemodialysis.**

c. Levodopa formulation:

i. Levodopa/carbidopa or levodopa/benserazide: 50/12.5 mg starting dose; may increase to maximum dose of 200/50 mg.

2. Pregabalin: initial: 50 to -75 mg daily, 1 to 3 hours before bedtime and may gradually increase every 5 to 7 days based on response. Usual effective dose of 150 to 450 mg/d. May require renal dosing adjustments.

3. Analgesics, such as acetaminophen (Tylenol), nonsteroidal anti-inflammatory drugs (NSAIDs), and opioids, may be used as needed for pain.

4. Benzodiazepines, such as clonazepam 0.5 to 2 mg at bedtime, may be used as needed.

5. Vitamin deficiencies should be treated with appropriate vitamins as diagnosed.

6. Medications should not be stopped abruptly. Please talk to a provider prior to discontinuing therapy. Discuss other medications with a provider before starting. Review renal dosing and pregnancy considerations if applicable on all therapies before prescribing.

FOLLOW-UP

A. Follow up in 1 to 2 weeks to evaluate effect(s) of medications.

B. Evaluate severity or minimization of symptoms.

C. Reassure clients that a diagnosis of RLS does not indicate the onset of another neurologic disorder such as PD.

CONSULTATION/REFERRAL

A. Consider referral to a neurologist if symptoms are not improving with treatment.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Symptoms are usually worse in the third trimester, but usually improve after delivery.

2. Based on animal data, gabapentin enacarbil (Horizant) may cause fetal harm.

3. Consider discussing with an obstetrician prior to initiating new medications.

B. Geriatrics:

1. Symptoms progress with age. Other chronic diseases are also usually present; it can be difficult to pinpoint the diagnosis of RLS because of other chronic problems.

2. Use of medications to treat RLS increases the risk of the following geriatric syndromes:

a. Falls: Side effects of medications include dizziness, somnolence, and dyskinesia. ▶

b. Perform a fall risk assessment when prescribing medications for RLS.

3. Beers Criteria caution: Benzodiazepines should be avoided in the elderly.

RESOURCES

Restless Legs Syndrome Foundation: www.rls.org
National Sleep Foundation: www.sleepfoundation.org

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SEIZURES

DEFINITION

A. In 2013, the International League Against Epilepsy (ILAE) adopted its commissioned task force's definition of epilepsy to be utilized in clinical diagnosis. The official report of the ILAE was published in 2014 in the journal *Epilepsia* and is available at www.ilae.org/files/dmfile/Definition2014-RFisher.pdf. Epilepsy is a disease of the brain that is defined by any of the three following conditions:

1. At least two unprovoked (or reflex) seizures occurring more than 24 hours apart.

2. One unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years.

3. Diagnosis of epilepsy syndrome.

B. The ILAE's clinical definitions also state that epilepsy is considered to be resolved for individuals who had an age-dependent epilepsy syndrome but are now past the applicable age or those who have remained seizure-free for the past 10 years, with no seizure medications for the past 5 years.

C. The 2017 ILAE classification of epilepsies now defines three seizure types:

1. Focal onset:
- a. Focal awareness seizure (FWS).
- b. Focal impaired awareness seizure (FIAS).
- c. Focal motor seizure (FMS).
- d. Focal nonmotor seizure (FNMS).
- e. Focal to bilateral tonic-clonic seizure (FBTCS).

2. Generalized onset:
- a. Generalized tonic-clonic seizure (GTCS).
- b. Generalized absence seizure (GAS).
- c. Generalized motor seizure (GMS).
- d. Generalized epileptic spasm (GES).

3. Unknown onset (motor and nonmotor):
- a. Unknown onset tonic-clonic seizure (UTCS).

D. Accurate classification of seizures is dependent on observations of witnessed seizures, full medical history including comorbidities, and clinical findings. The new basic classification is based on where the seizure begins in the brain, level of awareness, and other features of seizures:

1. Focal onset seizures: generally only in one area of the brain:

a. Motor symptoms: jerking (clonic), tense or rigid muscles (tonic), muscular limpness or weak (atonic), brief muscle twitching (myoclonus), or epileptic spams. May also have automatisms or repeated automatic movements such as clapping or rubbing hands, lip smacking or chewing, or running.

b. Nonmotor symptoms: does not affect movement such as changes in sensation, emotions, cognition change, lack of movement (behavior arrest), or autonomic function changes (gastrointestinal [GI] sensations, waves of heat or cold, goose bumps, racing heart rate).

2. GMS: involves networks on both side of the brain at the onset:

a. Motor seizures: generalized tonic-clonic. Seizure with stiffening and jerking; this loosely corresponds to "grand mal."

b. Nonmotor seizures are primarily absence seizures and correspond to the old term "petit mal." Involves brief changes in awareness, staring, and may have automatic or repeated movements like lip smacking.

3. Unknown onset seizures: may fall into the unknown onset category and later the seizure type becomes clear:

a. Motor seizures are described as tonic-clonic or epileptic spasms.

b. Nonmotor seizures include a behavior arrest; the movement stops and the person may stare and not make any other movements.

4. Focal to bilateral seizure: starts on one side or part of the brain and spreads to both sides.

E. Awareness: Whether a person is aware during a seizure is one of the main factors affecting a person's safety. Awareness is used instead of consciousness because it is simpler to evaluate:

1. Focal aware: The person's awareness remains intact, even if they are unable to talk or respond during a seizure (previous term simple partial).

2. Focal impaired awareness: impaired or affected awareness at any time of the seizure, even if a person has a vague idea of what happened (previous term complex partial seizure).

3. Awareness unknown: unable to determine if a person is aware or not, such as an unwitnessed seizure.

4. Generalized seizures: all presumed to affect a person's awareness or consciousness in some way. There are no special terms to describe awareness in generalized seizures.

F. Lennox–Gastaut syndrome (LGS) is a rare form of epilepsy consisting of multiple seizure types. Types include cognitive impairment and drop seizures. LGS clients may require antiseizure medications, steroids or immune globulin, vagus nerve stimulation, surgical resection, and ketogenic diet.

G. Eclampsia occurs anytime in pregnancy from the second trimester to the puerperium. It is notable for the occurrence of one or more generalized convulsions and/or coma in those with preeclampsia (in the absence of other neurologic conditions):

1. Eclampsia is self-limited, and delivery is the treatment.

2. The tonic-clonic seizure generally lasts 60 to 75 seconds.

3. Fetal bradycardia lasts 3 to 5 minutes but does not necessitate an emergent Cesarean section delivery. Compensatory fetal tachycardia and transient fetal heart rate decelerations occur. Delivery should be considered for the lack of improvement in 10 to 15 minutes after maternal/fetal resuscitative interventions.

4. Seizures due to eclampsia generally resolve within a few hours to days postpartum. HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome develops in approximately 10% to 20% of females with preeclampsia/eclampsia.

INCIDENCE

A. Epilepsy is the fourth most common neurologic disease and affects people of all ages.

B. 3.4 million Americans are affected by epilepsy (1.2% of the U.S. population). There are 150,000 new cases of epilepsy every year. One in 26 people in the United States will develop epilepsy at some point in their lives.

C. Seniors: most rapid population with comorbidity of epilepsy:

1. Stroke is the leading cause of new-onset epilepsy in adults after age 65.

D. Eclampsia:

1. Mild preeclampsia: 0.5%.

2. Severe preeclampsia: from 2% to 3%.

3. 48 hours postpartum: up to 33%.

E. Photosensitivity seizures are more common in children and adolescents.

PATHOGENESIS

A. Epilepsy is a functional disorder of the brain when neurons signal abnormally. The exact cause of epilepsy and eclampsia is unknown.

PREDISPOSING FACTORS

A. Tumors.

B. Alcohol/drugs.

C. Cerebral infarction/stroke.

D. Hypoglycemia.

E. Alzheimer disease (AD).

F. Posttrauma (head injury).

G. Surgery.

H. Pregnancy (eclampsia).

I. Febrile illness.

J. Photosensitivity.

K. Risk factors for recurrent seizures:

1. Identifiable brain disease.

2. Mental retardation.

3. Abnormal neurologic examination/EEG.

4. Seizures onset after age 10.

5. Multiple types of seizures

6. Family history/genetics.

7. Poor response to antiepileptic drugs (AEDs)/combination therapy at time of withdrawal.

8. Chronic alcoholism.

L. Eclampsia risk factors:

1. Nulliparous.

2. Pregnancy-induced hypertension (PIH).

3. Teens to lower 20s and again older than 35 years.

4. Other conditions to be ruled out:

a. Stroke.

b. Hypertensive disease.

c. Space-occupying lesion.

d. Metabolic disorders (hypoglycemia, uremia, water intoxication).

e. Meningitis or encephalitis.

f. Drug use (methamphetamine, cocaine).

g. Idiopathic epilepsy.

h. Thrombotic thrombocytopenic purpura (TTP).

COMMON COMPLAINTS

A. Aura: epigastric discomfort, fear, or unpleasant smells.

B. Automatisms (swallowing, chewing, fumbling, picking clothes, or lip smacking).

C. Stiffening, then jerking of limbs.

D. Staring with/without repetitive motor behaviors.

E. Eclampsia:

1. Headache: severe or persistent frontal or occipital.

2. Blurred vision.

3. Photophobia.

4. Right upper quadrant pain/epigastric pain.

5. Altered mental status.

6. Nausea and vomiting

7. Hyperreflexia.

OTHER SIGNS AND SYMPTOMS

A. Lack of memory of seizure.

B. Impaired consciousness.

C. Postictal:

1. Confusion.

2. Amnesia

3. Fatigue.

4. Headaches.

5. Loss of urine or bowel control.

SUBJECTIVE DATA

A. Obtain a history from the client or a person who witnesses the seizure:

1. Have the witness describe the duration, part of the body affected, and qualities of the seizure.

2. Does the client have a recollection of the seizure?

3. How long did it take to feel better after the seizure?

B. Evaluate if the client has ever had seizures and ask whether this was an isolated event:

1. Were there any warning symptoms prior to the seizure?

2. What kind of warning was noted?

C. Did the client have a fever or an active/recent infection?

D. Do a thorough review of the client's medical history, including head injury, pregnancy, diabetes, and cancer.

E. Ask if there is a family history of seizures.

F. Take a full medication history including over-the-counter (OTC) and herbal products:

1. Is the client on an AED?

2. Has the client missed any doses?

3. When was the last blood level checked to evaluate therapeutic dosing?

G. Review alcohol intake. Alcohol interferes with the efficacy of AEDs.

PHYSICAL EXAMINATION

A. Check blood pressure (BP), pulse, respirations, and temperature (if indicated to rule out infection).

B. Inspect:

1. Level of awareness, orientation.

2. General overall exam for secondary injuries from fall or striking objects.

C. Auscultate

1. Lungs for possible aspiration.

D. Palpate (if applicable for any injuries):

1. Elevate neck for nuchal rigidity.

E. Neurologic examination:

1. Central nervous system (CNS) testing:

a. Wrinkle forehead/raise eyebrows.

b. Smile and show teeth.

c. Stick out the tongue/lateral tongue movement.

d. Ocular movements.

e. Visual field.

f. Finger-to-nose test.

2. Motor strength:

a. Shrug shoulders.

b. Test muscle strength: Grasp hands and squeeze. ▶

c. Check reflexes of biceps, triceps, patellar, brachioradial, and Achilles.
3. Sensory testing: pinprick.
4. Gait and posture.

DIAGNOSTIC TESTS

A. Diagnosis is confirmed by the client's history, witness accounts, neurologic examination, blood work, and clinical testing, such as EEG.
B. EEG is essential in all cases of undiagnosed transient loss of consciousness, especially in the elderly, to rule out cardiac arrhythmias as the sudden loss of consciousness.
C. MRI is the gold standard for neuroimaging. MRI is more accurate than CT.
D. Blood glucose.
E. Drug/alcohol screen.
F. Serum level of anticonvulsant.
G. Lumbar puncture (LP) if indicated for signs of infection/meningitis.

DIFFERENTIAL DIAGNOSES

A. Seizures.
B. Brain tumor.
C. CNS infection.
D. Drug/alcohol use
E. Stroke/transient ischemic attack (TIA).
F. Hypoglycemia.
G. Trauma.
H. Migraine.
I. Ménière disease.
J. Syncope:
1. Long QT and cardiac syncope.
2. Reflex anoxic.
3. Orthostatic intolerance.
4. Hyperventilation.
5. Compulsive Valsalva.
6. Vasovagal

K. Psychogenic:
1. Daydreaming/inattention.
2. Panic attacks.
3. Dissociative states.
4. Hallucination in psychiatric disorder.
5. Fabricated/factitious illness.

L. Sleep-related conditions:
1. Sleep-related rhythmic movement disorders.
2. Hypnogogic jerks.
3. Parasomnias.

M. Metabolic derangements.

PLAN

A. General interventions:
1. Obtain a consultation with a neurologist for a thorough evaluation.
2. States vary on the driving requirements/restrictions for clients with epilepsy. Individual state driving requirements are noted on the Epilepsy Foundation website: www.epilepsy.com/driving-laws.
a. A person with epilepsy has the risk of a motor vehicle accident (MVA) while driving. It is considered similar or slightly higher than in individuals with other medical conditions (diabetes, cardiovascular disease) and compares with the risk of MVA in persons with sleep apnea, alcoholism, dementia, and cellular phone use.
b. Regulatory agents, as a measure of driving risk, require having a seizure-free interval of 3 to 12 months (state-dependent). In studies, the seizure-free interval was the strongest predictor of MVA.
c. Clinicians must warn clients about possible driving risks after reduction of medications or missing AED doses and must consider the client's neurologic deficits other than seizures (e.g., cognition and visual field deficits) when making recommendations about driving. The risk factors for an MVA for persons with epilepsy are as follows:
i. Medication noncompliance:
1) Forgetting when to take the medication.
2) Drug schedule is too complicated.
3) Affordability.
4) Unable to get medications on time due to insurance restriction of mail order delays.
5) Stopping medications against medical advice.
ii. Recent history of alcohol or drug abuse.
iii. Uncorrectable brain function or metabolic disorder.
iv. Structural brain disease.
v. Frequent seizure recurrence after seizure-free intervals.
vi. Prior crashes caused by seizures.

B. Client teaching:
1. Keeping a seizure diary is extremely important for both the client and the provider. The Epilepsy Foundation *My Seizure Diary* is a self-management tool for seizures and epilepsy with a focus on self-monitoring and tracking seizures and other symptoms, managing medication and other therapies, recognizing triggers and health events that may affect seizures and wellness, and communicating with care providers. The *My Seizure Diary* also allows organization of the client's health history, gives instruction on developing seizure response plans, and helps the client remember medicines and other important information. The *My Seizure Diary* platform includes a fully responsive web application on diary.epilepsy.com, as well as native iOS and Android companion apps. The iOS and Android companion apps are available on iTunes and Google Play.
2. Seizure triggers:
a. The most common cause is missed AED and/or sudden discontinuation of meds.
b. Sleep deprivation.
c. Obstructive sleep apnea.
d. Alcohol or recreational drug use.
e. Stress.
f. Hormone fluctuations.
g. Pregnancy.
h. Photosensitivity/strobe or flashing light/intense lights.
i. Television and video games (flicker frequency).
j. Contrasting visual patterns (grids, checkerboard, and stripes).
k. Computer monitors.
l. Visual fire alarms.
m. Sunlight "shimmering off" water/through trees/through window blinds.
n. Excessive caffeine intake.
3. Review the importance of medication adherence:
a. Give oral and written dosing instructions.
b. Refill the prescription prior to running out.
c. Take the medication on a schedule (set a watch alarm, use a pill container, "mark off" the calendar). ▶

Taking an extra pill when a seizure aura occurs will not stop the seizure because it is not absorbed fast enough.
d. New prescription interaction profiles should be evaluated before starting new drugs.
e. Clients should not use supplements, herbal, or OTC medications without checking with their healthcare provider.

4. Encourage the client to use a MedicAlert identification, such as a bracelet or charm (www.medicalert.org/product/catalog/medical-ids/bracelets).

5. Excessive alcohol use (more than three drinks per day) increases the likelihood of seizures. Clients should have no more than 1 to 2 drinks a day. Avoid binge drinking.

6. First aid for grand mal seizure:
a. Stay calm.
b. Time the seizure. Call 911 if the seizure lasts longer than 5 to 10 minutes.
c. Clear the area to prevent harm from surrounding objects.
d. Turn the person to the side and do not put anything in their mouth.
e. Do not hold the person down.
f. Place a soft object under the head to prevent head injury.
g. CPR is not necessary unless the person stops breathing after the seizure.
h. Stay with the person and reassure them.
i. Help get the person home; call family or friends.
j. Immediate transport to the ED is necessary for known conditions such as the following:
i. Diabetes/hypoglycemia.
ii. Heat exhaustion.
iii. Pregnancy.
iv. Infection/high fever.
v. Poisoning.
vi. Head injury.

7. First aid for generalized nonmotor seizures:
a. Stay calm.
b. Guide the client away from any dangers.
c. Block access to hazards.
d. Do not restrain the person.
e. Stay with the person until full awareness returns.

C. Pharmaceutical therapy:

1. There is controversy concerning whether to start an AED therapy for the first seizure. AEDs are generally started after a second seizure.

2. The prescription of AEDs should be individually weighed with a risk-versus-benefit decision that includes such factors as age; sex; family planning/desire for pregnancy; current driver; type/recurrence of seizure; abnormal EEG; concurrent medications used for other comorbid conditions; history of depression, anxiety, suicidal ideation, and hepatic and renal disease; cost; client preference and lifestyle issues; and side effect profile of medication(s). Table 23.4 is a list of AEDs.

3. A neurologist consultation should be obtained for full evaluation and prescription of AED with a neurologist or primary healthcare providers managing subsequent follow-up.

4. A single-agent AED is started and titrated slowly to the lowest dose that is the most effective in seizure control with the least number of side effects:
a. Ideally, the client should be maintained on one AED; however, combination therapy may be required or the first agent discontinued.
b. When a second AED is required, the second additional medication is started by titration. Once a therapeutic level is achieved, the first AED is subsequently tapered off. During this period of time, there is an increase in side effects.
c. Switching to a generic formulation has been noted to increase the risk of seizure activity.
d. Rectal diazepam gel (Diastat) may be prescribed for use at home for clients with a history of prolonged seizures.

5. Cannabidiol (CBD) is used as an adjunctive therapy in the treatment of epilepsy, particularly in Dravet syndrome and LGS, and has been found to be superior to placebo in clinical trials. There are limited data available on the risk of rebound seizures following abrupt or rapid discontinuance of CBD treatment.

6. Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) can occur up to 4 months after the institution of AEDs, including carbamazepine (Tegretol), oxcarbazepine (Trileptal), phenytoin (Dilantin), and lamotrigine (Lamictal).

7. Females should be routinely prescribed folate supplements of 0.4 to 0.8 mg/d. The folic acid recommendation is 4 mg/d for 1 to 3 months prior to conception when the client is on valproate or carbamazepine (Tegretol).

8. Other treatments include neuromodulator devices utilized when AEDs do not control seizures. The neuromodulation device sends electrical stimulation to the brain's nerve cells:
a. Vagus nerve stimulation (VNS) therapy.
b. Responsiveness neurostimulation (RNS) therapy.
c. External trigeminal nerve stimulation (eTNS).

FOLLOW-UP

A. Follow-up by primary healthcare providers is dependent on the frequency of seizures, toxicity profile of AED, and other comorbid conditions.

B. Subsequent visits include the following evaluation:

1. Drug compliance.

2. Seizure log/diary:
a. The Epilepsy Foundation has the Seizure Severity Questionnaire available for download for individual use for clients to describe their most common type of seizure. The PDF is located at www.epilepsy.com/sites/core/files/atoms/files/ssq_0.pdf.

3. Drug concentrations, blood counts, and hepatic and renal function.

4. Premenstrual serum levels when there is an increase in seizure activity the week before menstruation.

5. Yearly drug levels are required for clients on a stable dose with no seizures.

C. There is an increased risk of suicide associated with several AEDs. Evaluation of the client 1 week after institution of therapy is prudent. Instructions should be given to notify the office concerning depression:

1. Perform serial depression/suicide screening.

2. Psychiatric comorbid conditions should be treated promptly.

D. Osteopenia and osteoporosis are noted with long-term therapy with AEDs; therefore, a DEXA scan is warranted:

1. Vitamin D and calcium may be prescribed with AEDs.

2. Encourage weight-bearing exercises.

E. Clients taking AEDs need regular dental/oral care.

TABLE 23.4 ANTIEPILEPTIC MEDICATIONS

Generic Name (Trade Name)
Brivaracetam (Briviact)
Carbamazepine (Tegretol, Epitol, Carnexiv)
Clobazam (Onfi, Frisium)
Clonazepam (Klonopin, Epitril, Rivotril)
Diazepam (Diastat, Valium)
Dibenzazepine (Oxtellar XR)
Divalproex sodium (Depakote, Depacon, Epival)
Eslicarbazepine acetate (Aptiom)
Ethosuximide (Zarontin)
Ezogabine (Potiga)
Felbamate (Felbatol)
Gabapentin (Neurontin)
Lacosamide (Vimpat)
Lamotrigine (Lamictal)
Lorazepam (Ativan)
Levetiracetam (Keppra, Roweepra)
Magnesium sulfate
Oxcarbazepine (Trileptal, Oxtellar XR)
Perampanel (Fycompa)
Phenytoin (Dilantin, Epanutin, Phenytek)
Pregabalin (Lyrica)
Primidone (Mysoline)
Rufinamide (Banzel, Inovelon)
Tiagabine (Gabitril)
Topiramate (Topamax, Trokendi XR, Qudexy XR)
Valproate (Epilim, Epival, Episenta)
Valproic acid (Convulex, Depakene, Kepakine, Orfiril, Valporal, Valprosid)
Vigabatrin (Sabril)
Zonisamide (Zonegran)

F. Each state has the legal prerogative to grant driving privileges. Clinicians cannot grant or suspend driving privileges:

1. Six states have mandatory requirements for clinicians to report their clients with seizures (California, Delaware, Nevada, New Jersey, Oregon, and Pennsylvania).

2. Some states require a letter sent to the motor vehicle department stating, "My client has seizures and has been advised not to drive."

3. All states require drivers with epilepsy or seizures to report their condition.

4. Commercial driving restrictions are stricter. Restrictions for commercial vehicles involved in intrastate commerce vary among individual states. The U.S. Department of Transportation (DOT) allows people with a history of epilepsy who have been seizure-free off medication for 10 years to obtain a commercial driver's license (CDL).

G. Consider a sleep study to evaluate for obstructive sleep apnea.

EMERGENT ISSUES/INSTRUCTIONS

A. Emergency transport may be required. Seizures longer than 5 to 10 minutes require emergent care. If the client has a persistent headache after a rest period, unconsciousness with failure to respond, unequal pupil size or excessively dilated pupils, or weakness of the limbs, immediate medical attention is essential.

CONSULTATION/REFERRAL

A. A neurology consultation is necessary for evaluation, medication initiation, and a discussion on the use of a ketogenic diet.

B. A neurosurgical consultation is necessary to evaluate a VNS or surgical options:

1. The VNS has been approved for intractable epilepsy refractor to medications.

C. Refer to an obstetrician.

INDIVIDUAL CONSIDERATIONS

A. Females:

1. Preconception counseling and a planned pregnancy are imperative:

a. Discuss the teratogenicity of AEDs:

i. Attempt to decrease to monotherapy.

ii. Taper doses of AEDs to the lowest possible dose.

iii. If there is an absence of seizures for 2 to 5 years, consider a complete withdrawal of AEDs.

iv. First-trimester use of one AED has been noted to have a two- to five fold increase in major fetal anomalies such as neural tube defects, cleft lip and palate, and cardiac anomalies.

b. Increase folic acid to 4 mg/d to help prevent neural tube defects.

c. Stress the need for regular prenatal care.

d. Offer maternal alpha-fetoprotein screening test.

e. A fetal echocardiogram may be considered to diagnose cardiac defects.

f. All care providers including nurses, pediatricians, and anesthesiologists should be aware that the client has epilepsy on admission.

g. Pre-pregnancy seizure control is the most important predictor of seizure control during pregnancy.

2. Oral contraceptives are less effective on AEDs. The failure rate is 0.7 to 3.1 per 100 females. Females taking enzyme-inducing AEDs should use a backup method or alternative birth control. Enzyme-inducting AEDs include the following:

a. Phenytoin (Dilantin).

b. Phenobarbital.

c. Carbamazepine (Tegretol).

d. Ethosuximide (Zarontin).

e. Felbamate (Felbatol).

f. Topiramate (Topamax).

g. Oxcarbazepine (Trileptal).

3. Estrogen and progesterone act on the temporal lobe, where partial seizures often begin. Seizure patterns may change during menopause.

B. Geriatrics:

1. Seizures are likely to begin from 60 to 80 years of age.

2. After stroke and dementia, epilepsy is the most common serious neurologic disorder in the elderly.

3. Focus on cardiovascular and neurologic systems in the clinical physical evaluation in the elderly.

4. Management may be more difficult depending on comorbidities and the use of other medications.

5. Beers Criteria cautions for commonly prescribed AEDs in the elderly:

a. Phenytoin:

i. Most commonly prescribed AED in the elderly.

ii. Use caution; phenytoin interacts with digoxin and warfarin.

iii. Causes sedation.

b. Sodium valproate:

i. Ataxia and tremor are not uncommon.

ii. Can cause reversible extrapyramidal systems.

iii. Liver function studies should be performed prior to initiating therapy. Repeat lab work should be obtained routinely while on this medication.

c. Carbamazepine:

i. Hyponatremia can occur, especially with coadministration of a diuretic.

ii. Increases warfarin metabolism.

d. Lamotrigine:

i. Requires a slow titration in the elderly.

e. Levetiracetam:

i. Mood and behavioral problems may occur.

ii. May require dosage adjustments in those who are renally impaired.

f. Ginkgo biloba is the most commonly used herbal for seizures in the elderly.

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STROKE

DEFINITION

A. Strokes are a medical emergency. Treatment for hypertension (HTN) is effective in the reduction of strokes across all ages and populations and is a major component of primary and secondary stroke prevention.

B. Due to the aging population, it is estimated that by the year 2030 approximately 4% of the population in the United States will have had a stroke. The risk of having a stroke increases with age 65 and older and is especially prominent in the population older than or equal to 80 years.

C. Strokes are the fifth leading cause of death in males, but the third leading cause of death in females.

D. Strokes are the leading cause of disability in the United States:

1. Nearly half of stroke survivors have residual deficits, including weakness or cognitive dysfunction 6 months after stroke.

E. Stroke mortality varies by geographic location. The United States has a "Stroke Belt" in the southeastern states. Within this Stroke Belt there is an area described as the "buckle" region that has an approximate 40% increase in strokes. Georgia, North Carolina, and South Carolina are considered in the Stroke Belt Buckle area. The Pacific Northwestern states also have high stroke mortality rates.

F. Types of stroke include the following:

1. TIAs (see section "Transient Ischemic Attack" of this chapter).

2. Ischemic stroke (87% of all cases):

a. Cerebral thrombosis.

b. Cerebral embolism.

3. Hemorrhagic (13% of stroke cases—intracerebral hemorrhage or subarachnoid hemorrhage [SAH]):

a. Aneurysmal rupture.

b. Arteriovenous malformation (AVM) rupture.

INCIDENCE

A. The incidence of stroke differs by sex, race, and ethnicity. Someone in the United States has a stroke every 40 seconds. Every 40 minutes someone dies of a stroke. Approximately more than 795,000 people in the United States have a stroke, with 610,000 of these being first or new strokes. About 185,000 strokes (approximately one of four) are in people who have had a prior stroke.

1. Ischemic stroke is greater in Hispanics/Latinos and Blacks:

a. Hispanic females and males have the highest rates of diabetes, HTN, and obesity. Stroke is the third leading cause of death in Hispanic females. The average age for a stroke among non-Hispanic Whites is 80 years of age, but among Hispanics it is 67 years of age.

b. African American males and females are more likely to have a stroke than any other population in the United States. These males and females are more likely to have strokes at a younger age and have more severe strokes related to HTN, sodium intake, sickle cell anemia, smoking, obesity, and diabetes.

c. Stroke is the fifth leading cause of death in males, killing almost the same number of males each year as prostate cancer and Alzheimer disease (AD) combined in White males.

d. One in five females in the United States will have a stroke in their lifetime:

i. Nearly 60% of stroke deaths are in females, and strokes kill twice as many females as breast cancer.

ii. Pregnancy-related HTN is the leading cause of both hemorrhagic and ischemic stroke in pregnant and postpartum females.

iii. Presence of migraine with aura, contraceptive use, and smoking have a sevenfold higher odds of stroke in females.

▶

B. One out of four people will have another stroke within 5 years.
C. History of a stroke and/or TIA averages 10% yearly risk for a stroke.
D. The risk of a stroke within 90 days of a TIA may be as high as 17%, with the greatest risk during the first 7 days.
E. The rate for ischemic stroke in clients with atrial fibrillation (AFib) is 5% yearly.
F. Hemorrhagic strokes account for 13% of strokes.
G. Brain AVMs occur in less than 1% of the general population and are estimated to be in about 1 in 2,000 to 5,000 people. AVMs are more common in males than in females.

PATHOGENESIS

A. Ischemic strokes are a complication related to atherosclerotic deposits. Plaque builds up and occludes the blood flow. The lack of blood flow to the brain causes ischemic changes. The symptoms exhibited are dependent on the location and severity of the ischemia. Ischemic strokes are also a complication of a cerebral embolism.
B. Hemorrhagic strokes are related to an aneurysm/rupture of a blood vessel or an AVM.

PREDISPOSING FACTORS

A. Cardiac causes:
  1. AFib (increases risk for stroke fivefold).
  2. Mitral and aortic valve disease.
  3. Rheumatic heart disease.
  4. Atrial and ventricular septal defects.
  5. Carotid artery stenosis.
  6. Thrombosis.
  7. Embolism.
B. Modifiable risk factors:
  1. HTN.
  2. Smoking/exposure to smoke.
  3. Diabetes.
  4. Dyslipidemia.
  5. Obesity is an independent risk factor for stroke.
  6. Physical inactivity.
  7. Sickle cell disease.
C. Nonmodifiable risk factors:
  1. Age:
    a. The age of stroke onset is older for females, with the majority occurring after age 70.
  2. Sex:
    a. Several factors are noted to increase the risk of stroke in females:
      i. Pregnancy.
      ii. Preeclampsia/eclampsia (twice the risk factor for stroke).
      iii. Hormone therapy (HT).
      iv. Contraceptive use.
      v. Migraine with aura.
  3. Race/ethnicity:
    a. Stroke risk is increased in Blacks and Hispanic/Latinos.
  4. Family history/genetic predisposition:
    a. Inherited coagulopathies.
D. TIA.
E. Migraine with aura.
F. Iatrogenic anticoagulation.
G. Illicit drug use (e.g., cocaine or methamphetamines).
H. Postsurgical complications.
I. Cerebral amyloidosis.

COMMON COMPLAINTS

A. Signs and symptoms depend on the affected vessel and surrounding brain tissue.
  1. Acute onset of focal neurologic deficit:
    a. Partial or total loss of consciousness.
    b. Headache (often described as the worst headache ever predominantly with hemorrhagic strokes).
    c. Limb weakness or numbness.
    d. Contralateral hemiparesis.
    e. Facial weakness/drooping.
    f. Speech difficulty (ranges from a total loss of speech to the inability to express and comprehend).
    g. Dysphagia (ranges from an inability to chew and swallow to swallowing liquids that may lead to aspiration).
    h. Visual changes (loss of vision, blurring, and double vision).
    i. Ataxia.

OTHER SIGNS AND SYMPTOMS

A. Cognitive changes.
B. Behavioral changes.
C. Nausea and vomiting.
D. Fatigue.
E. Seizures.

POTENTIAL COMPLICATIONS

A. Long-term sequelae are dependent on the location and severity of the ischemia or hemorrhage.
B. Death.

SUBJECTIVE DATA

A. Ask detailed questions about symptoms before, during, and after the incident:
  1. Review the exact timing of onset of symptoms.
  2. How intense were the symptoms?
  3. What were the duration and the presence of any fluctuation of symptoms?
  4. Has there been a pattern that is becoming more frequent or escalating in symptoms?
B. Interview the client, family members, witnesses, and emergency personnel for their description of behavior, speech, gait, memory, and movement.
C. Review the medical history:
  1. Previous TIA or stroke.
  2. Previous/recent surgeries, specifically carotid or cardiac surgeries and any procedures such as coronary artery bypass graft (CABG) or stents.
  3. AFib.
  4. HTN.
  5. Pregnancy or recent delivery history.
  6. Migraine headaches.
  7. Seizures.
  8. Central nervous system (CNS) infection.
  9. Illicit drug use.
  10. Presence of any metabolic disorders.
  11. Recent trauma: blunt or torsion injury to the neck/recent chiropractor manipulation.
D. Focus on precipitating factors and state of consciousness after the acute event.
E. Review all medications, including anticoagulants, oral contraceptives, HT, and over-the-counter (OTC), and particular review of substances not prescribed, as well as illicit drugs such as cocaine. ▶

F. Herbals that alter bleeding time and interact with warfarin:
- **1.** Feverfew.
- **2.** Garlic.
- **3.** Ginkgo biloba.
- **4.** Ginger.
- **5.** Ginseng.

PHYSICAL EXAMINATION

Clients presenting with acute symptoms should be quickly assessed for the need to call emergency services 911 for immediate transport to the hospital. Time is of the essence for treatment with the drug (tissue plasminogen activator (tPA).

A. Check blood pressure (BP), pulse, respirations, and pulse oximetry. Weight and height should be obtained to calculate body mass index (BMI).

B. General observation:
- **1.** Observe overall appearance, level of consciousness (LOC), ability to interact, language, difficulty swallowing, tremors, and spasticity, as well as memory skills.
- **2.** Observe the client walking (cerebellar system).

C. Inspect:
- **1.** Dermal exam:
  - a. Overall hydration status.
  - b. Look for postcarotid endarterectomy scars, presence of a pacemaker, implantable cardioverter defibrillator, or other cardiac surgical scars.
- **2.** Check pupil size and reactivity to light.
- **3.** Perform a funduscopic exam to evaluate optic disc margins, retinal plaques, and pigmentation. A "cherry red spot" may be evident in the macula in clients with central retinal artery occlusion.

D. Auscultate:
- **1.** Heart for rate, rhythm, murmurs, or rubs.
- **2.** Lungs: Note respiratory rate and pattern.
- **3.** Carotid arteries for presence of bruit.

E. Palpate:
- **1.** Palpate extremities for pulses and peripheral edema.

F. Neurologic exam:
- **1.** Cranial nerve (CN) testing:
  - a. Wrinkle forehead/raise eyebrows.
  - b. Smile and show teeth.
  - c. Stick out the tongue/lateral tongue movement.
  - d. Ocular movements.
  - e. Visual field.
- **2.** Motor strength:
  - a. Shrug shoulders.
  - b. Test muscle strength: Grasp hands and squeeze.
  - c. Check reflexes of biceps, triceps, patellar, brachioradial, and Achilles.
- **3.** Sensory testing: pinprick.
- **4.** Gait and posture (cerebellar system evaluation):
  - a. Ocular movements.
  - b. Gait.
  - c. Finger-to-nose test.
  - d. Heel-to-knee test.

DIAGNOSTIC TESTS

A. There is no "gold standard" for diagnosis of CNS infarction.

B. Noncontrasted CT (NCCT) head is usually ordered first to exclude hemorrhagic stroke and is highly sensitive for this indication.

C. MRI can reliably diagnose both acute ischemic stroke and acute hemorrhagic stroke in emergency settings. Major advantage of MRI is that diffusion-weighted imaging (DWI) is much more sensitive than NCCT for detection of an acute ischemic stroke. Disadvantages include cost and lack of access (particularly smaller rural hospitals).

D. Other imaging:
- **1.** Computed tomographic angiography (CTA) is performed by administering a bolus of intravenous (IV) iodinated contrast. This is beneficial to evaluate for detection of large vessel stenosis or occlusion. It can be helpful to triage clients for acute therapy, specifically mechanical thrombectomy. It can also evaluate for underlying vascular abnormalities.
- **2.** CT perfusion imaging (CTP) consists of a series of CT scans that are obtained following an injection of IV iodinated contrast. There, the passage of the contrast through the brain can be evaluated. It is helpful to determine the size of the infarction core (tissue that is irreversibly damaged) and the ischemic penumbra (tissue that is critically hypoperfused and potentially salvageable).
- **3.** Magnetic resonance angiography (MRA) to locate a cerebral aneurysm or large vessel occlusions.
- **4.** Cerebral angiography is more invasive. A catheter is inserted usually into the femoral artery. A technician then injects a special dye into the blood vessels that lead to the brain. Subsequent images are taken to evaluate vascular abnormalities for possible intervention.
- **5.** Carotid duplex uses ultrasound imaging and Doppler ultrasound to detect increases in blood flow velocity which can help diagnose carotid stenosis.

E. Laboratory tests:
- **1.** Emergent labs:
  - a. Glucose.
  - b. Serum chemistry profile.
  - c. Coagulation and hypercoagulability testing.
  - d. Complete blood count (CBC).
- **2.** Urgent labs:
  - a. Erythrocyte sedimentation rate (ESR).
  - b. Cardiac enzymes.
  - c. Lipid profile.
- **3.** Other laboratory tests based on history:
  - a. Urine drug screen.
  - b. Blood alcohol level.
  - c. Antiphospholipid antibodies.
  - d. Hemoglobin A1C.

F. ECG to identify cardiac dysrhythmia.

G. Echocardiogram.

DIFFERENTIAL DIAGNOSES

A. Cardiovascular accident:
- **1.** Ischemic stroke.
- **2.** Hemorrhagic stroke.

B. TIA.

C. AVM.

D. Aortic dissection.

E. Thrombosis.

F. CNS infection.

G. Trauma.

H. Brain tumor.

I. Demyelination.

J. Intoxication.

K. Seizure causing Todd paralysis.

PLAN

A. General interventions:

1. Counsel clients with a history of stroke or TIA to follow a Mediterranean-type diet instead of a low-fat diet. The Mediterranean-type diet emphasizes vegetables, fruits, and whole grains, and includes low-fat dairy products, poultry, fish, legumes, olive oil, and nuts. It also limits intake of sweets and red meat.

2. Clients with ischemic stroke, TIA, or hemorrhagic stroke who are heavy drinkers should eliminate or reduce their consumption of alcohol. Limit intake of alcohol to light to moderate amounts. One drink per day for females and up to two drinks per day for males may be reasonable. Nondrinkers should be counseled not to start drinking.

3. Lower sodium intake. Consume no more than 2,400 mg/d of sodium. Further reductions to 1,500 mg/d of sodium are associated with greater reduction in BP. Even a modest reduction in salt intake for more than or equal to 4 weeks leads to a reduction in BP.

4. Healthcare providers should strongly advise every client with stroke or TIA who has smoked in the past year to quit. It is reasonable to advise clients after a TIA or ischemic stroke to avoid passive tobacco smoke.

B. Client teaching:

1. Therapeutic lifestyle changes (TLCs), including physical activity, diet, weight management, and smoking cessation, are recommended for all clients.

2. Physical activity:

a. All adults should avoid inactivity. Some physical activity is better than none, and adults who participate in any amount of physical activity gain some health benefits.

b. The American College of Cardiology/American Heart Association (ACC/AHA) guidelines on lifestyle management include physical activity recommendations advising adults to engage in 40 minutes of aerobic physical activity three to four times a week. The aerobic exercise should involve moderate to vigorous intensity to reduce BP, low-density lipoprotein-cholesterol (LDL-C), and non-high-density lipoprotein-cholesterol (HDL-C).

c. Refer to section "Exercise" of Chapter 3, "Adult-Geriatric Assessments," for information related to exercise in older adults and adults with disabilities.

d. Physical therapy or cardiac rehab at least on initiation of an exercise regimen may be considered for individuals with disability after ischemic stroke.

3. *See Client Teaching Guide for Chapter 25, "Nicotine Dependence."*

4. Weight reduction:

a. Explain that weight reduction in clients greater than 20% over ideal body weight can lower LDL-C and triglyceride levels. Start by setting small, realistic, attainable goals such as losing 5 to 10 lbs, which encourages successful attempts.

C. Dietary management:

1. Advise the client that diet modification is the first line of therapy for hyperlipidemia.

2. Explain cholesterol-lowering diet. Give dietary recommendation sheets. See Appendix B, "Dietary Recommendations," for low-fat/low-cholesterol and DASH dietary approaches to stop HTN.

D. Pharmaceutical therapy:

1. Reperfusion therapy with IV thrombolytic drug Alteplase IV recombinant tissue plasminogen activator (r-tPA) for *ischemic stroke* **is considered the gold standard. r-tPA is utilized in the hospital setting. Use of r-tPA reduces the effects of stroke if administered within 3 hours of symptom onset. Delayed hospital arrival is the single most important reason for failure to administer tPA within the window of 3 or 4.5 hours.**

a. The 2012 American College of Chest Physicians Guidelines for Antithrombotic and Thrombolytic Therapy for Ischemic Stroke recommends the following:

i. r-tPA to be initiated within 3 hours of symptom onset for clients with an acute ischemic stroke.

ii. r-tPA can be initiated within 3 to 4.5 hours of acute symptom onset.

iii. In clients with acute ischemic stroke for whom treatment cannot be initiated within 4.5 hours of symptom onset, the College recommends against r-tPA therapy.

iv. Inclusion/exclusion criteria will differ at each facility.

2. Antiplatelet therapy is used for both the management of acute ischemic stroke and for the prevention of stroke:

a. Aspirin (ASA) is the most commonly used antiplatelet therapy. Administration of aspirin is recommended in acute stroke clients within 24 to 48 hours after stroke onset. For clients treated with tPA, aspirin administration is generally delayed for 24 hours:

i. High-dose aspirin 150 to 325 mg/d.

ii. Lower dose of aspirin 75 to 100 mg daily.

iii. Lower doses of aspirin are associated with milder GI symptoms and less GI hemorrhage.

b. Clopidogrel (Plavix) 75 mg once daily as monotherapy.

c. For clients with recently symptomatic atherosclerotic intracranial large artery stenosis:

i. Clopidogrel 300 mg loading dose, then 75 mg daily, plus low-dose aspirin daily for 90 days. Aspirin monotherapy is recommended thereafter.

ii. Dual antiplatelet therapy with aspirin and clopidogrel is not recommended for *stroke prevention.*

d. Dipyridamole as monotherapy or as dual therapy for secondary stroke prevention:

i. Immediate-release 50 to 100 mg TID is not routinely recommended for secondary prevention of ischemic stroke.

ii. Aspirin plus extended-release dipyridamole 25/200 mg BID is used as an alternative for clients intolerant to aspirin.

e. Ticlopidine is no longer available in the United States.

f. Cilostazol 100 mg BID: Cilostazol, a phosphodiesterase 3 inhibitor used mainly for intermittent claudication in clients with peripheral artery disease, has also been found effective in preventing cerebral infarction in Asian ethnic groups. There is a lack of high-quality data regarding the use of cilostazol for secondary stroke prevention in non-Asian ethnic groups.

g. Long-term anticoagulant therapy is required for secondary prevention of AFib (see Chapter 13, "Cardiovascular Guidelines," for an in-depth review of AFib). Note: The CHA_2,DS_2,-VASc score evaluates the risk of ischemic stroke in clients with AFib. To calculate a CHA_2,DS_2,-VASc score, go to www.clincalc.com/cardiology/stroke/chadsvasc.aspx. In general, the lower the score, the stronger the consideration for oral ▶

anticoagulation. The one exception to this is when the client has a score of 1 due to sex alone.

3. In most stroke clients, atorvastatin 80 mg daily is recommended to reduce the risk of stroke recurrence and a low density lipoprotein (LDL) <70 mg/dL is recommended to reduce the risk of cardiovascular events.

4. The AHA/American Stroke Association (ASA) suggest that the goal BP for most stroke clients with high BP should be <130/80.

5. In stroke clients with diabetes, medical therapies for goal glycemic control should be individualized, but for most clients a hemoglobin A1C of ≤7% is recommended.

6. Further adjuvant therapy must be managed under the direction of a neurologist after a full evaluation.

E. Surgery:

1. Mechanical thrombectomy.

2. Carotid endarterectomy (CAE).

3. Carotid angioplasty and stenting (CAS).

4. Extracranial–intracranial (EC/IC) bypass.

5. Endovascular stenting.

6. Open surgical procedures, including vertebral artery endarterectomy and vertebral artery transposition.

FOLLOW-UP

A. Follow-up is determined by the client's needs, severity of stroke, and whether complications are present.

B. Cardiac rhythm studies for more than 24 hours should be considered for nonvalvular AFib (NVAF).

C. Sleep apnea is present in approximately half to three-quarters of clients with stroke or TIA. Consider a sleep study and treatment with continuous positive airway pressure (CPAP).

D. The AHA/ASA recommend that clients with sickle cell disease and prior strokes/TIA have chronic blood transfusions to reduce hemoglobin S to less than 30% of total hemoglobin.

E. Evaluate for depression at each office visit.

F. Stroke rehabilitation may include the following:

1. Speech therapy (ST).

2. Occupational therapy (OT).

3. Physical therapy (PT).

G. Rehabilitation sites include the following:

1. Nursing facility.

2. Skilled nursing facility (SNF).

3. Assisted living facility.

4. Home.

H. Identify area support groups for the client and/or caregivers.

I. Fitness for driving varies by state.

EMERGENT ISSUES/INSTRUCTIONS

A. If a stroke is suspected, call 911; symptoms of a TIA and a major stroke are not always distinguishable.

CONSULTATION/REFERRAL

A. Neurology.

B. Cardiology, if applicable.

C. Hematologist.

D. Maternal–fetal medicine for high-risk pregnancy.

INDIVIDUAL CONSIDERATIONS

A. Females:

1. Females older than 85 years of age have one of the highest risks of ischemic stroke. The increased risk may be accounted for by females living longer than males.

2. Females have a higher rate of SAH as well as a higher risk of rupture.

3. Two major risk factors for stroke in females are oral contraceptives and pregnancy.

4. Prepregnancy HTN increases the risk for pregnancy-induced hypertension (PIH) and stroke during pregnancy.

5. The highest risk for stroke occurs in the third trimester of pregnancy.

6. Postmenopause:

a. Research indicates that natural menopause is not associated with a risk of stroke.

b. Clients with hysterectomy with bilateral oophorectomy have a slightly elevated risk of total stroke compared with those with hysterectomy with ovarian conservation.

c. The 2014 AHA/ASA guidelines for prevention of stroke in a client's HT recommendations include the following:

i. HT, conjugated equine estrogen (CEE) with or without medroxyprogesterone should not be used for primary or secondary prevention of stroke in postmenopausal individuals.

ii. Selective estrogen receptor modulators, such as raloxifene, tamoxifen, or tibolone, should not be used for primary prevention of stroke.

7. Females with asymptomatic carotid stenosis should be screened for other treatable risk factors for stroke, and appropriate lifestyle changes and medical therapies should be instituted.

B. Geriatrics:

1. Worldwide, the elderly population is quickly growing, and in America geriatrics ≥85 years of age constitute 2% of the population and this is predicted to double by year 2060. Incidence of strokes doubles in octogenarians, making preventive measures a prominent focus for clinicians regarding the "very elderly."

2. 10% of intracerebral hemorrhages from cerebral amyloidosis occur in the elderly.

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TRANSIENT GLOBAL AMNESIA

DEFINITION

A. Amnesia is a partial or complete loss of memory. There are two forms of amnesia: retrograde and antegrade. Retrograde amnesia is a state in which the individual is unable to recall past events. Antegrade amnesia is a state in which the individual has a profound amnesia to recent events and is unable to create new memories. ▶

B. Transient global amnesia (TGA) is a spontaneous, reversible syndrome of significant antegrade amnesia and a milder reduction in retrograde episodic long-term memory. During the event, individuals have a profound amnesia to recent events, are disoriented as to time, have somewhat diminished executive function but maintain self-awareness, have no clouding of consciousness or loss of personality, and exhibit no agitation. There are no focal neurologic signs or symptoms. Individuals are able to perform complex tasks previously learned, such as driving or playing a musical instrument, but are unable to learn a new task or lay down new memory during the attack. After the attack resolves, there is amnesia of the events during the episode and a few minutes to prior to the beginning of the episode. Episodes are self-limited and by definition resolve by 24 hours.

INCIDENCE

A. The incidence of TGA is approximately 5.2 to 10 per 100,000 persons a year. The incidence increases with age, so that episodes of TGA with persons 50 years and older increase to approximately 23.5 to 32 persons per 100,000 per year. The majority of TGA episodes occur between the age of 50 and 80, with the mean onset occurring in the sixth decade of life. Some studies report that TGA occurs slightly more in males than in females, while others report no difference between genders.

PATHOGENESIS

A. The primary site of neurologic involvement causing TGA is the hippocampus, particularly the CA-1 and Sommer sector and the mediobasal temporal lobe. The hippocampus is critical in making new memories and converting short-term memory into long-term memory. The pathogenesis of TGA or what triggers the episode has not yet been clearly established, but several hypotheses have been suggested. A vascular etiology such as transient ischemic attack (TIA) was thought to be a contributing cause except that TGA episodes last longer than TIA episodes.

B. Venous congestion has also been suggested as another cause based on observations that specific maneuvers such as Valsalva that increase intrathoracic pressure or venous return have preceded TGA episodes.

C. Another suggested cause is a migrainous phenomenon or cortical spreading depression (CSD; self-propagating wave of neuronal and glial depolarization that spreads across the cerebral cortex) that is thought to occur with migraine headaches.

D. Epileptic seizures have been suggested as a contributing cause, but EEGs taken during episodes of TGA do not show epileptic electrical discharges, Finally, TGA was thought to be psychogenic, but most experts believe that TGA has an organic cause even though stressors and/or psychogenic predisposition may have a role.

PREDISPOSING FACTORS

A. Definite risks:
- **1.** Advancing age.
- **2.** Migraine headaches.

B. Possible risks:
- **1.** Arterial ischemia (ischemic heart disease and carotid artery sclerotic changes).
- **2.** Hyperlipidemia.
- **3.** Significant emotional event.
- **4.** Psychiatric disorders (posttraumatic stress disorders [PTSDs] and dissociative disorders).

COMMON COMPLAINTS

A. Significant antegrade amnesia and lesser retrograde amnesia.

OTHERS SIGNS AND SYMPTOMS

A. Cognitive impairment limited to amnesia.
B. No loss of personality or clouding of consciousness.
C. Repetitive questioning.
D. Absence of neurologic signs or symptoms.
E. Absence of epilepsy.
F. Attacks resolve within 1 to 10 hours.
G. Associated symptoms: headache, nausea, and vomiting immediately prior to attack.

SUBJECTIVE DATA

A. Determine onset, course, and duration of symptoms.
B. Note the extent and depth of loss of past memory.
C. Note repetitive questioning and ability to remember new information.
D. Note the individual's ability to perform previous learned complex tasks.
E. Note level of self-awareness, consciousness, and mood.
F. Determine if any signs and symptoms preceded the amnesic event, such as headache, nausea, or vomiting.
G. Significant life event stressors, psychiatric disorders, head injury, or epilepsy.
H. Review medication history, including prescribed drugs, over-the-counter (OTC) drugs, and herbal remedies.

PHYSICAL EXAMINATION

A. Obtain vital signs: blood pressure (BP), pulse, and respirations.
B. Inspect:
- **1.** Observe overall appearance.

C. Auscultate:
- **1.** Heart and lungs.
- **2.** Carotid arteries bilaterally.

D. Neurologic examination:
- **1.** Evaluate level of consciousness (LOC) and ability to perform cognitive testing.
- **2.** Perform cognitive assessment screening tests:
  - a. Mini-Mental State Examination (MMSE). Available at www.uml.edu/docs/mini%20Mental %20State%20 Exam_tcm18-169319.pdf.
  - b. Montreal Cognitive Assessment (MOCA). Available at www.Mocatest.org
  - c. Saint Louis University Mental Status (SLUMS) Examination. Available at https://echo.unm.edu / wp-content/uploads/2014/07/clinic-dementia-mental-status.pdf.
  - d. Check deep tendon reflexes (DTRs); assess for hyperreflexia.
  - e. Assess muscle strength/tone, testing upper and lower extremities for increased tone (spasticity), tremors, and/or decreased tone.
  - f. Assess sensory response, testing perception for sharp versus dull stimulus, heat versus cold stimulus, local pain perception, and proprioception, and evaluate sense of vibration with tuning fork.
  - g. Note gait, posture, coordination, and balance.

DIAGNOSTIC TESTS

A. The diagnosis of TGA is a clinical one and is made if the following diagnostic criteria by Caplan and Hodges are fulfilled:
- **1.** Presence of anterograde amnesia that is witnessed.
- **2.** No clouding of consciousness or loss of personal identity.
- **3.** Cognitive impairment that is limited to anesthesia.
- **4.** No focal neurologic or epileptic signs.
- **5.** No recent history of head trauma or seizures.
- **6.** Resolution of symptoms within 24 hours.

▶

B. CT and/or MRI.
C. EEG, if epilepsy suspected.
D. Complete blood count (CBC).
E. Chemistry profile.
F. Lipid levels.
G. Toxicology screen.

DIFFERENTIAL DIAGNOSES

A. TGA.
B. Transient epileptic amnesia.
C. TIA.
D. Dissociative amnesia.
E. Migraine headache.
F. Hypoxic states (such as aortic dissection).
G. Hypoxic states (ischemic events).
H. Drug or alcohol withdrawal.
I. Hypoglycemia.
J. Psychogenic amnesia.
K. Metabolic derangements.
L. Wernicke encephalopathy.

PLAN

A. General interventions:
1. Admit to hospital for duration of episodic event.
2. General supportive care during the amnesic event.
3. Provide ongoing treatment for comorbidities.
4. Thiamine 1,000 mg intravenous (IV; for possible Wernicke encephalopathy).
5. Ensure emotional support and reassurance.
B. Client teaching:
1. Help the individual and significant others to understand the circumstances surrounding TGA.
2. Advise the individual and significant others that TGA does reoccur rarely but most have a single episode.
3. Memory that has been lost during the episode is permanent.
C. Pharmaceutical therapy:
1. Thiamine 1,000 mg given IV during hospitalization (for possibility of Wernicke encephalopathy).
2. No other medication needed for episode once resolved.
3. Treatment for comorbid conditions should continue as usual.

FOLLOW-UP

A. Follow-up is not usually required once the TGA episode is resolved because most do not have a recurrence.

CONSULTATION/REFERRAL

A. Referral for follow-up care depends upon the findings of diagnostic studies that were conducted in the hospital setting.
B. Refer to a neurologist for long-term deficits.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. TGA is a disorder occurring in older persons. Comorbidities occur with greater frequency in this age group. However, experts report no significant morbidity or mortality associated with this disorder.
2. In the geriatric population, distinguishing between TIA and TGA could be challenging. Both are identified with transient memory loss that is reversible, and both occur within similar age onset (60–79 years). Statistical analysis from research has shown:
a. TGA has significantly lower profiles of coronary artery disease (CAD), hypertension (HTN), diabetes mellitus (DM), and embolic disease than TIA/stroke.
b. The absence of lateralizing weakness and visual field deficits do not usually occur with TGA events.
c. Ninety-seven percent of TGA episodes last more than 1 hour (mean duration 4–8 hours).
d. In contrast, majority of TIA last less than 60 minutes and often only a few minutes.
3. Multiple studies consistently indicate that history of TGA does not increase risk for strokes.
4. Healthcare providers' coaching and interventions to TGA geriatrics must include the following:
a. Support grief expressions.
b. Offer contact information of support groups.
c. Assist family members with positive coping strategies.
d. Emphasize a progressive pace unique to the older adult, acknowledge pleasant activities, and praise success of each memory restoration no matter how small it may seem.
e. Refer to individual/family counseling and encourage family discussions.
f. Introduce cognitive behavioral therapy (CBT) that assists clients with irrational thoughts, assumptions, "filling in the blanks," and negative beliefs. Learn about CBT: www.goodtherapy.org/learn-about-therapy/types/cognitive-behavioral-therapy.

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TRANSIENT ISCHEMIC ATTACK

DEFINITION

A. A transient ischemic attack (TIA) is a brief episode of neurologic dysfunction due to the focal brain, spinal cord, or retinal ischemia (without acute infarction/tissue injury) caused by vascular occlusion. **TIAs are a medical emergency**.
B. Symptoms generally last less than an hour; however, they may have permanent sequelae. TIAs are a risk factor for recurrent risk of stroke. The highest risk is in the first 48 hours following a TIA. Approximately 15% of diagnosed strokes are preceded by TIAs. TIAs can be difficult to diagnose because symptoms are transient. Assume that all stroke-like symptoms signal an emergency.
C. Persistence of neurologic deficit suggests a stroke rather than a TIA.

INCIDENCE

A. The incidence of TIA is hard to estimate given other conditions that mimic the same symptoms. Most recent data show TIA incidence in the United States to possibly be around half a million per year and estimates are about 1.1 per 1,000 in the U.S. population. Estimated overall prevalence among adults in the United States is 2%.
B. The early risk of stroke is approximately 5% at 2 days and as high as 11% at day 7 after a TIA.
C. The risk of a stroke within 3 months of a TIA is 10% to 15%. ▶

D. More than one-third of those who have a TIA will have a major stroke within a year if risk factors are not addressed.
E. The risk of death from coronary artery disease and stroke is as high as 6% to 10%, depending on other risk factors.
F. More males (101/100,000) than females (70/100,000) are affected. There is an increased number of Black adults with TIAs (98/100,000) than White adults (81/100,000) affected by TIAs.

PATHOGENESIS

A. The pathophysiology depends on the subtype. It is caused by a brief interruption of blood flow to an area of the brain supplied by the corresponding artery. The subtypes are as follows:
1. Large artery atherothrombosis (most common).
2. Small vessel ischemic disease.
3. Cardiac embolism.
4. Cryptogenic.
5. Hypercoagulable states or arterial dissections.

PREDISPOSING FACTORS

A. Hypertension (HTN):
1. Systolic blood pressure greater than 130 mmHg.
2. Diastolic blood pressure greater than 80 mmHg.

B. Atherosclerosis.
C. African American.
D. Age older than 40 years.
E. Hypotensive episodes.
F. Oral contraceptives/hormone therapy (HT).
G. Atrial fibrillation (AFib).
H. Smoking.
I. Familial hyperlipidemia.
J. Diabetes mellitus (DM).
K. Valvular heart disease.
L. Infective endocarditis.
M. Migraine with aura.
N. Herbal medications alter bleeding time and interact with warfarin:
1. Feverfew.
2. Garlic.
3. Ginkgo biloba.
4. Ginger.
5. Ginseng.

O. Underlying hypercoagulable disease.

COMMON COMPLAINTS

A. Signs and symptoms depend on the affected vessel and surrounding brain tissue.
1. Acute onset of focal neurologic deficit:
   a. Limb weakness or numbness.
   b. Facial weakness.
   c. Speech difficulty to aphasia.
   d. Visual loss/blurring.
   e. Ataxia.
2. Acute change in level of consciousness (LOC) or confusion.
3. Posterior circulation TIAs may have a headache as one of the prodromal symptoms that precede a stroke by days or weeks.
4. Basilar artery occlusion TIAs have vertigo, nausea, and headaches that may occur as early as 2 weeks or more prior to the onset of stroke.

OTHER SIGNS AND SYMPTOMS

A. Dysarthria.
B. Dysphagia.
C. Near syncope.
D. Hemiparesis.
E. Temporary monocular blindness.
F. Behavior changes.
G. Difficulty with balance and vertigo.
H. Dizziness.
I. Diplopia.

SUBJECTIVE DATA

A. If a TIA is suspected, call 911; symptoms from a TIA and a major stroke are not always distinguishable.
B. Ask detailed questions about symptoms before, during, and after the spell:
1. Review the exact timing of onset of symptoms.
2. How intense were the symptoms?
3. What was the duration of any fluctuation of symptoms?
4. Has there been a pattern that is becoming more frequent or escalating in symptoms?

C. Interview the client, family members, witnesses, and emergency personnel for their description of behavior, speech, gait, memory, and movement.
D. Focus on precipitating factors and state of consciousness after the acute event.
E. Question the client about risk factors such as HTN, smoking, cardiac disease, and heredity.
F. Review all medications, including anticoagulants, over-the-counter (OTC), and herbals and illicit drug use such as cocaine.
G. Review the medical history:
1. Recent surgeries, specifically carotid or cardiac surgeries.
2. Seizures.
3. Central nervous system (CNS) infection.
4. Illicit drug use.
5. Presence of any metabolic disorders.
6. Recent trauma (blunt or torsion injury to the neck).
7. AFib.
8. Migraine headaches.

PHYSICAL EXAMINATION

A. Check temperature (if infectious process is suspected), pulse, respirations, and blood pressure, including orthostatic blood pressure, as well as pulse oximetry.
B. General observation:
1. Observe overall appearance, LOC, ability to interact, language, difficulty swallowing, tremors, and spasticity, as well as memory skills.
2. Observe the client walking (cerebellar system).

C. Inspect:
1. Dermal exam:
   a. Overall hydration status.
   b. Look for postcarotid endarterectomy scars, presence of a pacemaker, implantable cardioverter defibrillator, or other cardiac surgical scars.
2. Check pupil size and reactivity to light.
3. Perform a funduscopic exam to evaluate optic disc margins, retinal plaques, and pigmentation.

D. Auscultate:
1. Heart for rate, rhythm, murmurs, or rubs.
2. Lungs: Note respiratory rate and pattern.
3. Carotid arteries for presence of bruit.

E. Palpate:
1. Palpate extremities for pulses and peripheral edema.

F. Neurologic exam:
1. Cranial nerve (CN) testing:
   a. Wrinkle forehead/raise eyebrows. ▶

b. Smile and show teeth.
c. Stick out the tongue/lateral tongue movement.
d. Ocular movements.
e. Visual field.

2. Motor strength:
a. Shrug shoulders.
b. Test muscle strength: Grasp hands and squeeze.
c. Check reflexes of biceps, triceps, patellar, brachioradial, and Achilles.

3. Sensory testing: pinprick.
4. Gait and posture (cerebellar system evaluation):
a. Ocular movements.
b. Gait.
c. Finger-to-nose test.
d. Heel-to-knee test.

DIAGNOSTIC TESTS

A. Pulse oximetry.
B. Laboratory tests:
1. Emergent labs:
a. Glucose.
b. Serum chemistry profile, including creatinine.
c. Coagulation and hypercoagulability testing.
d. Complete blood count (CBC).

2. Urgent labs:
a. Erythrocyte sedimentation rate (ESR).
b. Cardiac enzymes.
c. Lipid profile.

3. Other laboratory tests based on history:
a. Urine drug screen.
b. Blood alcohol level.
c. Antiphospholipid antibodies.
d. Rapid plasma reagin (RPR) for syphilis.

C. MRI or CT scan within 24 to 48 hours of symptom onset.
D. Carotid Doppler ultrasonography identifies clients with urgent surgical needs.
E. Cardiac imaging to evaluate cardioembolic sources.
F. EKG to evaluate dysrhythmias (i.e., AFib).
G. Lumbar puncture (LP) to rule out infection, demyelinating disease, and subarachnoid hemorrhage (SAH).
H. EEG as indicated for seizure activity.
I. Consider Holter monitor for suspected intermittent AFib.

DIFFERENTIAL DIAGNOSES

A. TIA.
B. Ischemia stroke.
C. SAH/subdural hematoma.
D. Migraine.
E. Hypoglycemia/HTN.
F. Epilepsy: postictal period.
G. Malignant HTN.
H. Brain tumor.
I. Bell's palsy.
J. Multiple sclerosis (MS).
K. Syncope.
L. Drug-induced.
M. Concussion.
N. Vertigo.

PLAN

A. General interventions:
1. Carefully assess the client for timely diagnosis of TIA.
2. Perform a full workup to determine the underlying disease process.
3. Prevent stroke by modification of risk factors.

B. Client teaching: *See Client Teaching Guide for this chapter, "Transient Ischemic Attack."*
C. Medical and surgical management:
1. Treat TIAs with antiplatelet drugs as soon as intracranial bleeding is ruled out.
2. Consider carotid endarterectomy.
3. Lipid control.
4. Glucose control.
5. Smoking cessation.
6. Eliminate or reduce alcohol consumption.
7. Start an exercise plan for losing weight. Recommend starting with about 30 minutes of exercise three times per week.

D. Pharmaceutical therapy: **The mainstay of treatment for TIA is pharmacologic management with antithrombotic agents**:
1. Antiplatelet therapy:
a. Aspirin 50 to 325 mg daily. **Aspirin should not be administered concomitantly with coumarin anticoagulants.**
b. Dipyridamole (Persantine) may be given as an adjunct with warfarin therapy.
c. Aspirin + dipyridamole extended release (Aggrenox) 25/200 mg BID.
d. Clopidogrel (Plavix) 75 mg daily:
i. Aspirin is not routinely recommended with clopidogrel due to the risk of hemorrhage.
ii. No dosage adjustment is necessary with clopidogrel for elderly clients or clients with renal disease.
e. Ticlopidine is no longer available in the United States.

2. Anticoagulation is usually recommended for clients with atrial fibrillation or other cardioembolic sources of TIAs if the client does not have contraindications. The timing of initiation of anticoagulants after an acute stroke should be discussed with the neurologist.
3. Antihypertensive therapy as indicated to maintain blood pressure below 130/80 mmHg with an angiotensin-converting enzyme (ACE) inhibitor or an angiotensin receptor blocker alone or in combination with a diuretic.
4. Initiate a daily statin to a goal low-density lipoprotein-cholesterol (LDL-C) less than 70 mg/dL.

EMERGENT ISSUES/INSTRUCTIONS

A. Rapid transfer is essential for a client with positive symptoms for risk stratification. Clients with a suspected TIA who are not admitted to the hospital should have rapid access (within 12 hours) for an urgent assessment and evaluation with CT or MRI brain scan, EKG, and carotid Doppler testing.

FOLLOW-UP

A. Clients managed as outpatients should be fully educated about the need to return to the clinic or ED immediately if symptoms recur.
B. Specific follow-up depends on etiology, severity, frequency, and duration of TIAs.
C. Follow-up laboratory testing as indicated (e.g., CBC, cholesterol, international normalized ratio).
D. Monitor the client for occult bleeding if started on antiplatelet and/or antithrombotic medications.

CONSULTATION/REFERRAL

A. TIAs should be viewed as a medical emergency because these clients have salvageable neurologic function; consult with a physician.

B. Cardiology and neurology consultations should be obtained when cardioembolic TIAs are treated with anticoagulation therapy.

C. Ophthalmologic consultation is indicated to assess the nature of transient visual symptoms.

D. Vascular surgeon consultation is necessary for clients with significant stenosis or occlusion. Clients with symptomatic carotid artery stenosis should have a surgical evaluation immediately.

E. Hematology consultation is indicated if concern is for underlying hypercoagulable disease.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. TIAs that occur in the younger adult population should be evaluated for embolism.

B. Geriatrics:

1. TIAs are most commonly seen in this population.

2. Screen for AFib in females, especially older than 75 years.

3. Anticoagulation is not recommended for females younger than 65 years with AFib who are otherwise at low risk for stroke. Antiplatelet therapy is recommended for this population.

4. Assess swallowing function of geriatric clients post TIA event, especially for those with cognitive impairment. Implement preventive measures for aspiration pneumonia and choking when eating/drinking and assist clients with communication devices/gestures to promote seamless social engagement during the recovery phase. Constantly assure clients of impending resolution and encourage them to verbalize their feelings regarding the episode (appearance, temporary functional inabilities, fears of strokes/TIA reoccurrence).

RESOURCES

A. The National Institutes of Health Stroke Scale (NIHSS) is a stroke scale that evaluates the effect of an acute cerebral infarct. The 15-item stroke scale requires less than 10 minutes to complete.

1. A free online training certification course for the NIHSS is available at www.nihstrokescale.org.

2. A hard copy of the NIHSS is located at www.ninds.nih.gov/sites/default/files/NIH_Stroke_Scale_Booklet.pdf.

3. An online NIHSS calculator is located at www.mdcalc.com/nih-stroke-scale-score-nihss.

B. The ABCD² Score for TIA estimates the risk of a stroke after a TIA:

1. An online ABCD² calculator is located at www.mdcalc.com/abcd2-score-for-tia.

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TRIGEMINAL NEURALGIA

DEFINITION

A. Trigeminal neuralgia (TN), also known as tic douloureux, is a chronic pain condition of the face. There are two types: type 1 or TN1 and type 2 or TN2:

1. TN1 causes extreme, sporadic, sudden burning or shock-like facial pain that may last from a few seconds to as long as 2 minutes per episode. These attacks can occur in succession lasting as long as 2 hours.

2. TN2 is characterized by constant aching, burning, or stabbing pain of somewhat lower intensity than TN1.

3. Both can occur at the same time, in the same person. The pain can be physically and mentally incapacitating.

INCIDENCE

A. TN occurs most often in people older than the age of 50, although it may occur at any age, including infancy. It is estimated that the incidence of TN roughly affects 4 to 13 people per 100,000 per year.

PATHOGENESIS

A. The trigeminal nerve is the fifth cranial nerve (CN). It is the nerve responsible for providing sensation to the face. One trigeminal nerve runs to the right side of the head, whereas the other runs to the left. Each of these nerves branches off after the nerve leaves the brain and travels inside the skull. It divides into three smaller branches:

1. The first branch controls sensation in the eye, upper eyelid, and forehead.

2. The second branch controls sensation in the lower eyelid, cheek, nostril, upper lip, and upper gum.

3. The third branch controls sensation in the jaw, lower lip, lower gum, and some of the muscles used in chewing.

B. The most common form of TN is caused by neurovascular compression (NVC). Because of this compression, demyelination of nerve fibers occurs, causing them to fire ectopically.

PREDISPOSING FACTORS

A. TN may be caused by a blood vessel pressing on the trigeminal nerve as it exits the brainstem, causing the wearing away of the protective coating of the nerve (myelin sheath).

B. TN may occur in people with multiple sclerosis (MS), a disease that causes deterioration of the trigeminal nerve sheath. The development of TN in a young adult suggests the possibility of MS.

C. Injury to the trigeminal nerve resulting from sinus surgery, oral surgery, stroke, or facial trauma may produce neuropathic facial pain.

D. Hypertension (HTN) is a risk factor for TN.

E. There is evidence that TN runs in families.

F. Rarely, TN may be caused by a tumor or tangle of arteries and veins known as arteriovenous malformation (AVM).

COMMON COMPLAINTS

A. Pain varies depending on the type of TN and may range from sudden, severe, and stabbing pain to a more constant aching and burning sensation. The usual pattern is for the attacks to intensify over time with shorter pain-free periods.

B. Flashes of pain may occur by vibration or contact with the cheek, such as with shaving, washing the face, applying makeup, blowing the nose, brushing teeth, eating, drinking hot or cold beverages, talking, or being exposed to the wind.

C. The pain may affect a small area of the face or it may spread. ▶

D. Bouts of pain rarely occur at night when the client is sleeping.

OTHER SIGNS AND SYMPTOMS

A. Classic pain occurs with definite periods of remission. The pain is intensely sharp, throbbing, and shock-like.
B. Atypical pain often presents as a constant, burning sensation affecting a more widespread area of the face. There may not be remission periods, and symptoms are more difficult to treat.
C. TN tends to run in cycles. Long stretches of frequent attacks will be followed by weeks, months, or even years with little or no pain.

POTENTIAL COMPLICATIONS

A. Uncontrollable facial twitching.
B. Attacks becoming more frequent and intense.
C. The attacks often worsen over time, eventually pain-free intervals disappear, and medication becomes less effective.
D. TN is not fatal, but it can be debilitating, causing clients to avoid daily activities or social contacts in fear of an impending attack.
E. TN may indicate an underlying tumor or diagnosis of MS.

SUBJECTIVE DATA

A. Ask client what activity brought about or preceded the episode.
B. Have client describe duration of pain and what time of day symptoms began.
C. Ask client to describe pain—for example, crushing, stabbing, or burning—and affected areas.
D. Ask the client where sensation began and in what direction it radiates.
E. Have client rate pain on a scale of 0 to 10, with 0 being no pain.
F. Ask client to list all medications currently being taken, particularly substances not prescribed and illicit drugs such as cocaine.
G. Ask the client about any other medical conditions for which they are being treated.
H. Ask the client if they have had sinus or dental surgery or facial trauma/injury.
I. Ask what treatments have been tried so far and if anything helps the pain.
J. Ask how the pain impacts quality of life and ability to perform daily activities.

PHYSICAL EXAMINATION

A. Start with vital signs: Check temperature (if infection is suspected), pulse, respirations, and blood pressure.
B. Inspect:
  1. Inspect general appearance, noting any client discomfort.
  2. Note facial appearance and evidence of tics.
C. Auscultate:
  1. Heart.
  2. Lungs.
D. Palpate:
  1. Complete a full neurologic exam, testing all CNs.

DIAGNOSTIC TEST

A. MRI to rule out tumor or MS.

DIFFERENTIAL DIAGNOSES

A. TN.
B. Brain tumor.
C. MS.
D. Temporal tendinitis (migraine mimic).
E. Temporomandibular joint syndrome (TMJ).
F. Occipital neuralgia.
G. Postherpetic neuralgia.
H. Glossopharyngeal neuralgia.
I. Migraines.

PLAN

A. General interventions:
  1. Order MRI to rule out a tumor or MS as the cause of the pain.
  2. Surgery procedures are available if medications do not control the pain: percutaneous (through the skin) or open. The benefits of surgery should always be weighed carefully against the risk involved. There is no guarantee that surgery will help every individual.
    a. Microvascular decompression (MVD) involves exposure of the trigeminal nerve root with gentle movement of the blood vessel away from the point of compression. MVD is the most invasive surgery for TN, but offers the lowest probability that pain will return. Approximately one-half of individuals post-MVD will experience recurrent pain within 12 to 15 years.
    b. Rhizotomy involves the use of electrocoagulation (heat), destroying the part of the nerve that causes pain and suppressing the pain signal to the brain. There are several types of rhizolysis:
      i. Balloon compression works by injuring the insulation on the nerve that involves the sensation of light touch on the face. A soft catheter with a balloon tip is threaded through a cannula inserted through the cheek through which one branch of the TN passes. The balloon is inflated to compress the nerve and is deflated and removed with the cannula a few minutes later. Pain relief from balloon compression usually lasts 1 to 2 years.
      ii. Glycerol rhizotomy utilizes glycerol injection into the area where the nerve divides into the three branches hoping to damage the nerve, selectively interfering with the pain transmission to the brain. Pain recurrence is generally within a year or two; however, the procedure can be repeated multiple times.
      iii. Stereotactic radiosurgery (SRS; Gamma Knife, Cyber Knife) involves delivering a single highly concentrated dose of radiation to a small target of the trigeminal nerve. This procedure avoids many of the risks and complications of open surgery and other treatments. The slow formation of a lesion in the nerve interrupts transmission of pain signals to the brain. The International Radio Surgery Association reports 50% to 78% who are treated with the Gamma Knife radiosurgery experience pain relief within a few weeks post procedure and almost one-half have recurrence of pain within 3 years.
      iv. Radiofrequency (RF) thermal lesioning (RF ablation or RF lesion) is most often the procedure performed on an outpatient basis under sedation. The procedure involves passing an electrode to the area, causing tingling in the area of the nerve. The nerve area is gradually heated, injuring the nerve fibers. The procedure can be repeated until desired sensory loss is obtained. Approximately half of the ▶

clients will have reoccurrence of symptoms 3 to 4 years post RF lesioning.

v. A neurectomy (partial nerve resection) involves cutting superficial branches of the TN. When performed on the face, the nerve may grow back and sensation may return.

B. Client teaching: *See Client Teaching Guide for this chapter, "Trigeminal Neuralgia."*

1. Provide client with education regarding the disease process.

2. Complementary therapies including low-impact exercise, yoga, aroma therapy, acupuncture, upper cervical chiropractic care, and biofeedback are often used in combination with drug treatment.

C. Pharmaceutical therapy:

1. Anticonvulsant medications such as carbamazepine and oxcarbazepine are the most common medications used to treat TN. In the early stages, these medications control pain for most people, but the effect of the drug decreases over time. Side effects include dizziness, double vision, drowsiness, and nausea. Oxcarbazepine has been used more recently as the first-line treatment and has fewer side effects, notably dizziness and double vision.

2. Muscle relaxants such as baclofen, Robaxin, Norflex, or Flexeril are especially effective when used with either carbamazepine or an anticonvulsant. Possible side effects include confusion, depression, and drowsiness.

3. Anticonvulsant medications such as phenytoin, which was the first medication used to treat TN, with possible side effects of gum overgrowth, balance disturbances, and drowsiness.

4. Other medications include gabapentin, clonazepam, sodium valproate, lamotrigine, and topiramate.

5. Botulinum toxin injections have been used with some success in blocking the sensory activity of the nerves.

FOLLOW-UP

A. Follow-up is determined by client needs, severity of symptoms, and whether complications are present. Monitor the client for depression and sleep disturbance.

B. Anticonvulsant therapies may lose their effectiveness over time, requiring an increase in dosage or adding a second anticonvulsant. Laboratory follow-up is necessary to evaluate bone marrow suppression, and renal and liver toxicity.

CONSULTATION/REFERRAL

A. Neurology referral is essential in confirming the diagnosis of TN.

B. Medication initiation, titration, and management should be instituted by the neurologist.

C. Neurosurgeon referral per neurology recommendations due to medication failure and possible surgery.

D. Some clients may require supportive counseling or therapy by a psychiatrist or psychologist.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Current research postulates that pregabalin (Lyrica) treatment for older adults suffering with TN is effective. The medication improved pain in 48.5% of clients in a clinical trial. Because of pregabalin's effectiveness at regular and low doses in older adults, it has been recommended to prescribe to TN geriatrics preceding surgical intervention.

2. Studies have given evidence to the safety and efficacy of treating TN geriatrics ≥80 years with botulinum toxin type A. The dosages were comparable with treatments given to TN adults younger than 60 years. Side effects were minimal and resolved within 3 weeks. More research is needed.

3. Research examined MVD and SRS in TN clients who were between 62 and 73 years old and compared the long-term pain control of each modality. Older adults who received MVD had longer intervals of pain control than those who elected SRS. These results could assist clinicians on what options to present to geriatrics considering surgery for TN.

4. Geriatric syndromes associated with TN:

a. Confusion related to medication side effects.

b. Depression and sleep disturbance secondary to pain.

c. Falls as a result of dizziness and sedation related to medication side effects.

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VERTIGO

DEFINITION

A. Vertigo is the illusion of self or environmental movement, typically rotating, spinning, tilting, even a sensation that you are going to fall down. Older individuals have an increased risk of falls and depression secondary to vertigo.

B. Vertigo is often classified as either central or peripheral in origin.

INCIDENCE

A. Dizziness and vertigo account for an estimated 4.4 million ED visits in the United States yearly, accounting for 4% of chief symptoms reported in the ED.

B. Strokes are the underlying cause of roughly 3% to 5% of these visits.

C. Both sexes, as well as all age groups, are affected.

D. Benign paroxysmal positional vertigo (BPPV) is the most common cause of vertigo, excluding central nervous system (CNS) lesions:

1. The prevalence of BPPV is 2.9%. The lifetime prevalence increases given suspected degeneration of the otolithic membrane secondary to aging.

2. BPPV rarely occurs in people younger than 35 years unless there is a history of head trauma.

3. BPPV commonly presents in ages between 50 and 70 years.

4. BPPV recurs in approximately one-third of clients after 1 year and in about 50% in all clients treated after 5 years. It is about two to three times more common in females versus males.

PATHOGENESIS

A. Distinguishing between peripheral and central vertigo is critical because the evaluation, treatment, and progress vary significantly. Central vertigo suggests brainstem dysfunction ▶

affecting the vestibular nuclei or their connections. This may be secondary to a structural lesion such as neoplasm or ischemia.

B. Vertigo due to vascular insufficiency is rarely isolated, and other symptoms of brainstem involvement are usually seen, such as diplopia, dysphagia, motor weakness, or disruption in sensation. Neoplasms are usually slow-growing, and the vestibular dysfunction is often insidious. Central vertigo secondary to brainstem or cerebellar ischemia usually lasts for 20 minutes to 24 hours. The client may be unable to ambulate during acute attacks of vertigo:

1. Other considerations for causes of central vertigo include multiple sclerosis (MS), seizures, and migraines. About 20% of clients with migraines experience vertigo, motion sickness, and mild hearing loss.

2. Progressive gait difficulty, memory loss, and urinary incontinence are commonly due to normal pressure hydrocephalus (NPH). Refer to the section "Normal Pressure Hydrocephalus" earlier in this guideline for further discussion.

C. Vertigo of peripheral origin is more common and may be caused by dysfunction of the inner ear or vestibular nerve. BPPV is the most commonly diagnosed of peripheral vestibular disorders. The cause of BPPV is unknown. The most common explanation is free otoconia within the semicircular canals that are dislodged by trauma, infection, or degeneration. The debris relocates when the head is repositioned and provokes vertigo. Causes of labyrinthine dysfunction include infection, trauma, ischemia, or toxins such as drugs or alcohol.

D. Ménière disease causes vertigo, hearing loss, and ringing of the ears. The exact cause is unknown, but a hypothesis is a buildup of fluid in the inner ear.

E. Viral infections may lead up to vestibular neuritis (labyrinthitis). The vertigo experienced with vestibular neuritis is sudden and severe and may last days.

F. Other possible causes of vertigo are psychogenic, cardiovascular, or metabolic conditions, as well as head trauma and migraines.

G. Medications that cause dizziness:

1. Anticonvulsants.
2. Antidepressants.
3. Antipsychotics.
4. Anxiolytic/sedatives.
5. Antihypertensives.
6. Nitrates.
7. Diuretics.
8. Insulin/oral hypoglycemic agents.

PREDISPOSING FACTORS

A. Head or body movement:

1. Rolling over in bed.
2. Getting out of bed.
3. Bending down from the waist.
4. Looking up.

B. Fear or anxiety.
C. Stress.
D. Recent infection, usually upper respiratory in cases of vestibular neuronitis.
E. Family history, especially in cases of vertiginous migraine.
F. Head trauma.
G. Migraines.
H. Idiopathic with no cause identified.
I. Hypoglycemia.
J. Alcohol intoxication.
K. Medication side effects.
L. Cerebellar or brainstem stroke.
M. Tumors.
N. MS.
O. Dehydration.

COMMON COMPLAINTS

A. Dizziness with or without change in body positioning.
B. Feeling of imbalance.
C. Nausea/vomiting.
D. Tinnitus.
E. Aural fullness.
F. Hearing loss.

OTHER SIGNS AND SYMPTOMS

A. Central origin, including vascular insufficiencies, strokes, neoplasms, migraine, MS, seizures:

1. Double vision.
2. Dysarthria.
3. Dysphagia.
4. Paresthesias.
5. Changes in motor or sensory exam.
6. Mild to moderate vertigo.
7. Multiple episodes of vertigo lasting seconds to minutes in duration with vascular insufficiency and seizures.
8. Constant complaints of vertigo with neoplasms or strokes.
9. Multiple episodes of vertigo lasting hours with migraines.
10. Single episodes of vertigo with MS.
11. Dix–Hallpike test: habituation common with delayed nystagmus.

B. Peripheral origin, including BPPV, Ménière disease, labyrinthitis, vestibular dysfunction, vestibular neuritis, and acoustic neuroma:

1. No associated signs of brainstem dysfunction.
2. Vertigo, usually described as severe.
3. Multiple episodes of vertigo lasting hours with Ménière disease.
4. Single or multiple episodes of vertigo with labyrinthitis.
5. Vestibular dysfunction, described as constant vertigo.
6. Severe nausea or vomiting.
7. Hearing loss or tinnitus; aural fullness may be present, as well as a roaring sound.
8. Triad of vertigo, tinnitus, and hearing loss is suggestive of Ménière disease.
9. Dix–Hallpike test: no habituation: nystagmus occurs immediately.

SUBJECTIVE DATA

A. Elicit onset, frequency, duration, and course of presenting symptoms:

1. Is this recurrent or new?

a. Acute vertigo is seen with trauma, stroke, meningitis, otitis media, mastoiditis, drug use, vestibular neuronitis, MS, and labyrinthitis.

b. Recurrent vertigo is seen in vestibular migraines, BPPV, motion sickness, seizures, and Ménière disease.

B. Elicit from the client a verbal description of the sensation(s) experienced. Ask the client to describe their symptoms by using words other than "dizzy." The word *dizzy* is nonspecific to describe vertigo, unsteadiness, generalized weakness, syncope, presyncope, or falling. Descriptions may include light-headedness, unsteadiness, motion imbalance, floating, or a tilting sensation. ▶

C. Note triggering and alleviating factors.
D. Query the client regarding associated symptoms such as hearing loss, tinnitus, nausea, difficulty with gait, aural fullness, or other neurologic manifestations such as nystagmus. As a result of dizziness, ask the client if they have ever fallen down when symptoms are present.
E. Review the client's medical history, including recent infections, trauma, and risk factors for cardiovascular disease, such as smoking, diabetes, and hyperlipidemia.
F. Evaluate alcohol intake.
G. Evaluate any recent change in the dosage and polypharmacy issues (number of prescriptions [>4 medications]). Review over-the-counter (OTC), herbal products, recreational drugs, and medication use: aminoglycoside, antibiotics, diuretics, antihypertensives, and antidepressants.
H. Has the client had any previous treatments for vertigo, such as an Epley procedure?
I. Has the client had any previous testing such as:
1. Audiometric testing.
2. Electronystagmogram (ENG)/videonystagmography (VNG) to evaluate balance.
3. Rotational/balance platform test.
4. CT or MRI.
5. Computerized dynamic posturography (CDP) to evaluate postural stability/motor control.

PHYSICAL EXAMINATION

A. Check temperature (if infection is suspected), pulse, respirations, and supine and standing blood pressures; note orthostatic hypotension.
B. Inspect:
1. Observe overall appearance. Generalized muscle weakness may be observed.
2. Note gait: difficulty with tandem gait. Note global weakness.
3. Inspect the eyes: Assess for nystagmus; a few beats of nystagmus on extreme lateral gaze may be normal.
4. Ear examination: Rule out otitis media. Whisper and ask the client to repeat heard words.
5. Evaluate for aphasia that may indicate a stroke.
C. Palpate:
1. Palpate extremities; note pulses and edema.
2. Perform Rinne and Weber tests.
D. Neurologic exam:
1. Perform complete neurologic exam.
2. Assess cranial nerves.
a. **Brainstem involvement is frequently seen with detailed neurologic exam. Signs of cerebellar dysfunction include difficulty with finger-to-nose testing, rapid alternating supination or pronation of hands, and gait disturbance.**
3. Perform Romberg test: The client stands with feet together and closes his or her eyes. Positive result is when the client sways. This may be seen with vestibular disease and acoustic neuroma.
4. The Dix–Hallpike test (also called Nylen–Barany maneuver test) is a provocative positional test:

The Dix–Hallpike maneuver is considered the gold standard for diagnosing BPPV. However, a negative test does not rule out BPPV if the client is asymptomatic on the date of the test. If a positive response is observed on the initial side, no further testing is required.

a. Perform the Dix–Hallpike: While the client is seated on the middle third of the examination table, turn the client's head 45° toward the affected side (problem ear). While holding the head in that position, assist the client to the reclining position past the supine position. BPPV has a distinctive nystagmus in which there is involuntary eye movement (predominately in a rotating fashion) starting slowly, progressing to a fast phase, and then reaching a resetting phase. The nystagmus generally lasts less than 20 seconds and reverses itself once the client sits upright:
i. Caution should be taken in performing the Dix–Hallpike test with clients who have cervical stenosis, severe kyphoscoliosis, spinal cord injuries, and significant vascular disease such as vertebral artery stenosis.
5. Test for nuchal rigidity if fever is present.
E. Auscultate:
1. Auscultate the heart, neck, and carotid arteries. **Physical examination may reveal cardiovascular abnormalities, such as a carotid bruit.**
2. Auscultate lungs: Pneumonia may cause dizziness.
3. Auscultate the abdomen.

DIAGNOSTIC TESTS

A. Laboratory testing:
1. Thyroid function studies: to rule out hypothyroidism.
2. Venereal Disease Research Laboratory (VDRL) to rule out secondary or early tertiary syphilis, which can have symptoms similar to those of Ménière disease.
3. Complete blood count (CBC) to rule out infection or severe anemia.
4. Electrolytes to rule out hyponatremia, hypokalemia, and dehydration.
5. Urine drug screen (if indicated).
6. Cardiac panel (if indicated).
7. Urinalysis to rule out a urinary tract infection (UTI) in the elderly.
B. CT scan for head trauma.
C. MRI with and without contrast to assess for mass, especially if a central origin is suspected.
D. Bithermal caloric test: definitive procedure for identifying vestibular pathology. Cold irrigation is an inhibitory stimulus and warm irrigation is excitatory. The three most important findings from the caloric tests are unilateral weakness, bilateral weakness (peripheral vestibular disease), and failure of fixation suppression (FFS) of caloric-induced nystagmus (central cerebellar disease).
E. Electronystagmography: most useful in chronic peripheral disorders to determine the degree and progression of vestibular deficit.
F. Audiogram: test for possible hearing loss.
G. Rotating chair test: interprets the slow component velocity of the nystagmus response with bilateral canals stimulated.
H. Lumbar puncture (LP) if meningitis is suspected.

DIFFERENTIAL DIAGNOSES

A. Vertigo.
B. Vascular insufficiencies.
C. Stroke.
D. Neoplasms.
E. Migraine.
F. MS.
G. Seizures.
H. BPPV.
I. Ménière disease.
J. Labyrinthitis.
K. Vestibular dysfunction.

L. Vestibular neuritis.
M. Acoustic neuroma.
N. Syncope/orthostatic hypotension.
O. Multiple sensory defects.
P. Parkinson disease (PD).
Q. Adverse reaction to medications.

PLAN

A. General interventions: Treatment of vertigo depends on the underlying pathology and duration of the symptoms:

1. Acute vertigo: Maintain the client on bed rest, with the reassurance that most clients with acute vertigo recover spontaneously over a period of several weeks to months.

2. Chronic vertigo: Refer the client for physical therapy with emphasis on vestibular rehabilitation:

a. The Cawthorne Cooksey physical exercise regimen encourages eye, head, and body movements to facilitate recalibration of the vestibuloocular and vestibulospinal reflexes.

b. Encourage ambulation when tolerated to induce central compensatory mechanism.

3. Ménière disease: The client needs bed-rest in the acute phase and nutritional therapy with restrictions of sodium, caffeine, alcohol, and tobacco.

4. BPPV:

a. Clients need bed rest for acute symptoms.

b. Canalith repositioning (Epley) procedures (CRP) provide immediate resolution of vertigo in 85% to 95% of clients. The CRP is safe, simple, inexpensive, quick, and easy to perform. It is likely to be unsuccessful in clients with bilateral positional nystagmus, and it is not recommended for clients with acute vertigo, many of whom may have vestibular neuronitis. See Section II: Procedure, "Canalith Repositioning (Epley) Procedure for Vertigo." Contraindications to performing the Epley procedure are the following:

i. Recent neck fracture or neck instability.
ii. A history of unstable carotid disease.
iii. Recent retinal detachment.
iv. Any physical condition that prevents the client from lying down quickly and rolling over, which are required for the procedure.

c. Instead, meclizine is used for 1 to 2 weeks, and then the client is reassessed. Stop meclizine on the day the client returns; it may suppress the positional nystagmus.

d. For clientswith severe positional vertigo during the Dix–Hallpike maneuver, premedicate with a prochlorperazine (Compazine) 25 mg suppository 1 hour prior to performance of the CRP.

e. If the Dix–Hallpike is positive on the left, use a left-sided CRP. Conversely, if it is positive on the right, use a right-sided CRP. If the client has bilateral disease, refer them to an otolaryngologist or treat the more symptomatic side first.

f. After the Epley procedure, different types of recommendations are made to prevent the otoconia from returning to the posterior semicircular canal, including the following:

i. Wear a cervical collar for two nights after the maneuver.
ii. Stay upright for 24 hours after the procedure or have the head elevated 30° for one to two nights after the procedure.
iii. Avoid sleeping with the affected ear down.
iv. Counsel to avoid abrupt head changes for 1 week after the procedure.
v. Avoid exercise such as yoga and sit-ups that would make similar motions.
vi. Avoid looking up, such as looking at items on the top shelves at the grocery.
vii. Symptoms may reoccur after tilting backward in dental chairs.
viii. Symptoms may reoccur after turning in the hairdresser's chair and/or tilting backward for a shampoo.

B. Client teaching: *See Client Teaching Guide in Chapter 3, "Safety Issues: Fall Prevention."*

1. Encourage compliance with bed rest and exercises.

C. Pharmaceutical therapy:

1. The American Academy of Otolaryngology—Head and Neck Surgery does not recommend the use of vestibular suppressant medications to control BPPV. The American Academy of Neurology also reports that there is no evidence to support the routine use of vestibular suppressant therapy as treatment for BPPV.

2. Antihistamines prevent the histamine response in sensory nerve endings and blood vessels that are affected in treating vertigo:

a. Meclizine (Antivert).
b. Dimenhydrinate (Dramamine).

3. Benzodiazepines:

a. Diazepam (Valium, Diastat).

4. Phenothiazine derivative to treat emesis:

a. Promethazine (Phenergan, Promethegan).
b. Prochlorperazine (Compro).

5. Anticholinergic agents:

a. Glycopyrrolate (Robinul, Cuvposa).
b. Scopolamine (Transdermal).

6. Antivirals are not useful in the treatment of vestibular neuritis.

7. Steroids have been used in the treatment of vestibular neuritis.

8. Vestibular migraines respond to antimigraine medications.

D. Surgery:

1. Pneumatic equalization tubes.

2. BPPV surgery: canal partitioning or canal plugging.

3. Vestibular nerve section (vestibular neurectomy) is a treatment for intractable violent episodes of BPPV.

4. Labyrinthectomy to remove the semicircular canals, utricle, and saccule, the balance organs. This procedure is considered only when a person has already lost all hearing functions in the affected ear.

5. Chemical labyrinthectomy: Gentamicin infusion destroys the vestibular hair cells.

FOLLOW-UP

A. The American Academy of Otolaryngology—Head and Neck Surgery Foundation recommends managing clients with BPPV as follows:

1. Canalith repositioning/Epley procedure should be offered unless there is a risk for impaired mobility or balance or the client is at increased risk for falls.

2. Clients should be reevaluated in 1 month after an Epley procedure/CRP to confirm that the procedure resolved the symptoms of vertigo.

3. The recurrence rate after an Epley procedure is 30% to 50%.

B. Follow up as needed according to the origin of the diagnosis and the client's needs.

C. If indicated, transfer to the ED/call 911 for cardiovascular and cerebrovascular symptoms for treatment of stroke or cardiac events.

CONSULTATION/REFERRAL

A. Refer to a physician if the client does not experience improvement in 2 to 4 weeks. If symptoms worsen, consider referring to an ear, nose, and throat specialist.

B. Refer to an otolaryngologist for testing, including:

1. Audiometric testing.

2. ENG to evaluate balance.

3. Rotational/balance platform test.

C. Occupational and physical therapists are helpful in addressing home safety and providing a structured balance rehabilitation program.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Normal aging affects degeneration of neural structures that manage balance performance and equilibrium. Disequilibrium is one of the most frequent complaints from geriatrics to their healthcare provider, and vestibular disorders account for 48% of dizziness reported.

2. Possible etiologies for vertigo in the elderly are:

a. CNS: vestibular neuritis, late-onset Meniere disease, transient ischemic attack (TIA)/stroke, vestibular migraine.

b. Cardiovascular: arrhythmia, congestive heart failure (CHF), heart valve disease, postural hypotension, occluded anterior vestibular artery.

c. Age-related disorders: presbyastasis, labyrinthitis, breast/prostate neoplasia, musculoskeletal disorders, somatosensory and proprioception losses.

3. Vestibular abnormalities may mimic stroke/TIA symptoms and must be further explored to determine appropriate treatment. Vertigo and balance dysfunctions have improved with pharmacologic treatments and physical therapy.

4. For older adults with vestibular symptoms, nurse practitioners (NPs) should practice special considerations of the following geriatric syndromes:

a. Falls: Consistently assess for falls as part of routine primary care visits:

i. Refer to section "Fall Assessment" in Chapter 3, "Adult-Geriatric Assessments."

ii. Safety measures to reduce falls in the home include removing small area carpets, keeping night lights along the path to the bathroom, and installing a toilet seat or nearby grab bars. An in-depth safety review is noted in the client teaching: *See Client Teaching Guide for Chapter 3, "Safety Issues: Fall Prevention."*

iii. Evaluate need for assistive device.

b. Depression is increased in clients with symptoms of disequilibrium. The fear of falling may cause a person to cut down on their activities of daily living and become sedentary, which in turn increases weakness and risk of falls.

5. Beers Criteria caution: Many of the medications used for vestibular suppression, individually and especially if prescribed in combination with the client's multiple other prescriptions, are potentially inappropriate medications for older adults:

a. Medications include antihypertensives, benzodiazepines, antiepileptics, and hypnotics/anxiolytics.

b. Review all medications for side effect profiles and consider deprescribing.

RESOURCES

American Speech-Language-Hearing Association (ASHA): www.asha.org
Vestibular Disorders Association: www.vestibular.org

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Saber Tehrani, A. S., Kattah, J. C., Kerber, K. A., Gold, D. R., Zee, D. S., Urrutia, V. C., & Newman-Toker, D. E. (2018). Diagnosing stroke in acute dizziness and vertigo: Pitfalls and pearls. *Stroke*, *49*(3), 788–795. https://doi.org/10.1161/STROKEAHA.117.016979

CLIENT TEACHING GUIDE

BELL'S PALSY

PROBLEM Bell's palsy is a disorder that can occur at any age, but most frequently occurs between the ages of 20 and 60. The disorder affects the muscles associated with expression on one side of the face, including the muscles that allow the mouth to smile, the eyes to close, and the eyebrows to rise.

The exact cause of Bell's palsy remains unknown. Possible causes include viral infections, a type of inflammatory process, or an autoimmune disease.

PREVENTION/CARE

A. Bell's palsy is usually treated with a steroid, such as prednisone.

B. Other medications, such as acyclovir, may be used to possibly shorten the course of the symptoms. It is believed that Bell's palsy may be linked to viral infections, such as the herpes infection.

C. Pain is usually managed with acetaminophen (Tylenol) or another over-the-counter pain medication such as nonprescription anti-inflammatory medications (NSAIDs]), such as ibuprofen or naproxen.

D. Sometimes it is helpful to use gentle massage or electrical stimulation of the nerve to help with the pain. Applying heat or cold packs for 15 to 20 minutes three to four times a day may also help with pain. When applying ice packs, do not directly apply ice to the skin, and use caution to avoid frostbite to the area.

E. Protection of the eye is very important if there is loss of lid function. Eye drops and lubricating ointment may be recommended, along with taping the affected eye while sleeping. Wearing eyeglasses and/or sunglasses is recommended to protect the eye.

F. Physical therapy may also be helpful with recovering function of the muscles that are weak.

G. Symptoms usually resolve within 3 or 4 weeks to a few months; occasionally, clients have symptoms lasting longer. The degree of paralysis varies with each person. If symptoms change or worsen, notify your healthcare provider immediately.

Activity: Engage in activities as tolerated. Use caution when performing activities with visual demands, such as depth perception (driving, walking, etc.).

Diet: Eat a regular diet as tolerated.

Medications:_____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If You Have:

A. No relief in symptoms in 4 weeks.

B. New symptoms such as headaches, visual changes, or other problems, such as trouble walking.

C. Other: _____

Phone: _____

RESOURCE The Bell's Palsy Network: www.bellspalsy.ws

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CLIENT TEACHING GUIDE

DEMENTIA

PROBLEM Dementia is a progressive mental impairment that can lead to memory changes and loss, altered thinking processes, and behavior changes.

CAUSES Dementia is caused by degeneration and loss of the gray matter from the brain; common causes are Alzheimer disease, inadequate blood supply to the brain, alcoholism, chronic infections, inherited conditions, brain injury, or brain tumors.

PREVENTION Early medical treatment is required for reversible causes of dementia. Prevention includes protection from head injury, eating a balanced diet, preventing alcoholism, avoiding drug abuse, and preventing atherosclerosis.

TREATMENT PLAN

A. Minimize changes in daily routines.
B. Provide simple memory reminders such as notes, calendars, and clocks.
C. Encourage social contacts: Participate in local support groups and share stories.
D. Treat with respect.
E. Provide a safe environment:
  1. Remove scatter rugs.
  2. Install handrails and stairs.
  3. Discourage driving.
  4. Install stove cutoff switch.
  5. Lock closets.
  6. Lock up matches and firearms.
F. Encourage "thinking" games such as puzzles, word games, and reading, which stimulate the brain and assist with memory.
G. Provide frequent gentle reorientation to the surroundings; orient yourself daily to your environment and belongings.
H. Reduce stress. Less stress will improve overall concentration, decision-making ability, and your quality of life:
  1. Remove yourself from stressful situations.
  2. Establish boundaries and share these with your family.
  3. Reduce stimulation in the environment. Focus on relaxation and avoid stressful situations.
I. Discuss financial plans and organize documents. Include family members whom you trust and discuss potential care costs and planning for financial concerns for the future.

Activity: Client may engage in as much activity as possible with *supervision and direction*.

Diet: Eat a well-balanced diet low in saturated fat.

Medications: A variety of medications are available to treat symptoms.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. The symptoms/behaviors are getting worse.
B. You are unable to tolerate the medications due to side effects.
C. You need help with social services.
D. Other: _____

Phone: _____

RESOURCE Alzheimer's Association: www.alz.org

The Alzheimer's Association's website includes information on locating the closest support group; search by zip code: www.alz.org/Local_Resources/Find_Your_Local_Chapter

24/7 Helpline: 1-800-272-3900

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CLIENT TEACHING GUIDE

MANAGING YOUR PARKINSON DISEASE

PROBLEM Parkinson disease (PD) is a problem with the central nervous system that causes progressive muscle rigidity and tremors. The ability to move your muscles becomes difficult and you may also notice difficulty with walking and swallowing. Common symptoms you may experience include tremor at rest, rigidity (feeling stiff and finding it hard to start moving), bradykinesia (movements of your muscles slow down), having a difficult time maintaining your posture, and feeling like you are going to fall down. Other symptoms may include difficulty speaking and swallowing, drooling more, and changes in mood and sleep patterns.

CAUSE The brain is not able to produce or use the correct amount of a chemical called dopamine required by the body, and therefore the nervous system reacts by producing a loss in control of your muscles and movements.

TREATMENT PLAN/CARE

A. There is no cure for PD. However, medications may relieve a lot of your symptoms. Your healthcare provider will share the medications that are available to you.

B. People with PD can be very sensitive to heat. During hot weather, stay outside for very short periods of time, stay inside during the hottest part of the day, and increase your fluid intake.

C. Balance problems are common with PD and increase the risk of falls. Move slowly when changing positions. Avoid turning around in one spot. Make wide turns with change of direction. When moving from one position to another, rise slowly and take your time to prevent falls. Some ways to avoid injury are no loose rugs or other floor coverings; grab bars around the tub and toilet; a sturdy rail on stairs; adequate lighting so you can see where you are going; and straight-backed, firm chairs with arms.

D. Wear comfortable shoes that fit correctly. Lift your feet when walking and avoid shuffling to avoid falls. Use your arms to assist you in walking by swinging your arms in a natural fashion.

E. Tremor increases the risk of accidents. Use sturdy plastic cups instead of glassware, use an electric razor to shave, and be cautious with sharp objects and power tools.

F. For clothes, Velcro fasteners, zippers, and snaps are easier to fasten than buttons. Loose clothing is also easier to put on and take off.

G. To avoid sleep problems, stay busy during the day and avoid naps. Discuss any problems you have with your neurologist; sometimes changes in your medication schedule can help.

H. If you have speech problems, work on ways to make your needs known. Practice speech exercises or consider speech therapy with a speech therapist.

I. Stay active in your daily work, hobbies, and other daily activities you enjoy.

J. If you are having signs of anxiety, depression, sleep problems, or other symptoms, please discuss these with your provider. These symptoms are common and can be addressed and treated with other medications.

Activity: Regular exercise helps maintain muscle flexibility and may reduce medication needs. Exercises to improve face, jaw, and tongue movement are encouraged.

Diet: Your diet should include plenty of fluids and adequate fiber to prevent or manage constipation. Have bran cereal in the morning and eat five or more servings of fruits and vegetables throughout the day. Bananas are low in fiber and should be avoided.

Plan medication schedules so that you are functioning well at mealtimes. Be sure you allow plenty of time to finish your meal.

If you need help planning your meals, your healthcare provider can suggest a dietitian with whom you can talk about food choices.

Alcoholic beverages are discouraged, as alcohol can interfere with the effectiveness of your medications.

If you are having difficulty with swallowing, let your provider know. Take your time with eating meals. Sit upright. Thick liquids are easier to swallow than thin liquids.

Medications:

A. The medicines you are taking can improve your ability to carry out everyday activities, but they cannot totally eliminate your symptoms. It is important that you know why you are taking each medication as well as the possible side effects of each.

(*continued*)

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CLIENT TEACHING GUIDE

MANAGING YOUR PARKINSON DISEASE (*CONTINUED*)

B. Check with your neurologist before starting any new medicine to be sure it does not interact with your PD medicines. Even vitamins can be a problem, so take only those recommended by your neurologist.
C. In case of emergency, keep with you a list of your medicines, including the amounts you take and your schedule for taking them.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. You have a reaction to your medication.
B. You are unable to tolerate your medication due to side effects.
C. Your symptoms become worse.
D. Other: _____

Phone: _____

RESOURCES Michael J. Fox Foundation for Parkinson's Research: Grand Central Station, PO Box 4777, New York, NY 10163-4777, www.michaeljfox.org

National Parkinson Foundation: 200 SE 1st Street Suite 800, Miami, Florida 33131, 1-800-473-4636, www.parkinson.org

Parkinson's Disease Foundation: 1359 Broadway, Suite 1509, New York, NY 10018, 212-923-4700, www.pdf.org

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CLIENT TEACHING GUIDE

MIGRAINE HEADACHES

PROBLEM You have been diagnosed with migraine headaches. There are many ways that a migraine headache is described, including pulsating on one side of the head or behind one eye, seeing spots before your eyes, and a possible loss of vision for a short time.

Other symptoms of a migraine include nausea and vomiting; sensitivity to sound; sensitivity to light, especially to flickering lights; sensitivity to smells; and pain that gets worse with activity.

CAUSES

A. Migraines run in families; your mother or sister may also have migraines.
B. Head trauma may trigger headaches.
C. You may have a migraine headache when you start your period due to change in your hormones.
D. Medicines such as birth control pills and hormones may cause migraines.
E. Many odors, such as perfumes, cigarette smoke, scented candles, and food, may trigger a headache.
F. Changes in weather and high altitude may also make you have a migraine.
G. Alcohol and some foods can trigger a headache.
H. Another cause of a migraine headache is a rebound from taking too many over-the-counter pain medications, such as ibuprofen, Aleve, aspirin, and Excedrin products. The headache gets better with the pain products but comes back the next day so that you take the medicine again, causing a vicious cycle.

PREVENTION

A. Keep a diary to identify food triggers to your headache:
  1. Alcohol, especially red wine.
  2. Foods such as hot dogs, ham, and bacon.
  3. Aged cheese.
  4. Citrus foods such as oranges, lemons, limes, and tomatoes.
  5. Not eating.
B. Read labels for the following preservatives:
  1. Aspartame: often found in yogurt.
  2. Saccharin: often found in diet drinks and artificial sweeteners.
  3. Tyramine: found in aged cheese such as feta, blue cheese, pickles, and olives.
  4. Phenylethylamine: often found in sugarless gum and breath mints.
  5. Monosodium glutamate (MSG): often found in Chinese food.
  6. Nitrates and nitrites: found in cured meats, such as ham and hot dogs.
C. Keep a diary to identify visual triggers:
  1. Strobe lights.
  2. Flickering light from going from sun into shade.
  3. Fluorescent lights.
  4. Sunlight glare off shiny objects and water.
D. Keep a diary to identify other triggers, including the following:
  1. Too much sleep/too little sleep.
  2. Strong odors.
  3. Medicines.
  4. Changes in weather.

TREATMENT PLAN

A. One of the first things to look at is your diary of triggers in order to avoid or modify them.
B. You may be sent to a neurologist to evaluate if you need any special testing.
C. You may be sent to have your eyes examined for glasses.

(*continued*)

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CLIENT TEACHING GUIDE

MIGRAINE HEADACHES (*CONTINUED*)

D. Many medications are used to treat migraines:
1. Over-the-counter pain medications, including ibuprofen, Aleve, aspirin, and Excedrin in limited amounts.
2. Medications for depression are commonly used.
3. Medications for seizures are also commonly used.
4. Medications for blood pressure have been found to control migraines.
5. Medications for nausea and vomiting.

E. A special class of drugs called triptans is used to treat the migraine when it first starts. These medicines come in pill form and nasal spray, and are also given as a shot.

Activity: Relaxation training, such as yoga and biofeedback, helps headaches. Physical therapy and hypnosis have also helped migraine headaches.

Diet: There is no special diet for migraines.
A. Avoid the food triggers that you identified from your migraine diary.
B. Drink plenty of liquids.

You Have Been Prescribed the Following Medication for Acute Pain to Help Stop Migraines: _____

You Need to Take: _____

You Have Been Prescribed the Following Medication to Take Every Day to Manage Migraines: _____

You Need to Take: _____

You Have Been Prescribed the Following Medication for Nausea: _____

You Need to Take: _____

You Need to Notify the Office If:
A. You feel that this is the "worst headache you have ever had."
B. You have difficulty with speech.
C. You have fever with a stiff neck.
D. Other: _____

Phone: _____

RESOURCES Mayo Clinic: www.mayoclinic.com/health/migraine-headache/DS00120

Migraine diaries are available for iPhones, iPod touch, and the iPad on iTunes.

The Migraine Trust migraine diaries: https://www.migrainetrust.org/wp-content/uploads/2015/11/FS05aMigraineDiaries.pdf

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CLIENT TEACHING GUIDE

MILD HEAD INJURY

PROBLEM Mild head injury includes any bump, jolt, or blow to the head that affects normal brain function, possibly causing confusion or a loss of consciousness.

GUIDELINES FOR CARE AT HOME

Activity:

A. Have someone stay with you for the next 24 hours to be sure your injury does not worsen.
B. Limit physical activity, especially sports or heavy lifting.
C. Slowly increase your activities. Pace yourself, using rest periods whenever you are mentally or physically tired.
D. Do not drive or operate heavy machinery during the next 24 hours.
E. You must have approval from your primary care provider before returning to work or school.
F. Coaches or trainers must be notified of any head injury.

Diet: Do not eat or drink if you feel sick to your stomach. When you feel better, you can eat or drink anything except alcohol until cleared by your primary care provider.

Medications: You should not take any medications, including those regularly taken, unless approved by your primary care provider.

INSTRUCTIONS FOR CAREGIVERS: OBSERVATION AFTER HEAD INJURY

Check the client every 2 hours for the first 24 hours after discharge. If they are asleep, awaken them for testing.

HOW TO TEST THE CLIENT

A. Test orientation: Ask the client to tell you their name, where they are, the year, and who you are.
B. Test the client's strength: Ask the client to squeeze one or two of your fingers as hard as they can, checking both hands at the same time, and compare them. Also have the client lift one leg at a time off the bed and hold it up for a few seconds.
C. Have the client open both eyes at the same time and check their pupils to make sure they are the same size. (The pupil is the black circular part in the center of the eye.)
D. If you are uncertain about these test results, repeat them every 5 minutes until you are sure of what you see.

EMERGENT ISSUES/INSTRUCTIONS

Call 911 or go to the ED if:

A. The client cannot be easily awakened or sleeps constantly in between tests for no apparent reason.
B. The client cannot answer orientation questions correctly or appears to be confused.
C. The client has new or increasing weakness in any arm or leg.
D. The client becomes very restless, agitated, is acting unusual, or has any other change in behavior.
E. One pupil is larger than the other.
F. You cannot understand what the client is saying, or the client has difficulty understanding what you have said.
G. The client complains of a severe or worsening headache.
H. The client continues to complain of nausea or begins vomiting.
I. The client has a fever over 101.4°F.
J. The client has a convulsion or seizure, or passes out.
K. The client has fluid leaking from the nose or ear.

You need to return to the office the day after the head injury.

Phone: _____

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CLIENT TEACHING GUIDE

MYASTHENIA GRAVIS

PROBLEM Myasthenia gravis (MG) is a chronic disorder in which the body's immune system mistakenly attacks and destroys proteins that help muscles respond to nerve impulses. This causes people to have muscle weakness that is worse with use and better with rest.

Eye muscles and muscles that help with chewing, swallowing, and talking tend to be affected the most. MG can be treated but not cured.

People with MG generally have to take medicine for the rest of their lives to control the disorder. Surgery to remove their thymus gland may be necessary.

TREATMENT PLAN/CARE

A. Wear a medical alert tag and keep a list of medicines and the dosing schedule, along with the name and telephone number of your neurologist, in case of emergency.

B. Dentists and any other healthcare providers should know that you have MG because many medicines can make your MG worse. Some examples of medicines that can be problems for people with MG are birth control pills, some antibiotics, and some local anesthetics. Many medicines can be problematic, so you should *never* start a new medicine without talking to your neurologist first, even if you took it in the past without having problems.

C. Take your time when eating. Establish routines and take small breaks between eating. Small frequent meals throughout the day are advised instead of large meals. Choose foods that are soft and easier to chew and avoid foods that require a lot of chewing.

D. Any time you feel worse, particularly if you are having trouble with chewing and swallowing, you should contact your neurologist and plan to go to the hospital, because MG could possibly affect your breathing. You should have a plan for emergencies that includes how you will get to a hospital, childcare arrangements, and how to contact your neurologist.

E. An annual flu shot is recommended because infections can make MG worse.

F. Emotional upset and stress can also make MG worse. Your healthcare provider can supply you with information on handling stress effectively or assist in referring you to a counselor.

G. Surgery can make MG worse, so it should, when possible, be planned with your neurologist. Some changes in your medication schedules in the weeks before the surgery can make problems less likely.

H. Plasmapheresis may be recommended to you. This procedure is performed to remove the antibodies that make the disease process worse. This procedure is similar to donating blood. Blood is removed from a vein, the antibodies are removed, and then the blood is donated back to you in another vein. This procedure may be offered to you many times, since the effects are not permanent.

Activity: It is best to plan your activities to take advantage of the peak effects of your medicine and to avoid getting overtired.

Diet: A diet high in potassium-rich foods has been found by some to be helpful because low body potassium is associated with muscle weakness.

Medications: _____

You may be prescribed steroids or medications that improve muscle strength.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:

A. You have worsening or new symptoms.

B. You are unable to tolerate your medicines due to side effects.

C. Other: _____

Phone: _____

RESOURCE Myasthenia Gravis Foundation of America, Inc.: www.myasthenia.org

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CLIENT TEACHING GUIDE

TRANSIENT ISCHEMIC ATTACK

PROBLEM/CAUSE A transient ischemic attack (TIA) is a temporary loss of brain function caused by a decrease of blood flow to the brain. It is considered a "mini stroke." The symptoms may last only a few minutes, but the risk for a stroke is increased over the next week.

Neither you nor your family should drive you to the hospital ED. An ambulance is needed for the earliest evaluation and treatment.

RISK FACTORS FOR TIAS

A. High blood pressure.
B. Irregular heartbeat (atrial fibrillation).
C. Smoking, including secondhand smoke.
D. High cholesterol.
E. Being overweight.
F. Diabetes.
G. Obstructive sleep apnea.
H. Lack of exercise.

SUDDEN SIGNS AND SYMPTOMS OF A STROKE

F.A.S.T. is an easy way to remember:
F. FACE DROOPING.
A. ARM WEAKNESS.
S. SPEECH DIFFICULTY.
T. TIME TO CALL 911.

PREVENTION/CARE

A. A full workup is necessary to decide why you are having TIAs.
B. You may be referred to a neurologist—a doctor who specializes in medical problems involving the brain.
C. Special tests or surgery may be done to evaluate your heart and blood vessels. Treatment depends on your symptoms and test results. Surgery may be necessary.
D. Treatment starts with modifying your risk factors:
  1. Lose weight.
  2. Eat a healthy diet including fruits/vegetables each day of the week. Eat lean meats/fish most days. Limit your intake of sweets.
  3. Limit your salt intake. Avoid canned, packaged, and fast food. Season food with herbs instead of salt.
  4. Lower your cholesterol. Know your cholesterol results and work on improving them if elevated with low-fat/low-cholesterol diet.
  5. Eliminating alcohol is encouraged.
  6. If you are a diabetic, you must keep your diabetes under control to lower your risk.
  7. Do not smoke or use any tobacco products to lower your risk of a TIA and stroke.
E. Take your medicines as prescribed. Do not skip your medications.
F. If your TIA was related to high blood pressure, you will be prescribed medicines to lower it. Learn to take your blood pressure and bring your blood pressure readings to your providers.
G. Aspirin is commonly prescribed after a TIA. You may be prescribed a blood thinner. Some blood thinners require regular laboratory testing. Discuss treatment with your healthcare provider.
H. If you are taking a blood thinner, get a MedicAlert bracelet/necklace, or carry a card in your wallet and car in case you are in an accident. MedicAlert jewelry can be purchased at local drugstores, and there are multiple websites for ordering MedicAlert identification. You can build your own identification bracelets or neck chains from American Medical ID: www.americanmedical-id.com.

Activity: Exercise most days of the week. You need to exercise at least 30 minutes daily three to four times a week. Walking is a great exercise. Please make sure you have been released by your healthcare provider to begin exercising.

(*continued*)

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CLIENT TEACHING GUIDE

TRANSIENT ISCHEMIC ATTACK (*CONTINUED*)

Diet: Eat a low-fat, low-cholesterol, and low-sodium diet.

Medications: _____

You Have Been Prescribed a Blood Thinner: _____

You Need to Take: _____

You Have Been Prescribed a Medicine to Lower Your Cholesterol: _____

You Need to Take: _____

WARNING SIGNS OF A TIA If you experience any of these signs, seek medical attention (call 911) immediately:

A. Weakness or numbness of the face, arm, or leg on one side of the body.
B. Trouble talking or understanding others when they talk.
C. Changes in eyesight, such as dimness, double vision, or loss of vision.
D. Dizziness, unsteadiness, or sudden falls.
E. Sudden severe headaches.

RESOURCES American Heart Association: 7272 Greenville Avenue, Dallas, TX 75231-4596, www.heart.org

National Stroke Association: 9707 East Easter Lane, Suite B, Centennial, CO 80112-3747, www.stroke.org

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CLIENT TEACHING GUIDE

TRIGEMINAL NEURALGIA

PROBLEM Trigeminal neuralgia is a chronic condition causing extreme, sporadic, sudden burning or shock-like pain of the cheek area of the face. It occurs most often in people older than age 50. The symptoms arise from the trigeminal nerve that carries sensation from your face to your brain.

PREVENTION

A. Avoid triggers such as the following:
   1. Shaving.
   2. Stroking face.
   3. Too much pressure putting on makeup.
   4. Avoiding wind exposure.
   5. Vigorous washing of face.
   6. Drinking too hot or too cold beverages.
   7. Eating tough meat or hard candies.

B. If prescribed medication, take your medication as ordered and report any side effects to the primary care provider or neurologist. Do not stop taking your medicines without talking to your provider.

C. Pain varies and may be sudden and sporadic, affecting a small area of the face or a more widespread. If pain worsens, notify your healthcare provider immediately.

Activity: Engage in activities as tolerated, avoiding triggers as much as possible. Use caution in activities if experiencing side effects of balance impairment, drowsiness, or dizziness.

Medications: _____

You Have Been Prescribed: _____

You Need to Notify the Office If You Are Experiencing:

A. Worsening pain.
B. Side effects of medications.
C. Any new symptoms.
D. Other: _____

Phone: _____

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CHAPTER 24

ENDOCRINE GUIDELINES

Jill C. Cash and Hsiao-Hui "Joyce" Ju

ADDISON DISEASE

DEFINITION

A. Primary adrenal insufficiency resulting in glucocorticoid and mineralocorticoid deficiency.

INCIDENCE AND PREVALENCE

A. Incidence is approximately five to six cases per million people. Prevalence is approximately 60 to 140 cases per million; idiopathic Addison disease as part of the autoimmune polyglandular syndrome (APS-1, APS-2) is more common in females and children. There is no racial predilection.

PATHOGENESIS

A. Autoimmune dysfunction of the adrenal cortex accounts for up to 80% to 90% of cases; 10% to 20% of cases are attributed to tuberculosis. The condition results in progressive destruction of the adrenal cortex. With at least 90% of the adrenal cortex being destroyed, adrenocortical hormones deficiency results, which may include reduced cortisol, aldosterone, and androgens. Therefore, volume and sodium depletions occur with potassium retention. The risk of death in clients with Addison disease is twofold that of the general population due to higher rates of cardiovascular disease (CVD), cancer, and infectious disease. A life-threatening crisis may occur during periods of increased stress.

PREDISPOSING FACTORS

A. Other autoimmune disorders:
  1. Insulin-dependent diabetes mellitus (IDDM).
  2. Pernicious anemia.
  3. Thyroid disorders.
B. Infections:
  1. Disseminated tuberculosis.
  2. AIDS.
  3. Systematic fungal disease.
  4. Chronic active hepatitis.
C. Gonadal failure.
D. Hypoparathyroidism.
E. Vitiligo.
F. Alopecia areata.
G. Metastatic disease (especially lung and breast cancer).
H. Certain medications (e.g., ketoconazole, anticoagulant).
I. Bleeding diathesis (e.g., disseminated intravascular coagulation [DIC]).
J. Sepsis.
K. Metabolic stress.
L. Trauma.
M. Bilateral nephrectomy.
N. Pituitary tumors.

COMMON COMPLAINTS

A. Weakness.
B. Fatigue.
C. Anorexia.
D. Nausea.
E. Diarrhea.
F. Abdominal pain.
G. Weight loss.
H. Hyperpigmentation.
I. Low libido.
J. Salt craving.

OTHER SIGNS AND SYMPTOMS

A. Proximal muscle weakness.
B. Muscle and joint pain.
C. Reduced axillary/pubic hair in women.
D. Amenorrhea.
E. Hypotension (especially orthostatic changes).
F. Anemia with:
  1. Lymphocytosis.
  2. Eosinophilia.
  3. Neutropenia.
  4. Hyponatremia.
  5. Hyperkalemia.
  6. Hypoglycemia.
  7. Hypercalcemia.
G. Positive antiadrenal antibodies.
H. Low plasma cortisol or failure to rise after cosyntropin (adrenocorticotropic hormone [ACTH]) administration.
I. EKG changes: low P waves, decreased voltage, slowed conduction with prolonged PR interval, widened QRS complex, and peaked T waves from hyperkalemia.

Missed or delayed diagnosis can lead to acute adrenal crisis, a medical emergency evidenced by sudden low back, abdominal, or leg pain; severe vomiting or diarrhea; hypotension; and loss of consciousness.

SUBJECTIVE DATA

A. Determine extent of fatigue.
B. Elicit degree and location of weakness.
C. Question the client regarding appetite, nausea, or diarrhea.
D. Evaluate food and fluid intake. ▶

The contributions of Julie Adkins, Mellisa A. Hall, Cheryl A. Glass, Angelito Tacderas, and Jenny Nelson Muller to this chapter in prior editions are acknowledged here.

E. Discuss hypopigmentation or hyperpigmentation and whether it occurs on unexposed areas as well as exposed areas of the skin.
F. Note presence of abdominal, muscle, and joint pain.
G. Assess for postural dizziness and/or fainting and when it occurs.
H. Inquire about the client's history of cancer or fungal infections.
I. Note last date of tuberculosis evaluation (purified protein derivative [PPD]) and results.
J. Determine HIV status or risk.
K. For females, discuss pubic and axillary hair distribution and note menstrual patterns.
L. Inquire about libido.
M. Inquire regarding cold intolerance.
N. Ask about recent illness and treatments.
O. Determine client's occupation to assess safety risk if weakness or dizziness is present.

PHYSICAL EXAMINATION

A. Check pulse, respirations, and blood pressure (BP); pulse and BP while seated and standing, and weight.
B. Inspect:
1. Observe overall appearance.
2. Note hair distribution and skin pigmentation, especially sun-exposed surfaces.
C. Auscultate:
1. Auscultate the heart, lungs, and abdomen.
D. Palpate:
1. Palpate the abdomen.
E. Musculoskeletal exam:
1. Perform complete musculoskeletal examination.

DIAGNOSTIC TESTS

A. Serum chemistry, electrolytes, blood urea nitrogen (BUN), creatinine, glomerular filtration rate (GFR).
B. Complete blood count (CBC).
C. PPD.
D. Rapid ACTH test: Rapid ACTH stimulation test, which measures how the adrenal glands respond to ACTH by checking the cortisol levels, excludes or establishes adrenal insufficiency but does not differentiate between primary and secondary adrenal insufficiency. With abnormal results (plasma cortisol <18 to 20 mcg/dL), proceed to plasma ACTH levels.
E. Determination of serum cortisol and plasma ACTH level, and corticotropin stimulation test as the confirmatory test.
F. Serum creatinine kinase (CK) levels.

Plasma ACTH level differentiates between primary (adrenal) and secondary (pituitary) or tertiary (hypothalamus) etiologies (high plasma ACTH with primary insufficiency, whereas normal or low with secondary insufficiency). The clinician can use ACTH-releasing hormone (CRH) to distinguish between pituitary and hypothalamic etiologies.

G. Antiadrenal antibodies: A negative adrenal antibody test is observed in only 30% to 50% of persons with idiopathic Addison disease and does not rule out adrenal insufficiency of autoimmune etiology.
H. CT scan of adrenal glands.
I. EKG.

DIFFERENTIAL DIAGNOSES

A. Secondary adrenal insufficiency (usually after exogenous glucocorticoid therapy).
B. Hypothalamic/pituitary lesions.
C. Diabetic coma.
D. Salt-losing nephritis.
E. Acute infections.
F. Cancer.
G. Anorexia nervosa.
H. Hemochromatosis.
I. Acute poisoning.
J. Myasthenia gravis.
K. Pigmentation due to racial/ethnic variations.
L. Premature primary ovarian failure.
M. Testicular failure.
N. Pernicious anemia.
O. Pyelonephritis.
P. Iatrogenic Cushing's syndrome.

PLAN

A. General interventions:
1. If primary adrenal insufficiency is established and the cause is not apparent, order an adrenal CT scan to look for metastatic disease, sarcoidosis, and tuberculosis.
B. Client teaching: *See Client Teaching Guide for this chapter, "Addison Disease."*
1. Teach the client regarding adrenal crisis and encourage treatment before symptoms begin.
2. Encourage the client to avoid contacts that predispose a person to infections.
C. Pharmaceutical maintenance therapy:
1. Hydrocortisone (drug of choice) in two or three divided doses to mimic natural diurnal rhythm; twice a day regimen includes two-thirds in the morning and the remainder in the afternoon or early evening; three times a day regimen consists of reduced doses in the morning, early afternoon, and late afternoon/early morning. Starting dose of hydrocortisone is 15 to 20 mg/d. Most adults need a total of 20 to 30 mg of hydrocortisone a day. Prednisone or dexamethasone may also be substituted.
2. During stress (illness, surgery, trauma), increase hydrocortisone therapy or additional prednisone must be given if ill. For minor illness, two to three times the routine glucocorticoid dose is recommended. For severe illness, hydrocortisone is given intravenously (IV). In emergency situations: give hydrocortisone 100 mg IV bolus. If the client has not been previously diagnosed, then give dexamethasone 4 mg IV bolus because it does not affect the diagnostic tests.
3. Fludrocortisone 0.1 mg/d is used for mineralocorticoid replacement to prevent sodium loss, postural hypotension, and hyperkalemia. During stress with an increased dose of hydrocortisone, a reduced dose of fludrocortisone at 0.05 mg/d may be sufficient.
4. If serum aldosterone is undetectable, mineralocorticoid replacement is likely necessary in addition to glucocorticoid.
5. Routine antiemetic prescription (ondansetron [ODT] 8 mg tablets as needed) may be used for nausea.
6. Abrupt discontinuation of exogenous glucocorticoid administration after a course as short as 3 weeks may induce temporary secondary adrenal insufficiency, leading to decreased cortisol but normal or near-normal ▶

aldosterone production. This may occur up to 12 months after discontinuation of glucocorticoid therapy.

FOLLOW-UP

A. Plasma renin activity: When less than 10 ng/mL, this is a probable indication of adequate fludrocortisone dose.

B. Serum and urinary cortisol and serum ACTH to monitor hydrocortisone dose. Urinary-free cortisol greater than 70 mcg/24 hours indicates excessive hydrocortisone dose, whereas values less than 20 mcg/24 hours indicate inadequate hydrocortisone dose.

C. Monitor BP and serum electrolytes to determine fludrocortisone dose.

D. Do annual adrenal function studies.

CONSULTATION/REFERRAL

A. If Addison disease is suspected, consult with a physician and refer to an endocrinologist.

B. Consider Addison disease in any client with unexplained hypotension and hyperkalemia.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Because of physiologic changes in plasma cortisol, diagnosis is based on lack of rise in plasma cortisol concentration after ACTH administration.

2. If nausea and vomiting are problems, intramuscular glucocorticoid may be necessary.

3. Delivery requires increased glucocorticoid dose.

B. Geriatrics:

1. Urinary excretion rate of cortisol decreases by about 25%; serum level and response to ACTH stimulation are unchanged.

2. Symptoms of depression and elevated levels of cortisol are associated with atrophy of the prefrontal and limbic areas of the brain.

3. Decreased serum sodium and hydration compromise are frequent electrolyte concerns in geriatrics due to increased antidiuretic hormone and frequent antidiuretic prescriptions. Monitor older adult clients closely to differentiate from an adrenal disorder versus medications and/or natural physiologic aging process.

4. Medications prescribed with consideration to the Beers Criteria:

a. Geriatric clients with Addison disease: Check sodium levels when starting oxcarbazepine (anticonvulsant), Remeron (sleep/appetite enhancer), tricyclic antidepressants, selective serotonin reuptake inhibitors (SSRIs), and selective serotonin-norepinephrine reuptake inhibitors (SNRIs; antidepressants). Alternatives for neuropathic pain that are safe for geriatrics with Addison disease may include gabapentin, capsaicin, or lidocaine patch depending on associated conditions.

b. Check potassium level at initiation of spironolactone due to risk of hyperkalemia. It is recommended to not use this medication in the geriatric population if there has been a history of hyperkalemia. An alternative suggestion for heart failure treatment would be a low-dose angiotensin-converting enzyme inhibitor (ACEI) with a thiazide while regularly monitoring the basic metabolic panel (BMP), including electrolytes and renal function.

c. Regarding special concerns for clients with cognitive impairment, history of delirium, dementia, or history/risk for falls, check sodium level when starting/changing any antipsychotic medications. Nonanticholinergic agents have been acceptable for older adult clients suffering from mental health diseases and/or dementia-related behavioral problems. Use the lowest dose for the shortest time possible.

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CUSHING SYNDROME

DEFINITION

A. Cushing syndrome is a cluster of symptoms, signs, and biochemical abnormalities arising from glucocorticoid overproduction. The most common cause is iatrogenic (induced by administration of glucocorticoids). Cushing disease is depicted by an increase secretion of adrenocorticotropic hormone (ACTH) from the anterior pituitary, which stimulates the adrenal glands to release an overproduction of cortisol. Another cause is ectopic ACTH production by tumors outside the pituitary gland.

INCIDENCE AND PREVALENCE

A. Two to four new endogenous cases per 1,000,000 are diagnosed annually. The prevalence is estimated to be 75 individuals per million and is five times more frequent in females. Cushing disease occurs more often in females between 25 and 45 years old and is associated with increased mortality rates, especially from cardiovascular diseases.

PATHOGENESIS

A. The cause is exogenous (chronic glucocorticoid or ACTH administration) or endogenous (increased ACTH secretion or excessive cortisol secretion). The endogenous type is divided into ACTH-dependent and ACTH-independent. ACTH-dependent type is most commonly due to either excessive pituitary or ectopic ACTH secretion, resulting in signs of androgen excess. ACTH-independent is often due to autonomous cortisol overproduction with depressed ACTH production and absent signs of androgen excess. The etiology of spontaneous Cushing (adults) comprises:

1. 70% to 80% pituitary ACTH hypersecretion (90% pituitary adenoma, 10% pituitary hyperplasia).

2. 10% to 15% autonomous adrenal tumor (adenoma or carcinoma).

3. 5% to 15% ectopic ACTH secretion (nonpituitary neoplasm, usually lung).

4. Less than 1% bilateral nodular hyperplasia without ACTH.

PREDISPOSING FACTORS

A. Exogenous glucocorticoid administration.

B. Excessive alcohol intake.

C. Pituitary adenoma.

D. Thoracic tumors.

E. Adrenal neoplasms.

F. Tumors of the pancreas.

G. Thyroid and thymus disease.

H. Pheochromocytoma.

COMMON COMPLAINTS

A. Excessive coarse hair on face, chest, and back.
B. Rapid weight gain.
C. Easy bruising.
D. Muscle weakness.
E. Oligo- or amenorrhea.
F. Impotence or decreased libido.
G. Depression.
H. Poorly controlled diabetes.
I. Irregular menses.

OTHER SIGNS AND SYMPTOMS

A. Cervicodorsal "buffalo hump" and supraclavicular fat pad.
B. Hirsutism in females.
C. Acne and/or folliculitis.
D. Increased intraocular pressure.
E. Purple striae.
F. Increased blood pressure (BP).
G. Polydipsia/polyuria, increased serum glucose, and glycosuria.
H. Osteopenia/osteoporosis.
I. Mood lability/changes.
J. Spontaneous hypokalemia.
K. Erythrocytosis.
L. Round "moon-like" face.
M. Bruising tendency.

SUBJECTIVE DATA

A. Ask whether the client has taken exogenous glucocorticoids.
B. Determine whether the onset of complaints was acute or subacute.
C. Assess for bruising and determine if bruising was precipitated by trauma.
D. Assess for muscle weakness and, if present, whether it is proximal weakness.
E. Review the client's menstrual history, including characteristics of menstrual periods.
F. Identify the client's family history of similar problems.
G. Question the client regarding any vision impairment.
H. Rule out the presence of abdominal pain.
I. Review the client's history of neoplasms and location.
J. Identify the current pattern of sexual function.
K. Question the client regarding mood swings or recent treatment for psychiatric disorder.
L. Identify the pattern of weight gain and effectiveness of weight loss interventions, if implemented.
M. Assess for the presence of leg or arm pain.
N. Review the client's history of fractures, especially if postmenopausal.
O. Determine the amount of alcohol consumed.

PHYSICAL EXAMINATION

A. Check BP, pulse, height, and weight.
B. Inspect:
  1. Inspect the skin, noting hair distribution, lesions, bruising, and striae.
  2. Observe the face and note shape.
  3. Observe the neck.

Note that a "moon-shaped" face and fat pads in posterior neck ("buffalo hump") are characteristics of clients with Cushing syndrome.

  4. Complete funduscopic examination. Be alert for cataracts, glaucoma, and/or signs of benign intracranial hypertension (HTN).
C. Auscultate:
  1. Auscultate the heart and lungs.
D. Palpate:
  1. Complete musculoskeletal examination. Be alert for septic necrosis of femoral and/or humeral head. Perform range of motion, noting decreased range of motion, pain, or weakness of joint. Consider diagnostic testing if abnormal exam.
  2. Assess for kyphosis or back pain associated with osteoporosis and long-term cortisol excess.

INITIAL DIAGNOSTIC TESTS

A. Serum electrolytes.
B. Complete blood count (CBC) and glucose.
C. 24-hour urine-free cortisol (at least two measurements).
D. Late-night salivary cortisol (at least two measurements).
E. Overnight dexamethasone suppression test, 1 mg.
F. Longer low-dose dexamethasone suppression test (2 mg/d for 48 hours).
G. Bone density studies.

The overnight dexamethasone suppression test has a false-positive rate of 20% to 30%; false-positives can occur with obesity, stress, depression, alcoholism, pregnancy, or medications that increase the hepatic metabolism of cortisol and dexamethasone (e.g., antiseizure drugs, estrogen, and rifampin). The false-negative rate is less than 3%. Use the low-dose dexamethasone test as an alternative.

The 24-hour urine-free cortisol test is the most sensitive and specific test and is the best choice for screening.

H. If any of the earlier initial screenings is positive, the client should be referred to endocrinology for additional evaluation.

DIFFERENTIAL DIAGNOSES

A. Depression.
B. Severe obesity.
C. Chronic stress.
D. Familial cortisol resistance.
E. Medication-induced (e.g., phenytoin, phenobarbital, primidone).
F. Pituitary adenoma.
G. Adrenal and other neoplasms.
H. Alcoholism.
I. Nephrolithiasis.
J. Psychosis.

PLAN

A. General interventions:
  1. Taper glucocorticoid dose as appropriate for underlying disease.
  2. Begin alcohol detoxification if applicable.
  3. Consider hormone replacement therapy for postmenopausal females.
B. Client teaching: *See Client Teaching Guide for this chapter, "Cushing Syndrome."*
C. Medical–surgical management:
  1. If noniatrogenic etiology, surgery is the treatment of choice, followed by irradiation and/or chemotherapy. ▶

D. Pharmaceutical therapy:

1. Calcium 1,000 mg/d and vitamin D 400 IU/d.

2. Mitotane (Lysodren) 2 to 6 g daily for control of adrenocortical carcinoma progression.

3. Metyrapone inhibits adrenal steroid biosynthesis.

4. Ketoconazole (most useful for blocking adrenal steroidogenesis) and suramin inhibit adrenal steroid biosynthesis. Ketoconazole is associated with hepatic failure and has multiple drug-to-drug interactions, which may result in fatal ventricular arrhythmias. An endocrinologist should be consulted before the use of oral ketoconazole.

a. Ketoconazole should be started at a dose of 200 mg two or three times daily and increased rapidly to 400 mg TID; higher doses are seldom more effective. Increase every 4 to 7 days. Avoid use in pregnancy.

5. Hydrocortisone may be given in physiologic doses to avoid adrenal insufficiency after surgical resection of the adrenal, pituitary, or ectopic ACTH-secreting tumor.

Most persons on daily steroid program for over 2 to 4 weeks have some degree of hypothalamic–pituitary–adrenal axis suppression.

6. Mifepristone (RU-486) is given for ectopic ACTH production or adrenal carcinoma. Mifepristone is an abortifacient.

FOLLOW-UP

A. At the provider's discretion, 1 to 2 weeks after tests are complete, follow up to discuss results and possible therapy.

CONSULTATION/REFERRAL

A. If Cushing syndrome is suspected, consult with an endocrinologist regarding treatment and therapy.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Urinary-free cortisol increases in the third trimester, but females still have normal 17-hydroxycorticosteroids and normal diurnal variability of serum cortisol. Dexamethasone testing is not recommended in the initial screening for Cushing syndrome during pregnancy.

B. Geriatrics:

1. Loss of brain volume and neurocognitive impairment have been reported in clients with Cushing syndrome. Surgical treatment of adrenal hyperplasia for clients with Cushing syndrome may be useful in treating neuropsychiatric symptom and reducing caregiver burden.

2. Assess for depression in older adult clients with Cushing syndrome; they are particularly susceptible to emotional as well as cognitive impairments when suffering from the disease.

3. Beers Criteria: Corticosteroids may worsen delirium in geriatrics. Use lowest effective dose for the shortest amount of time and avoid combining with aspirin (ASA)/nonsteroidal anti-inflammatory drugs (NSAIDs) to prevent gastrointestinal (GI) side effects.

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DIABETES MELLITUS

DEFINITION

A. Diabetes mellitus (DM) is a group of diseases characterized by high levels of blood glucose either due to a complete insulin deficiency, a progressive defect in insulin secretion or action, and/or an impaired response of tissues and cells to insulin. There are four categories of diabetes: type 1, type 2, gestational diabetes, and diabetes from secondary causes.

1. Type 1 diabetes, formerly referred to as insulin-dependent diabetes mellitus (IDDM), type 1, or juvenile-onset diabetes, is an endocrine condition in which there is complete destruction of pancreatic beta cells or a complete absence of insulin.

2. Type 2 diabetes, formerly referred to as noninsulin-dependent diabetes mellitus (NIDDM), type 2, or adult-onset diabetes, describes a condition in which individuals have an impairment in insulin production and/or insulin resistance.

3. Gestational diabetes is diagnosed during pregnancy. It usually resolves when the pregnancy is completed. It will increase the client's risk of developing type 2 diabetes later in life.

4. Diabetes resulting from secondary causes is due to genetic defects and/or other diseases, such as cystic fibrosis, chronic pancreatitis, or pancreatic cancer. Secondary diabetes can also be drug-/chemical-induced from medications or therapies used when treating other conditions including HIV/AIDS and in the management of organ transplant recipients.

B. People with type 2 diabetes are more likely to have elevated triglyceride levels and decreased high-density lipoprotein (HDL) cholesterol levels placing them at greater risk for atherosclerotic cardiovascular disease (ASCVD). Clients with diabetes are two to four times more likely to have comorbidities including cardiovascular disease (CVD) and stroke. The American College of Cardiology (ACC) and the American Heart Association (AHA) published guidelines on the assessment of cardiovascular risk, lifestyle management, and treatment of cholesterol to reduce ASCVD risks. An estimation of 10-year and lifetime risk for ASCVD and a web-based calculator can be found at www.cvriskcalculator.com. These tools are used to drive conversations on client risk factors for ASCVD and potential risks as well as benefits for cholesterol management. The assessment of ASCVD risk factors is recommended every 4 to 6 years in healthy adults 20 to 79 years of age. Long-term and lifetime risk information may be used to motivate therapeutic lifestyle changes and encourage adherence to lifestyle as well as pharmacologic therapies.

INCIDENCE AND PREVALENCE

A. It is estimated that more than 34 million Americans have diabetes, which is approximately 10.5% of the population. Diagnosed cases account for 26.8 million, and an estimated 17.3 million have not been diagnosed. Type 2 diabetes accounts for majority of the diagnosed cases (90%–95%), while type 1 diabetes accounts for 5% to 10% of the diagnosed cases. There are 1.5 million new cases reported each year, not counting those 19 years old or younger.

PATHOGENESIS

A. Type 1 diabetes develops from an autoimmune condition leading to the destruction of insulin-producing beta cells, ▶

often triggered by an inherited defect and/or environmental factors. Environmental factors that may influence the etiology of diabetes include viral illnesses: mumps, coxsackievirus, cytomegalovirus, and hepatitis. Other factors that may influence the developmental of disease include emotional and physical stress, and/or environmental toxins.

B. Type 2 diabetes involves impaired insulin secretion, insulin resistance, and/or an abnormally elevated glucose production by the liver. Genetics, inactivity, and obesity are major risk factors.

C. The severity of carbohydrate intolerance in gestational diabetes is unknown. Clients identified at risk should be screened during the 24th and 28th weeks of gestation.

D. Genetic defects and medications/chemicals are thought to affect the beta-cell function and alter insulin function. Hemoglobin A1C (HgbA1C) levels may not be interpreted correctly in clients with blood disorders such as anemia/hemoglobinopathies. See www.ngsp.org/interf.asp for a complete list of laboratory methods recommended to be used to measure HgbA1C values for clients with hemoglobin variants (sickle cell trait, HbC, HbS, HbE, HbD trait, or elevated HbE).

PREDISPOSING FACTORS

A. First-degree relative with type 1 or type 2 diabetes.

B. Physical inactivity.

C. Body mass index (BMI) greater than or equal to 27 kg/m^2

D. HbgA1C greater than or equal to 5.7%, impaired glucose tolerance (IGT), or impaired fasting glucose on previous testing.

E. Native American, Hispanic, Asian, African American, and Pacific Islander heritage.

F. Hypertension (HTN) with systolic pressure greater than 140 mmHg and diastolic pressure greater than 90 mmHg.

G. HDL level of 40 mg/dL or less for males and 50 mg/dL or less for females, and/or triglyceride level of greater than or equal to 150 mg/dL.

H. History of giving birth to babies larger than 9 lbs or gestational diabetes.

I. History of IGT or fasting glucose.

J. Acanthosis nigricans or severe obesity.

K. Polycystic ovarian syndrome (PCOS).

L. History of CVD.

M. The U.S. Preventive Services Task Force (USPSTF) recommends screening for abnormal blood glucose as part of cardiovascular risk assessment in adults aged 35 to 70 years who are overweight or obese.

COMMON COMPLAINTS

A. Classic triad of symptoms:
1. Polyuria.
2. Polydipsia.
3. Polyphagia.

B. Weight loss.

C. Lack of energy.

D. Recurrent infections (urinary tract, vaginal, skin breakdown that is slow to heal).

E. Asymptomatic.

OTHER SIGNS AND SYMPTOMS

A. Weakness.

B. Fatigue.

C. Nausea and vomiting.

D. Abdominal pain.

E. Anorexia.

F. Sexual dysfunction, including impotence or dyspareunia.

G. Itching.

H. Visual disturbances.

I. Signs and symptoms related to nephropathy, neuropathy, and/or retinopathy.

SUBJECTIVE DATA

A. Obtain a detailed history regarding onset, duration, and course of presenting symptoms.

B. Question the client regarding all characteristic signs and symptoms of diabetes.

C. Determine the client's nutritional status, 24-hour recall, weight history, and eating patterns.

D. Review the family history of diabetes or other endocrine disorders.

E. Note predisposing factors to diabetes.

F. Review the client's social history, including smoking, alcohol, and exercise.

PHYSICAL EXAMINATION

A. Check pulse, respirations, blood pressure (BP), and weight.

B. Inspect:
1. Observe overall appearance.
2. Perform oral examination. Clients with diabetes are prone to thrush, gingivitis, plaque, and infections. A dental examination should be done every 6 months.
3. Complete funduscopic examination. Proliferative diabetic retinopathy is the leading cause of new blindness in adults in the United States. Clients with diabetes are 25 times more at risk for blindness and have four to six times the risk for cataracts and twice the risk for glaucoma.
4. Inspect the skin, including the feet, hands, fingers, skin folds, and insulin injection sites for erythema and deformities.
5. Perform foot examination to assess skin integrity, nail care, circulation, sensation (monofilament testing), ankle reflexes, and appropriate footwear.

C. Auscultate:
1. Heart.
2. Lungs.
3. Carotid arteries.

D. Percuss:
1. Percuss the chest, abdomen, and deep tendon reflexes (DTRs).

E. Palpate:
1. Palpate the neck (thyroid).
2. Palpate the abdomen.
3. Palpate the extremities and check pulses.

DIAGNOSTIC TESTS

A. HgbA1c of 6.5% or higher.

B. Fasting plasma glucose: greater than or equal to 126 mg/dL. All clients should have a baseline fasting blood sugar (fasting for at least 8 hours) performed at 45 years of age, then repeated every 3 years. The baseline should be performed earlier if any predisposing factors exist.

C. Random plasma glucose: greater than or equal to 200 mg/dL with symptoms of diabetes.

D. Oral glucose tolerance test (OGTT): After 75-g glucose load, a 2-hour plasma glucose greater than or equal to 200 mg/dL; IGT is a fasting plasma glucose greater than or equal to 126 mg/dL.

According to the Diabetes Control and Complications Trial, an HgbA1c of 7.2% or below decreases the risk of retinopathy, neuropathy, and nephropathy by 50% to 70%.

DIFFERENTIAL DIAGNOSES

A. Benign pancreatic insufficiency.
B. Pheochromocytoma.
C. Cushing syndrome.
D. History of corticosteroid use.
E. Stress hyperglycemia.
F. Acromegaly.
G. Hemochromatosis.
H. Somogyi phenomenon: early morning hyperglycemia due to very early morning (2:00–3:00 a.m.) hypoglycemia.

PLAN

A. General interventions:
1. Establish, review, and evaluate individual goals with the client on a routine basis.
2. Center goals around normal metabolic control and the prevention and delay of complications while maintaining a flexible, normal, high-quality life.
3. After a new diagnosis is made and treatment has begun, be alert for an initial remission or honeymoon phase with decreased insulin needs and better control that may last 3 to 6 months.
4. Include the following in the treatment plan:
a. Exercise plan:
i. Develop a consistent, individualized exercise plan with the client to improve insulin sensitivity, blood sugars, weight reduction, and reduction of cardiovascular complications.
ii. Evaluation by a healthcare provider, including a complete physical examination and EKG, must precede any exercise program.
iii. Generally, the goals for physical activity are to reduce low-density lipoprotein cholesterol (LDL-C) and non-high-density lipoprotein cholesterol (non-HDL-C) and to lower BP. The exercise should involve moderate-to-vigorous intensity.
iv. Exercise should not be done if the fasting blood sugar is greater than 250 mg/dL and ketones are present in the urine or if the glucose level is greater than 300 mg/dL at any time regardless of the presence of ketones.
v. Because exercise can lower blood sugar concentration, special precautions such as medication adjustment and meal planning should be done before and after exercise if the client is taking insulin or a glucose-lowering medication.
b. Self-monitoring blood glucose (SMBG): The process of monitoring the client's blood gives valuable information to the client on a daily basis and assists the provider in identifying trends.
i. Several different meters are available with a variety of options. A certified diabetes educator can show examples of different types before the client purchases one.
ii. Frequency of testing depends on the type of prescribed medication, the client's adherence, and the schedule of insulin administration, if applicable.
iii. Additional testing should be done at times of medication changes, meal plans, exercise, and during illness or stressful events.
iv. Continuous blood glucose (CBG) monitoring is recommended for clients who require tight blood glucose control and those with hypoglycemia unawareness or frequent nocturnal hypoglycemia. Additional fingerstick glucose checks are required with CBG monitoring if hypoglycemia occurs.
c. Psychosocial support: It is important from the beginning of treatment to give the client a sense of control.
I. Consistent involvement of family members will likely promote treatment adherence.
II. Assess and discuss psychosocial issues at each visit, including diabetes distress and depression.
B. Client teaching: *See Client Teaching Guide for this chapter, "Diabetes."*
1. Topics in the educational plan include the pathophysiology of diabetes, procedures for SMBG and medication therapies, recognition and treatment of hypoglycemia, and instructions for special situations such as pregnancy, illness, and traveling.
2. Include preventive care, instructions for family members, and the importance of wearing a MedicAlert tag.
3. Smoking cessation and avoidance of all tobacco products should be advised to all clients. Counseling regarding smoking/tobacco cessation methods and resources should be offered.
C. Dietary/physical activity management:
1. Nutritional plan: The client should meet with a dietitian and/or a diabetes educator who has expertise in diabetes nutritional therapy.
2. Eating patterns and ideal percentage of calories from protein, carbohydrates, and fat should be individualized for each client.
3. Involve the family to improve adherence with nutritional therapy.
4. Overweight/obese clients are encouraged to set nutrition goals to achieve a health weight.
5. Along with dietary management, exercise programs should be individualized and tailored to the specific needs of each client. The American Diabetes Association (ADA) recommends at least 150 minutes per week of moderate-to vigorous-intensity physical exercise for at least 3 days a week, with not more than 2 consecutive days of rest. Resistance training is recommended for 2 to 3 days a week on nonconsecutive day. Exercise recommendations should be individually tailored to ensure the safety of each client.
D. Pharmaceutical therapy (Figure 24.1):
1. Type 1 diabetes depends on exogenous insulin for treatment.
2. Type 2 diabetes treatment depends on individual factors and the severity of disease at diagnosis. If HgbA1C is above goal (7.5%–8%), pharmaceutical treatment should be initiated along with an individualized exercise program and nutrition plan. If HgbA1C is near goal (<7.5%), lifestyle modifications for 3 to 6 months may be considered in highly motivated individuals, with a reassessment of HgbA1C to determine the need for pharmaceutical therapy.
a. First line for type 2 diabetes: Metformin is the preferred oral medication for type 2 diabetes. If monotherapy at the maximum dose and goal is not achieved after 3 months, additional agents should be added based on client factors. For some clients, insulin may eventually be required.
i. Metformin is contraindicated in those with an estimated glomerular filtration rate (eGFR) less than 30 mL/min/1.73 m^2.

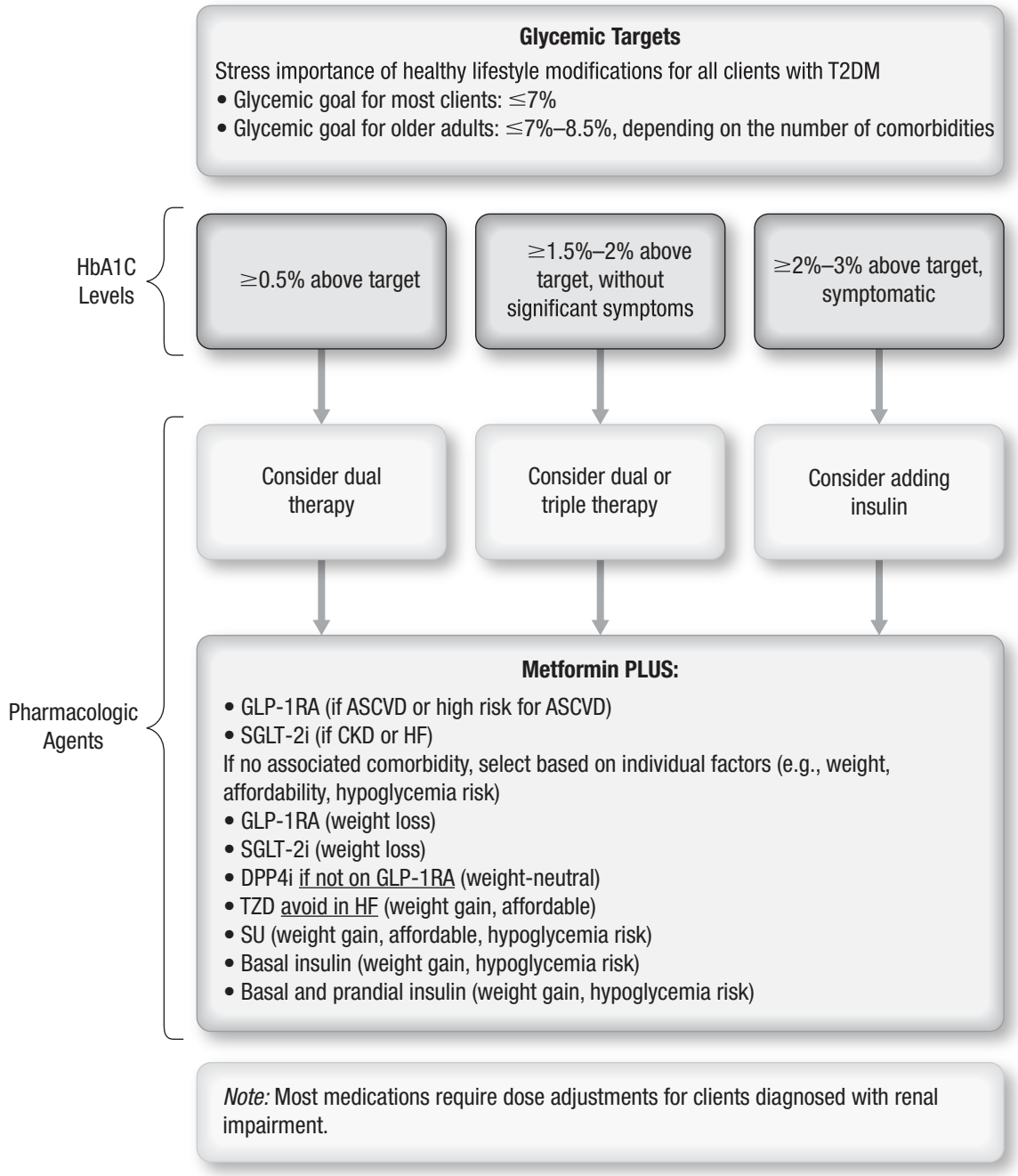


FIGURE 24.1 Type 2 diabetes mellitus pharmacologic management.

ASCVD, atherosclerotic cardiovascular disease; CKD, chronic kidney disease; DPP4i, dipeptidyl peptidase-4 inhibitor; GLP-1RA, glucagon-like peptide 1 receptor agonist; HbA1C, hemoglobin A1C; HF, heart failure; SGLT-2i, sodium glucose cotransporter-2 inhibitors; SU, sulfonylurea; T2DM, type 2 diabetes mellitus; TZD, thiazolidinedione.

ii. Insulin should be considered initially for clients presenting with random glucose levels greater than 300 mg/dL or an HgbA1C greater than 10%.

b. Add additional agents when glucose control is not achieved with metformin alone. These are recommendations based on ADA and American Association of Clinical Endocrinologists (AACE) guidelines:

i. For those with or at high risk of ASCVD, consider:

1) Glucagon-like peptide-1 (GLP-1) receptor agonists (RAs).

OR

2) Sodium glucose cotransporter-2 (SGLT-2) inhibitors.

ii. For those with heart failure (HF) or chronic kidney disease, consider:

1) SGLT-2 inhibitors.

a) However, not recommended in eGFR less than 45 mL/min/1.73 m^2; refer to drug specific label for differences in each medication.

b) Avoid SGLT-2 inhibitors in clients with frequent urinary tract or genitourinary yeast infections, decreased bone density, ▶

foot ulcerations, and those at increased risk of developing diabetic ketoacidosis.

ii. For those with nondialysis eGFR less than 30 mL/min/1.73 m², consider:

1) Linagliptin, repaglinide, low-dose sulfonylurea (SU), GLP-1 RA, or insulin.

a) Teach clients to monitor for dehydration from nausea with the use of GLP-1 RAs (e.g., liraglutide, semaglutide, dulaglutide). Exenatide should not be used if creatinine clearance is less than 30 mL/min.

b) Discuss the risk of hypoglycemia with repaglinide, SU, and insulin.

c. For those with a HgbA1C ≤9% and have no cardiovascular or kidney disease, multiple options are available.

i These have less hypoglycemia risk:

1) GLP-1 RAs, SGLT-2 inhibitors, dipeptidyl peptidase-4 (DPP-4) inhibitors, or pioglitazone.

ii These tend to cause weight loss:

1) GLP-1 RAs or SGLT-2 inhibitors.

iii These are affordable:

1) SU or thiazolidinediones (TZD)

d. For those with a HgbA1C greater than 9% and have no cardiovascular or kidney disease, consider:

i Insulin or GLP-1 RA.

e. Combination therapy:

i. Two-drug combination:

1) Metformin + GLP-1 RA.

2) Metformin + SGLT-2 inhibitor.

3) Metformin + DPP-4 inhibitor.

4) Metformin + TZD.

5) Metformin + SU.

6) Metformin + insulin (basal) and/or before meals (prandial).

ii. Three-drug combinations:

1) Metformin + GLP-1 RA + SGLT-2 inhibitor or TZD.

2) Metformin + SGLT-2 inhibitor + GLP-1 RA or DPP-4 inhibitor or TZD.

3) Metformin + DPPT-4 inhibitor + SGLT-2 inhibitor or TZD.

4) Metformin + TZD + SGLT-2 inhibitor or DPP-4 inhibitor or GLP-1 RA.

5) Metformin + SU + TZD.

iii. Complicated, uncontrolled clients may require insulin.

f. Consider initiation of insulin therapy:

i. Glucose level at diagnosis of type 2 diabetes greater than 300 mg/dL.

ii. HgbA1c greater than or equal to 9% to 10%.

iii. Ketonuria.

*Although clients with type 2 diabetes do not depend on exogenous insulin, many will require supplemental insulin during times of stress, illness, or pregnancy or routinely along with oral medication (*Tables 24.1 and 24.2*).* ▶

TABLE 24.1 ACTION TIMES OF INSULIN

Insulin	Onset	Peak	Duration
Rapid-Acting: Lispro (Humalog) Aspart (NovoLog) Glulisine (Apidra)	5–15 min	1–2 hr	4–6 hr
Short-Acting: Regular (Humulin Regular, Novolin Regular)	30–60 min	2–3 hr	6–8 hr
Intermediate-Acting: NPH (Humulin NPH, Novolin NPH)	1–2 hr	4–10 hr	12–18 hr
Premixed: Humulin 70/30; Novolin 70/30 (70 NPH/30 Regular)	30–60 min	2–12 hr	10–16 hr
Premixed: Humalog mix 50/50 (50% insulin lispro protamine and 50% insulin lispro)	5–20 min	1–2 hr	10–16 hr
Long-Acting: Detemir (Levemir)	1–2 hr	Relatively flat	6–24 hr
Long-Acting: Glargine U-100 (Lantus, Basaglar)	1–4 hr	Relatively flat	20–24 hr
Long-Acting: Glargine U-300 (Toujeo)	6 hr	Relatively flat	Up to 36 hr
Ultra Long-Acting: Degludec (Tresiba)	1 hr	Relatively flat	Up to 42 hr

NPH, neutral protamine hagedorn.

TABLE 24.2 DIABETES MEDICATION/CLASS

Drug	Brand Name	Drug Classification
Acarbose	Precose	Alpha-glucosidase inhibitor
Pioglitazone + metformin	ActoPlus Met	TZD plus biguanide
Glimepiride	Amaryl	SU
Insulin glulisine	Apidra	Rapid-acting insulin
Rosiglitazone + metformin	Avandamet	TZD plus biguanide
Rosiglitazone + glimepiride	Avandaryl	TZD plus SU
Exenatide	Byetta	GLP-1 receptor agonist
Exenatide extended-release	Bydureon	GLP-1 receptor agonist
Glyburide	DiaBeta	SU (second generation)
Pioglitazone + glimepiride	Duetact	TZD plus SU
Glucagon IM or SQ	N/A	Antihypoglycemic
Metformin extended-release	Glucophage	Biguanide
Glipizide extended-release	Glucotrol	SU (second generation)
Glyburide + metformin	Glucovance	SU plus biguanide
Glyburide micronized	Glynase PresTab	SU (second generation)
Miglitol	Glyset	Alpha-glucosidase inhibitor
Insulin lispro	Humalog	Rapid-acting insulin
NPH/regular insulin	Humulin 70/30	Short- and intermediate-acting insulin
NPH/regular insulin	Humulin 50/50	Short- and intermediate-acting insulin
Regular insulin	Humulin R	Short-acting insulin
NPH insulin	Humulin N	Intermediate-acting insulin
Sitagliptin + metformin	Janumet	Dipeptidyl peptidase-4 inhibitor plus biguanide
Sitagliptin	Januvia	Dipeptidyl peptidase-4 inhibitor
Insulin glargine	Lantus	Long-acting insulin
Insulin detemir	Levemir	Long-acting insulin
Linagliptin	Tradjenta	Dipeptidyl peptidase-4 inhibitor
Liraglutide	Victoza	Glucagon-like peptide-1 receptor agonist
Metformin + glipizide	Metaglip	Biguanide plus SU
Glyburide	Micronase	SU (second generation)
Miglitol	Glyset	Alpha-glucosidase inhibitor
Nateglinide	Starlix	Meglitinide
Insulin isophane + regular insulin	Novolin 70/30	Short- and intermediate-acting insulin
Insulin aspart	NovoLog	Rapid-acting Insulin
Insulin aspart protamine/insulin aspart	NovoLog Mix 70/30	Rapid- and intermediate-acting insulin
Insulin lispro protamine/insulin lispro	Humalog Mix 75/25	Rapid- and intermediate-acting insulin

(*continued*)

TABLE 24.2 DIABETES MEDICATION/CLASS (*CONTINUED*)

Drug	Brand Name	Drug Classification
Insulin lispro protamine/insulin lispro	Humalog Mix 50/50	Rapid- and intermediate-acting insulin
Insulin isophane suspension NPH	Novolin N	Intermediate-acting insulin
Saxagliptin	Onglyza	Dipeptidyl peptidase-4 inhibitor
Pioglitazone	Actos	TZD
Repaglinide + metformin	Prandimet	Meglitinide analogue plus biguanide
Pramlintide	Symlin	Amylin analogue/amylinomimetic
Repaglinide	Prandin	Meglitinide analogue
Rosiglitazone	Avandia	TZD
Semaglutide	Ozempic	GLP-1 receptor agonist
Ertugliflozin	Steglatro	Sodium-glucose cotransporter inhibitor
Ertugliflozin + metformin	Segluromet	Sodium-glucose cotransporter inhibitor + biguanide
Ertugliflozin + sitagliptin	Steglujan	Sodium-glucose cotransporter inhibitor + dipeptidyl peptidase-4 inhibitor

GLP-1, glucagon-like peptide-1; IM, intramuscular; NPH, neutral protamine hagedorn; SQ, subcutaneous; SU, sulfonylurea; TZD, thiazolidinedione.

g. Additional medications that may need to be considered:
   i. Angiotensin-converting enzyme (ACE) inhibitors are the antihypertensive drug of choice to delay renal dysfunction associated with diabetes.
   ii. ACE inhibitors or angiotensin II receptor blockers (ARBs) are recommended for clients with an elevated urinary albumin excretion (30–299 mg/24 hours).
   iii. Calcium channel blockers may reduce microalbuminuria and proteinuria.
   iv. Aspirin therapy (75–162 mg/d) is recommended for primary prevention in clients with type 1 or type 2 diabetes with an increased risk of CVD (males >50 years, females >60 years with one or more risk factors, such as CVD, HTN, smoking, dyslipidemia, or albuminuria). For clients who have a history of CVD with an allergy to aspirin, clopidogrel 75 mg/d should be used.

h. Some medications may adversely affect diabetes:
   i. Nicotinic acids may affect glycemic control by increasing insulin resistance.
   ii. Beta-blockers may alter symptoms of hypoglycemia.
   iii. Thiazide diuretics may increase insulin resistance.

3. The ACC/AHA published guidelines on the primary prevention and treatment of cholesterol with people with DM. These include:

a. Moderate-intensity statin therapy initiated or continued for adults with DM 40 to 75 years of age.

b. High-intensity statin therapy is reasonable for adults with DM and multiple risk factors to reduce LDL-C level by ≥50%.

c. In adults with DM who are older than 75 years of age, it is reasonable to evaluate the potential for ASCVD benefits and for adverse effects, to assess drug–drug interactions, and to consider client preferences when deciding to initiate, continue, or intensify statin therapy.

FOLLOW-UP

A. Determine follow-up appointments by the type of diabetes, age, client compliance, any treatment changes, and presence of any complications related to diabetes or other health problems.

B. HgbA1C determination every 3 months can assist the provider in measuring control. There are several computer software packages that print the glucometer readings for assessing compliance; this can be done by the provider or the client.

C. ADA management goals:

1. Preprandial glucose level 80 to 130 mg/dL.

2. A glucose level less than 180 mg/dL 2 hours after meals.

3. A bedtime glucose level 90 to 150 mg/dL.

4. HgbA1c less than 7% for most individuals; less than 7% to 8.5% for older adults depending on the number of comorbidities.

5. The ADA and the ACC/AHA recommend adults with diabetes, HTN, cardiovascular risks, and chronic kidney disease be treated to a systolic blood pressure (SBP) goal of less than 130 mmHg and a diastolic blood pressure (DBP) of less than 80 mmHg.

6. Other goals are triglycerides less than 150 mg/dL, HDLs greater than 35 mg/dL, and LDLs less than 100 mg/dL or less than 70 mg/dL for high-risk clients.

D. Tests or examinations should include a dilated funduscopic examination by an ophthalmologist to screen for retinopathy every 1 to 2 years, monofilament foot examination, flu vaccination and other adult vaccines as recommended, serum creatinine, blood urea nitrogen (BUN), glomerular filtration rate, lipid panel, urinalysis, and urine for albuminuria. Additional tests may be needed if complications develop.

CONSULTATION/REFERRAL

A. Refer to a physician if the client experiences the following:

1. Diabetic ketoacidosis.

2. Severe or frequent hypoglycemia that is unresponsive to conventional pharmaceutical therapy.

3. Hyperosmolar hyperglycemic nonketotic syndrome.

4. Pregnancy.

5. Symptoms from an acute complication related to retinopathy.

6. Nephropathy develops 35% to 45% of the time in type 1 and 20% of the time in type 2 diabetes; it is the leading cause of end-stage renal disease (ESRD) requiring dialysis.

7. Neuropathy: 60% to 70% of clients experience impaired sensation or pain in feet/hands, or carpal tunnel syndrome; over half of all amputations of lower extremities are related to diabetes. All clients should be screened for neuropathy at the diagnosis of type 2 diabetes and at 5 years after the diagnosis of type 1 diabetes, and then annually. Early diagnosis is imperative to prevent nerve damage from occurring. Tight control of the blood glucose levels can slow down the progression of nerve damage but cannot reverse neuronal loss. The pain experienced with neuropathy can be treated with oral medications. Pregabalin and duloxetine are both Food and Drug Administration (FDA)-approved for neuropathic pain. Other medications that may be used include opioids such as tramadol or morphine, along with venlafaxine, amitriptyline, gabapentin, or valproate.

8. Persistent uncontrolled diabetes.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy/preconceptual:

1. Clients considering pregnancy should be switched to insulin before conception and during pregnancy:

a. Fetal anomalies increase proportionally with uncontrolled diabetes.

b. HgbA1c goal prior to conception is less than 7%.

c. Gestational diabetes screening may be completed by performing the "one-step" 2-hour 75-g OGTT or a "two-step" approach with 1-hour 50-g nonfasting Glucola followed by a 3-hour 100-g OGTT for clients who test positive for the 1-hour screening.

2. Hypertensive medications may need to be changed if considering pregnancy because ACE inhibitors, beta-blockers, and diuretics are contraindicated during pregnancy.

3. Increased monitoring of blood glucose is necessary during pregnancy.

B. Adults:

1. HgbA1C target recommendations:

a. American College of Physicians:

i. Clinicians personalize glycemic control in clients with type 2 diabetes and discuss benefits/harms of pharmacotherapy, client's preferences, client's life expectancy and general health, treatment burden, and cost of care.

ii. HgbA1C level aims between 7% and 8% in most clients with type 2 diabetes.

b. ADA:

i. HbA1C goal of less than 7% for most clients.

c. AACE and the American College of Endocrinology (ACE):

i. HbA1C goal of less than 6.5% for most clients.

C. Geriatrics:

1. Rates of major morbidity, including amputation, myocardial infarction (MI), blindness, and ESRD, and death are much higher in clients diagnosed with diabetes who are over 65 years of age. Complications for clients with diabetes increase at 75 years and older.

2. Diabetes is associated with the emergence/worsening of the following geriatric syndromes: cognitive dysfunction, functional impairment, falls and fractures, depression, and visual and hearing impairment. There is also strong evidence to indicate that adults with diabetes aged 65 years and older are a high-priority population for depression screening and treatment.

3. Older adult clients are sensitive to fluctuating blood glucose (severe highs and lows). Treat older adult clients with type 2 diabetes cautiously to control hyperglycemia. However, monitoring for signs and symptoms of hypoglycemia from overtreatment is also important.

4. Carefully monitor the HgbA1c of clients with a life expectancy less than 10 years due to advanced age (80 years or older), those residing in a long-term care facility, or those with chronic conditions (dementia, cancer, ESRD, chronic obstructive pulmonary disease [COPD], or HF) secondary to the harms outweighing the benefits to this population.

5. Glucose testing and treatment for geriatric clients:

a. Liberalization of control: Functional and cognitive status is an important consideration when determining goals for glycemic control in the older adult.

i. Functional and cognitively intact older adults with long life expectancy should have care and goals similar to those for younger clients with diabetes.

ii. Glycemic goals may be relaxed based on selected individuals, but hyperglycemia leading to symptoms or risk of acute hyperglycemic complications should be avoided.

iii. The HbA1C goal for healthy adults over age 65 is less than 7.5%.

iv. The HbA1C goal for older adults who have medical and functional comorbidities, are frail, and have a life expectancy less than 10 years is 8.0% or less.

v. The "old–old," those over 85 years of age, can be allowed to have HbA1C up to 8.5% as long as this preserves quality of life and avoids the complications of hypoglycemia.

b. Falsely high HbA1C: This may result from increased age of red cells when turnover/production is low, as in iron or nutrient deficiency anemia, or asplenia.

c. Falsely low HbA1C: This may result from more rapid cell turnover as in hemolysis, or in repletion of iron or other nutrients as red cell production is increased.

d. Chronic kidney disease can both falsely lower and raise HbA1C.

6. Treatment considerations:

a. Metformin is first-line therapy; however, it is contraindicated in clients with eGFR less than 30 mL/min/1.73 m^2.

b. Avoid SUs due to the risks of hypoglycemia. Glyburide should be avoided in older adults.

c. GLP-1 RAs and SGLT-2 inhibitors are options if metformin is inadequate. ▶

7. The ADA recommends the following:

a. Implement periodic individualized screening for diabetes and/or diabetic complications in adults older than 65 years. A full assessment of medical, psychological, functional, and social domains would provide a framework to determine individualized targets and decisions.

b. Consider revisiting self-management capabilities for the geriatric population and evaluating for any limitations in activities of daily living (ADLs).

c. Aspirin and lipid-lowering agents may benefit geriatric clients whose health presentation suggests longer life expectancies.

d. For a palliative/hospice geriatric client, consider hydration and discontinuation of all oral diabetic agents, except a low-dose basal insulin for type 1 diabetes clients.

e. Beers Criteria:

i. Glyburide and chlorpropamide may increase the risk of hypoglycemia in the geriatric client. An alternative would be glipizide.

ii. Insulin must be meticulously titrated to avoid severe rebound hypoglycemia. The sole use of sliding scale insulin (SSI) should be avoided because it is often difficult for older adults to manage multiple tests and injections, and SSI carries a higher risk of hypoglycemia without improvement in hyperglycemia management. SSI may be used in the short term for acute illness, or in titration of basal insulin, or when rapid-acting insulin is used as "correction" insulin in conjunction with scheduled insulin.

iii. Pioglitazone and rosiglitazone tend to increase fluid retention and worsen HF in geriatrics.

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GALACTORRHEA

DEFINITION

A. The production of a milky discharge excreted from the nipple occurring beyond the 6-month period of pregnancy and/or breastfeeding cessation.

INCIDENCE

A. It is estimated that 1% to 50% of reproductive females will experience galactorrhea at some time in their life.

PATHOGENESIS

A. The pathogenesis depends on the etiology. Physiologic galactorrhea is caused by pregnancy. The anterior pituitary gland secretes prolactin, which stimulates milk production. Milk production is normal for 6 months after pregnancy and/or after breastfeeding has ceased. The majority of cases are of benign etiology. Malignancy is responsible for 5% to 10% of cases. Malignancy evidenced by nipple discharge is more common after age 60.

PREDISPOSING FACTORS

A. Reproductive females.

B. Medications: oral contraceptions, pimozide, risperidone, molindone, olanzapine, clomipramine, desipramine, haloperidol (Haldol), metoclopramide (Reglan), cimetidine, isoniazid, Aldomet, reserpine, verapamil, codeine, morphine, and imipramine (Tofranil).

C. Lesions involving chest wall: breast surgery, burns, herpes zoster, spinal cord injury, trauma.

COMMON COMPLAINTS

A. Milky discharge from the nipple.

SUBJECTIVE DATA

A. Note the onset, duration, location (unilateral or bilateral), and course of presenting symptoms.

B. Ask whether the client has been pregnant and/or breastfed within the past 6 months. If so, how long did the client nurse?

C. Review their menstrual history and pattern.

D. Ask client to describe the discharge, noting color, consistency, and/or presence of blood.

E. Determine the mechanism of production of discharge: spontaneous or with manual expression.

F. Assess for any palpable mass in the breast and axillae.

G. Review current medications, including use of oral contraceptives.

H. Note any previous experience with galactorrhea. If so, discuss testing performed and treatment, if any.

I. Identify any family history of breast cancer or other tumors.

PHYSICAL EXAMINATION

A. Check pulse, respirations, and blood pressure.

B. Inspect:

1. Inspect the breast and nipples for symmetry.

2. Assess the discharge.

3. Inspect the skin; note dimpling, retraction, and irregularities.

4. Eyes: complete funduscopic examination.

5. Perform visual field testing.

▶

C. Palpate:
1. Palpate the breasts for masses and fibrocystic changes.
2. Squeeze the nipple to induce discharge.
3. Palpate the axillary lymph nodes.
4. Palpate the neck, thyroid, and lymph nodes.

DIAGNOSTIC TESTS
A. Prolactin level: Normal level is 1 to 20 ng/mL.
B. Thyroid-stimulating hormone (TSH).

Many cases of galactorrhea are considered idiopathic. Usually endocrine studies will be normal.

C. Beta human chorionic gonadotropin (beta HCG).
D. Hemoccult of breast discharge.
E. Breast discharge for pathology.
F. Periareolar ultrasound (all ages).
G. CT/MRI of sella turcica if pituitary mass is suspected.
H. Diagnostic mammogram in females older than 30 years if tumor is suspected.
I. Ductoscopy.
J. Skin punch biopsy for abnormal skin presentations.

DIFFERENTIAL DIAGNOSES
A. Fibrocystic disease.
B. Mastitis.
C. Breast tumor.
D. Medication induction.
E. Breast cancer: bloody nipple discharge, painless, firm fixed mass.
F. Pituitary adenoma: can produce permanent visual field loss and headaches.
G. Hypothalamic disorders.
H. Chiari–Frommel: galactorrhea occurring after 6 months postpartum.
I. Pituitary and thyroid disorders.
J. Pregnancy.

PLAN
A. General interventions:
1. Treat underlying cause of nipple discharge.
2. If induced by medications, consider stopping medications if side effect outweighs benefits.
3. If benign cause, no treatment is necessary with medication. Monitor symptoms. If symptoms progress, reevaluate.
4. If any of these are noted, clients must undergo a comprehensive evaluation:
a. Unilateral or blood nipple discharge.
b. Nipple discharge with a palpable mass or lesion.
B. Client teaching:
1. Teach self-examination of the breast.
C. Pharmaceutical therapy:
1. No pharmaceuticals are advised except for tapering or discontinuing medications that may be causing the discharge. This course is recommended only after cautious consideration of why the medication is being used (antipsychotics).
2. The American Academy of Family Physicians (AAFP) recommends the following if galactorrhea is caused by hyperprolactinemia and/or prolactinoma:
a. First choice of medication is cabergoline because it is considered to be more effective and better tolerated. Cabergoline 0.25 to 1 mg twice a week, or 0.5 to 2 mg once a week. Discontinue once pregnancy is confirmed.
b. Second choice is bromocriptine 2.5 to 15 mg daily. It is of lower cost, yet with higher incidence of side effects. Discontinue once pregnancy is confirmed.

FOLLOW-UP
A. Monitor prolactin level every 6 to 12 months.
B. Recommend yearly vision evaluation.
C. Order MRI at 1 year, then every 2 to 5 years if symptoms persist.

CONSULTATION/REFERRAL
A. Consult a physician regardless of normal imaging results if breast tumor is suspected with bloody discharge or palpable mass noted.

INDIVIDUAL CONSIDERATIONS
A. Pregnancy:
1. Galactorrhea during pregnancy is a normal physiologic response.
2. If galactorrhea persists after pregnancy/lactation has ceased for 6 months, a full workup evaluation is required.
B. Adults:
1. Males: Galactorrhea is rare in males; however, it can occur with prolactinoma.
C. Geriatrics:
1. Causes of galactorrhea include antipsychotic medications, renal insufficiency, or pituitary/hypothalamus disorders impairing dopamine secretion.

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GYNECOMASTIA

DEFINITION
A. Gynecomastia is an enlargement of the breast tissue in males.

INCIDENCE AND PREVALENCE
A. Common in newborns and will usually regress in 2 to 3 weeks after delivery, approximately 40% to 69% of adolescent males will experience breast enlargement. It is also seen in adult males (between the ages of 50 and 80) with excessive weight gain.

PATHOGENESIS
A. Male breast duct proliferation occurs due to a hormonal imbalance of estrogen and androgen levels. Pathologic conditions such as pituitary tumors, systemic disorders, chronic kidney disease, thyroid disorders, and liver disease can cause symptoms to occur. Certain drugs may also induce the development of gynecomastia. Potential medications include spironolactone, antiandrogens, antidepressants, cimetidine, ranitidine, ketoconazole, chemotherapeutic agents, amiodarone, diltiazem, nifedipine, digoxin, methyldopa, reserpine, hormones, and sedatives.

PREDISPOSING FACTORS
A. Age (males 50–80 years) with excessive weight gain.
B. Family history.
C. Malnutrition with severe weight loss. ▶

D. Obesity.
E. Peutz–Jeghers syndrome.
F. Hypogonadism (primary—testicular or secondary—central).
G. Thyroid disease, primarily thyrotoxicosis.
H. Estrogen or human chorionic gonadotropin (HCG) secreting tumors.
I. Medications associated with gynecomastia: antiandrogens, ketoconazole, metronidazole, calcium channel blockers, spironolactone, omeprazole, cimetidine, ranitidine, steroids, marijuana, methadone, antivirals, antipsychotics, statins/fibrates, and digitalis.

COMMON COMPLAINTS
A. Enlargement of breast tissue, usually bilateral, with or without discomfort.

OTHER SIGNS AND SYMPTOMS
A. Asymptomatic.
B. Type I: nodule present under areola tissue area.
C. Type II: nodule palpable under and beyond the areola area.
D. Type III: breast enlargement without contour separation of tissue.

SUBJECTIVE DATA
A. Identify when breast development first appeared.
B. Determine whether enlargement is unilateral or bilateral.
C. Review the progression of enlargement.
D. Note any pain, discharge, or masses that are palpable.
E. List current medications, drugs, and alcohol and substance abuse.
F. Review the client's medical history.
G. Note the client's family history of gynecomastia.
H. Explore nutritional intake.
I. Discuss the client's level of physical activity (sports, hobbies, etc.).
J. Note use of herbal products or anabolic steroids.

OBJECTIVE DATA
A. Inspect breasts bilaterally and surrounding nodes for enlargement or skin changes.
B. Palpate breast tissue systematically and surrounding nodes. Gynecomastia can usually be appreciated once the glandular tissue reaches 0.5 cm or larger in diameter.
C. Palpate testes for masses or atrophic changes.
D. Body mass index (BMI).
E. Assess blood pressure (BP).

DIFFERENTIAL DIAGNOSES
A. Obesity: fatty breast enlargement without glandular involvement.
B. Breast cancer: fixed, firm nodule in tissue with dimpling and/or breast discharge.
C. Neurofibroma.
D. Lipoma.

DIAGNOSTIC TESTS
A. Prolactin level.
B. Thyroid-stimulating hormone (TSH).
C. HCG.
D. Serum luteinizing hormone (LH).
E. Testosterone level.
F. Estradiol level.
G. Mammography for suspicious breast masses in adult males.

PLAN
A. General interventions:
  1. Identify any pathologic condition. If none is identified, reassure the client that normal resolution will occur over time.
B. Client teaching:
  1. Reinforce weight reduction if weight gain is a factor in the condition.
C. Pharmaceutical therapy:
  1. Antiestrogens (tamoxifen).
  2. Androgens (testosterone replacement).
  3. Aromatase inhibitors (anastrozole).
  4. Discontinue medications that may induce symptoms if possible.

FOLLOW-UP
A. Follow-up is dependent on etiology and/or client needs.

CONSULTATION/REFERRAL
A. Consult a physician or refer to an endocrinologist if the male client is noted to have breast enlargement for longer than 2 years.

INDIVIDUAL CONSIDERATIONS
A. Adults:
  1. Adult males diagnosed with prostate cancer who are treated with an antiandrogen monotherapy are at higher risk for the development of gynecomastia.
B. Geriatrics:
  1. In geriatric males, gynecomastia is a common finding secondary to a decrease in testosterone production and an increase in sex hormone binding globulin that lowers free testosterone levels. Testosterone treatment for geriatrics increases the risk of prostate cancer and cardiac events.

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HYPOGONADISM

DEFINITION
A. Hypogonadism in males is failure of the testis to produce physiologic levels of testosterone and a normal number of spermatozoa.

INCIDENCE
A. An estimated 40% of males older than 45 years of age have below-normal values of serum testosterone.

PATHOGENESIS
A. Hypogonadism in males can be the result of testicular dysfunction or nondevelopment (primary hypogonadism) or dysfunction of the pituitary or hypothalamus (secondary hypogonadism). The two clinical manifestations of impaired spermatogenesis are infertility and decreased testicular size. There are several possible clinical manifestations of ▶

testosterone deficiency, which are determined by its time of onset during reproductive development:

1. In utero first or second trimesters: incomplete virilization of external genitalia, incomplete development of Wolffian ducts to form male internal genitalia.
2. Third trimester in utero: micropenis.
3. Prepuberty: incomplete pubertal maturation, eunuchoidal body habitus, poor muscle development, and reduced peak bone mass.
4. Postpuberty: decreased energy, mood, and libido; reduced or zero sperm count; decrease in axillary and pubic hair, hematocrit, muscle mass and strength, and bone mineral density.

PREDISPOSING FACTORS

A. Hypogonadism associated with Klinefelter syndrome.
B. Chemotherapy.
C. Radiation therapy.
D. Excessive alcohol consumption.
E. Painful testicular swelling.
F. Anosmia associated with Kallmann syndrome.
G. Use of medications that cause hypogonadism: ketoconazole or extended-release opiates.

COMMON COMPLAINTS

A. Decreased vigor and libido.
B. Depressed mood.

OTHER SIGNS AND SYMPTOMS

A. Fatigue.
B. Difficulty concentrating.
C. Hot flashes.
D. No change in deepening of the voice.
E. Weight gain.
F. Signs of metabolic syndrome.
G. Erectile dysfunction.
H. Poor physical performance.

SUBJECTIVE DATA

A. Inquire about history of known chromosomal abnormalities in family or client.
B. Ask about history of cryptorchidism.
C. Is there any history of muscular weakness?
D. Is there a history of varicocele unresolved within 6 months of birth?
E. Inquire about known infections affecting the scrotum and testes.
F. Has the client had therapeutic radiation to the area or a history of chemotherapy?
G. Ask about use of long-term ketoconazole, glucocorticoids, or long-acting opiates.
H. Any known testicular trauma?
I. Is there any history of known torsion?
J. Evaluate for a history of autoimmune disorders and/or chronic illnesses including cirrhosis, chronic renal failure, or HIV.
K. Has the client noted a decrease in spontaneous erections?

PHYSICAL EXAMINATION

A. Inspect:
1. Testes for appropriate size.
2. Upper and lower body musculature.
3. Full/dense male-pattern beard.
4. Testes should be bilaterally descended.
5. Rule out eunuchoid appearance.
6. Breasts for gynecomastia.
7. Clinical findings of hypogonadism develop gradually and become obvious after puberty.
8. Penis for hypospadias.

B. Palpate:
1. Scrotum and testes for masses.
2. Breasts for masses (both male and females).

DIAGNOSTIC TESTS

A. Serum testosterone (morning measurements: 8:00–10:00 a.m.); repeat to confirm.
B. Measurement of free testosterone and sex hormone binding globulin (SHBG) if total testosterone is not near the lower limit.
C. Avoid lab tests during acute illness.
D. Measure SHBG if there are comorbidities and if clinical presentation is inconsistent with serum total testosterone.
E. If indicated, additional tests may be ordered: follicle-stimulating hormone (FSH), luteinizing hormone (LH), prolactin, thyroid-stimulating hormone (TSH), free T4, complete blood count (CBC), comprehensive metabolic panel (CMP), iron, transferrin, cortisol, and vitamin D levels.

DIFFERENTIAL DIAGNOSES

A. Hypogonadism.
B. Moderate obesity.
C. Nephrotic syndrome.
D. Hypothyroidism.
E. Use of glucocorticoids, progestins, and androgenic steroids.
F. Acromegaly.
G. Diabetes mellitus (DM).
H. Hepatic cirrhosis.
I. Hypopituitarism.
J. Malnutrition.
K. Klinefelter syndrome.
L. Depression.
M. Psychological sexual dysfunction.
N. Pituitary adenoma.

PLAN

A. General interventions:
1. Testosterone replacement's effect on reducing adverse health outcomes in the general population is unknown.
2. Testosterone levels vary significantly with circadian rhythms, illness, and medications.
3. Measurement of bone mineral density is recommended to assess fracture risk.
4. LH and FSH concentrations can help distinguish between primary and secondary hypogonadism.
5. Differentials for secondary hypogonadism my include pituitary neoplasia, hyperprolactinemia, hemochromatosis, HIV, obstructive sleep apnea (OSA), and genetic disorders.
6. Testosterone replacement should be initiated only after a comprehensive evaluation of no contraindications and obtaining baseline levels of hemoglobin, hematocrit, prostate-specific antigen (PSA), and digital rectal exam (DRE). For the first year of treatment, testosterone (morning level), liver function tests, PSA level, lipid profile, hemoglobin, hematocrit, and DRE should be followed again in 3 to 6 months once replacement therapy started, then annually thereafter. ▶

B. Client teaching:

1. Individuals with hypogonadism can lead normal lives with hormone replacement therapy.

2. Hormone replacement may continue throughout life with ongoing monitoring of the potential risks and benefits.

3. Potential warnings with testosterone replacement therapy should be discussed prior to therapy, including gynecomastia, worsening benign prostatic hyperplasia (BPH) and dyslipidemia, sleep apnea, peripheral edema, as well as increased risk for myocardial infarction (MI) and cerebrovascular accident (CVA) from polycythemia.

4. Clients using topically absorbed testosterone gel or creams need to be warned of the possibility of transferring testosterone to female partners or children by direct skin-to-skin contact.

5. Reduced sperm production and fertility are potential side effects of testosterone replacement.

C. Pharmaceutical therapy:

1. Injectable testosterone replacement: 50 to 100 mg weekly or 100 to 200 mg intramuscular (IM) every 2 weeks.

2. 1% testosterone gel: AndroGel, Testim, Axiron.

3. Transdermal testosterone patch.

4. Buccal testosterone.

5. Implanted subcutaneous testosterone pellets.

6. Avoid testosterone therapy in the following clients: males who are planning fertility measures, breast/prostate cancer, elevated hematocrit, thrombophilia, severe multiple urinary tract infections (UTIs), untreated OSA, uncontrolled heart failure, and/or stroke/MI within the last 6 months.

FOLLOW-UP

A. Clients receiving hormone replacement should be reevaluated every 3 to 6 months or more frequently, including screening for prostate cancer and monitoring of potential risks.

B. Routine monitoring should be conducted to evaluate symptoms, screen for adverse effects to testosterone therapy, determine adherence to regimen, and obtain hematocrit levels, cholesterol panel, and prostate function.

C. Testosterone replacement is contraindicated in prostate cancer, breast cancer, baseline hematocrit over 48% prior to treatment, PSA above 3 ng/mL in high-risk clients, undiagnosed prostate nodules, and uncontrolled heart failure.

CONSULTATION/REFERRAL

A. Physician consultation prior to initiating testosterone therapy is advised.

B. Endocrinology referral is recommended in clients not responsive to the replacement therapy.

C. Clients with primary hypogonadism should be referred to an endocrinologist for initial workup and management.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Current recommendations are not in favor of testosterone therapy for all older clients. Providers should cautiously consider the benefits and risks for older individuals. The benefits of testosterone replacement are unproven, and the long-term risks are unknown.

B. HIV clients:

1. Short-term testosterone replacement should be considered for HIV males with low testosterone, weight loss, and muscular wasting.

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METABOLIC SYNDROME/INSULIN RESISTANCE SYNDROME

DEFINITION

A. Metabolic syndrome is an association of several complex disorders with major features that include central obesity, hyperglycemia, hypertension (HTN), and hyperlipidemia. The coexistence conditions increase the development of atherosclerotic cardiovascular disease (ASCVD) and diabetes mellitus. Metabolic syndrome is considered a proinflammatory and prothrombotic state. Elevated triglycerides and low high-density lipoprotein (HDL) cholesterol are strong predictors of vascular events. Triglycerides and the waist circumference are considered the strongest predictors for the development of metabolic syndrome.

B. Currently, there is no unifying criteria for metabolic syndrome. According to the National Cholesterol Adult Treatment Panel (ATP) III, metabolic syndrome in adults is the coexistence of any three of five conditions (Table 24.3).

C. Complications associated with metabolic syndrome may include type 2 diabetes mellitus (T2DM), coronary artery disease (CAD), fatty liver disease, cirrhosis, chronic kidney disease, polycystic ovarian syndrome (PCOS), obstructive sleep apnea (OSA), and gout. ▶

TABLE 24.3 ADULT TREATMENT PANEL III CRITERIA FOR METABOLIC SYNDROME

Metabolic Syndrome Traits	Definition
Central/abdominal obesity	Adult waist circumference: males ≥40 in. (102 cm); females ≥35 in. (88 cm)
Serum triglycerides	Adults: ≥150 mg/dL or drug treatment for elevated triglycerides
Serum HDL cholesterol	Adults: <40 mg/dL in males; <50 mg/dL in females
Blood pressure	Adults: ≥130/85 or drug treatment for hypertension
FPG	Adults: ≥100 mg/dL or drug treatment for elevated blood glucose

FPG, fasting plasma glucose; HDL, high-density lipoprotein.

Source: From National Institutes of Health. (2002). *Third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults* (Adult Treatment Panel III): Final report. Bethesda, MD: Author.

D. Metabolic syndrome is noted in the literature under other names, including insulin resistance syndrome and obesity dyslipidemia syndrome. Previously, the term "Syndrome X" was used; however, Syndrome X is noted in those with angina that have normal coronary arteries.

INCIDENCE AND PREVALENCE

A. The prevalence of metabolic syndrome increases with age. Overall the prevalence is estimated at 34.7% in the United States.

B. Mexican Americans have the highest age-adjusted prevalence. Among African Americans and Mexican Americans, the prevalence is higher in females.

C. Ethnic background:

1. Native Americans are at the greatest risk.

2. Mexican Americans have the highest prevalence (31.9%).

3. Black and Hispanic females are 1.5 times more likely than non-Hispanic White females.

PATHOGENESIS

A. The exact etiology is unknown; however, abdominal obesity has been associated with insulin resistance. Vascular endothelial dysfunction occurs secondary to insulin resistance, hyperglycemia, hyperinsulinemia, and adipokines. Along with high blood pressure and abnormal lipids, vascular inflammation places the individual at high risk for a cardiovascular insult.

PREDISPOSING FACTORS

A. Genetic predisposition.

B. Weight gain, especially central/abdominal obesity.

C. Females, especially postmenopausal.

D. Smoking.

E. High-carbohydrate diet, especially soft drink consumption.

F. Lack of exercise:

1. Sedentary lifestyle.

2. Too much screen time (television or electronic device).

G. Insulin resistance.

COMMON COMPLAINTS

A. Complaints are all related to the individual coexisting comorbid symptoms.

OTHER SIGNS AND SYMPTOMS

A. All are related to the individual coexisting comorbid symptoms.

SUBJECTIVE DATA

A. Review the client's medical history related to comorbid conditions, including obesity, HTN, and any abnormal laboratory testing (lipids, triglycerides, and glucose tolerance tests [GTTs]).

B. Review the client's family history including T2DM.

C. Review all prescription medications, over-the-counter (OTC) drugs, and herbals.

D. Review the client's current level of exercise.

E. Review the client's usual diet (24-hour recall), noting high fat and high glucose consumption.

F. Review the client's reproductive history.

PHYSICAL EXAMINATION

A. Check height, weight, waist circumference, blood pressure (BP), pulse, and respirations.

B. Calculate the body mass index (BMI) and waist-to-hip measurement. The Centers for Disease Control and Prevention (CDC) and the National Institutes of Health (NIH) websites have BMI, body fat, and waist-to-hip ratio calculators.

C. Make observations for acanthosis nigricans, peripheral neuropathy, retinopathy, and skin tags signifying insulin resistance.

D. A comprehensive physical examination is guided by the client's medical history and presenting signs and symptoms. Xanthomas may be present in those with dyslipidemia.

DIAGNOSTIC TESTS

A. Fasting glucose and/or hemoglobin A1C.

B. Fasting lipid panel.

C. Triglycerides.

D. Consider thyroid function and basic metabolic panel.

E. Consider C-reactive protein (CRP; optional) and liver panel.

F. EKG and other cardiac imaging studies if indicated.

DIFFERENTIAL DIAGNOSES

A. Cushing syndrome.

B. Hypothyroidism.

C. OSA.

D. **Hyperlipidemia**.

PLAN

A. General interventions:

1. Aggressive lifestyle modification focusing on increased physical activity and weight reduction is a cornerstone of treatment.

B. Client teaching:

1. Dietary recommendations include a low-fat, low-cholesterol, and/or dietary approach to stop HTN (Dietary Approaches to Stop Hypertension [DASH] diet). Reduce the consumption of simple sugar, saturated and trans fats, and cholesterol (see Appendix B, "Dietary Recommendations," for dietary information).

2. Exercise recommendations include a minimum of 30 minutes a day of walking at a brisk pace or other activity at a moderate intensity. Start by using a pedometer, walking at breaks, or household work.

3. Weight loss of 5% to 10% or more should be encouraged; gradual weight loss of 1 to 2 kg/mo. Even small losses are associated with health benefits.

4. BP control strategies include a low-sodium diet (DASH), smoking cessation, and alcohol in moderation.

5. Counsel on smoking cessation, if applicable.

6. Abdominoplasties do not lower the risk for CAD or improve insulin sensitivity.

C. Pharmaceutical therapy:

1. Currently, the treatment for metabolic syndrome is to treat each individual component/diagnoses for the individual.

2. Insulin-resistant clients with impaired glucose testing may benefit from metformin.

3. Statins for elevated lipids; aggressive treatment if ASCVD risk score is greater than 7.5%.

4. A low-dose aspirin may be prescribed related to the client's risk of cardiovascular diseases (CVDs) or prothrombotic state.

5. Oral hypoglycemic agents used to treat type 2 diabetes are not currently recommended for prevention of metabolic syndrome unless the client has an impaired fasting glucose or abnormal glucose tolerance testing.

6. HTN should be controlled with appropriate antihypertensives.

FOLLOW-UP

A. Follow-up involves assessment of metabolic syndrome at a regular intervals for anyone with one or more risk traits. Follow-up testing includes the following:

1. BMI calculation.
2. Waist-to-hip calculation.
3. Fasting lipid profile.
4. Fasting glucose or other hyperglycemia screening methods.
5. BP.

CONSULTATION/REFERRAL

A. Refer to an obstetrician/gynecologist for consultation and management for infertility/pregnancy.

B. Refer to a specialist for any comorbid condition as needed.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:

1. Metabolic syndrome greatly increases the risk of CVD and diabetes mellitus. Monitoring food choices, weight, and activity/exercise must be added to treatment regimens, lab evaluations, and preventive measures.
2. Traditional risk factors of metabolic syndrome for the general population may differ with geriatrics. Current research is evaluating the "reverse epidemiology" or "risk factor paradox" where a higher BMI, mild hyperlipidemia, and HTN suggest a better survival during aging. Some studies have shown that a low BP, BMI, and cholesterol give heart failure (HF) clients a worse prognosis. Currently this intriguing phenomenon is continuing to be investigated.

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OBESITY

DEFINITION

A. Obesity is a multifactorial disease with physical, psychological, and social consequences. Body mass index (BMI) is a standard measuring tool. BMI is calculated by using the formula weight in kilograms divided by height in meters squared (weight [kg]/height [m]^2). In adults, obesity is defined by a BMI greater than 30 kg/m^2 (Table 24.4).

INCIDENCE AND PREVALENCE

A. Obesity is a chronic condition that is considered a global epidemic. In the United States, the overall prevalence is estimated at 42.4%.

B. Obesity is higher among middle-aged adults (40–59 years; 44.8%) and older adults (≥60 years; 42.8%) than younger adults (20–39 years; 40%).

PATHOGENESIS

A. Numerous factors contribute to the development of obesity, including the following:

1. Imbalance between energy intake and energy output.
2. Genetics (40%–70% presumed explanation).
3. Environmental factors.
4. Drug-induced obesity:
   a. Tricyclic antidepressants.
   b. Antipsychotics.
   c. Sulfonylureas.
   d. Anticonvulsants (sodium valproate, carbamazepine).
   e. Glucocorticoids.
5. Sleep disturbance-induced obesity.

TABLE 24.4 ADULT OBESITY BY BMI

Classification of adult obesity by BMI	BMI (kg/m^2)
Underweight	<18.5
Normal	18.5–24.9
Overweight (preobese)	25.0–29.9
Obesity	30.0–34.9
Severely obese	>40.0
Morbidly obese	40.0–49.9
Super obese	>50.0
SSO	>60.0

BMI, body mass index; SSO, super-super obese.

PREDISPOSING FACTORS

A. Consuming too many calories/high-fat diet.

B. Poor dietary choices.

C. Readily available food sources, especially fast foods.

D. Lack of exercise/sedentary lifestyle.

E. Decreased/elimination of physical education requirements in public schools.

F. Television, computer, and use of handheld devices for more than 3 hours a day.

G. Increased leisure time.

H. Lack of funding and planning for community parks and recreation areas.

I. Ethnic background: African American, Hispanic.

J. Family history of obesity.

K. Pregnancy.

L. Insomnia, difficulty staying asleep, and frequent wakefulness.

COMMON COMPLAINTS

A. Difficulties with activities of daily living (ADLs) or functional impairment.

B. Lack of interest/inability to tolerate exercise.

C. Shortness of breath and/or asthma exacerbations.

D. Difficulty with personal hygiene.

E. Urinary incontinence.

F. Desire to lose weight.

OTHER SIGNS AND SYMPTOMS

A. Obstructive sleep apnea (OSA).

B. Increased asthma symptoms.

C. Infertility/polycystic ovarian syndrome (PCOS).

D. Symptoms associated with cholelithiasis.

E. Hypertension (HTN).

F. Early sexual maturity in females.

G. Other complications: type 2 diabetes, gallbladder disease, depressions/anxiety, osteoarthritis (cartilage and joint breakdown of lower extremities), dyslipidemia, stroke, coronary artery disease (CAD), low quality of life.

SUBJECTIVE DATA

A. Review onset of weight gain and duration of obesity. Identify when the client first noticed the weight gain.
B. Ask the client about other symptoms secondary to obesity.
C. Review full medical history.
D. Review medications, including over-the-counter (OTC) herbals and diet products.
E. Review the client's history of weight loss attempts.
F. Assess ADL and function limitations and the presence of exercise intolerance.
G. Elicit history of sleep disorders (e.g., snoring and obstruction, sleep apnea).
H. Review 24-hour dietary recall. Review the client's normal average meals per day, including snacks.
I. Review consumption of high-caloric drinks and alcohol intake.
J. Assess for history of binge eating, purging, lack of satiety, food-seeking behaviors, and other abnormal eating habits.
K. Assess for depression.
L. Assess for readiness and commitment to weight loss. People who voluntarily enroll in a weight-loss program generally lose weight.
M. Elicit client's activity level, exercise routine, and daily activity (work activity).

PHYSICAL EXAMINATION

A. Check pulse, respirations, and blood pressure (blood pressure)—supine, sitting, and standing.
B. Measure height and weight to calculate BMI.
C. Measure waist and hip circumferences to calculate the waist-to-hip circumference ratio. The waist-to-hip ratio is the strongest anthropometric measure that is associated with myocardial infarction risk and is a better predictor than BMI. A waist-to-hip ratio that is greater than 0.8% usually has some form of premetabolic syndrome or insulin resistance.
D. Inspect:
  1. Observe the overall appearance and note body fat distribution.
  2. Examine the skin.
  3. Mouth and teeth: Assess dental enamel for signs of purging.
E. Auscultate:
  1. Carotid arteries.
  2. Heart.
  3. Lungs.
  4. Abdomen.
F. Palpate:
  1. Neck and thyroid.
  2. Palpate the extremities: Note edema.

DIAGNOSTIC TESTS

A. Thyroid function.
B. Lipid panel.
C. Triglycerides.
D. Pregnancy test.
E. Fasting blood sugar/3-hour glucose tolerance test (GTT).
F. Sleep study (if indicated).
G. Nocturnal hypoxemia study.

DIFFERENTIAL DIAGNOSES

A. Obesity.
B. Pseudotumor cerebri.
C. Binge eating.
D. Genetic syndrome (e.g., Prader–Willi syndrome).
E. Cushing syndrome.
F. Diabetes mellitus (DM).
G. Insulin resistance syndrome.
H. Primary pulmonary HTN.

PLAN

Manage obesity as a chronic relapsing disease, including the comanagement of other diseases secondary to obesity (e.g., diabetes, HTN):

A. General interventions:
  1. Reinforce the positive impact that weight-loss measures (diet, exercise) can have and the overall health benefits of weight loss.
  2. Identify and monitor any cardiovascular complications.
  3. Behavior modification: Intensive behavioral therapy has been shown to increase weight-loss success and to help achieve sustainable weight loss for longer periods of time. Behavioral therapy includes weekly meeting with healthcare professionals for at least 6 to 8 weeks:
    a. Dietary plan:
      i. Low-calorie diet.
      ii. Increase intake of fruits and vegetables.
      iii. Eliminate alcohol and beverages containing sugar.
      iv. Reduction of high-glycemic foods such as candy.
      v. Reduction in high-caloric foods and drinks.
      vi. Reduction of fat intake.
      vii. Reduction of portion sizes.
      viii. Reduction of the amount of processed foods in the diet.
      ix. Increase in water intake.
    b. Exercise:
      i. U.S. Department of Health and Human Services (HHS) (2019) physical activity guidelines recommend that adults must achieve at least 150 to 300 minutes (2½–5 hours) of moderate-intensity aerobic physical activity per week. Strengthening exercises (push-ups, sit-ups, lifting weights) at least two times a week are also recommended. There is a wide range of benefits, including the following:
        1) Helps manage BP.
        2) Improves cholesterol.
        3) Helps manage glycemic control in diabetes.
        4) Helps strengthen bones.
        5) Promotes weight loss.
        6) Improves depression.
        7) Boosts immune system.
        8) Reduces stress.
        9) Improves a sense of well-being.
        10) Believed to be a major driving force in lifestyle change.
      ii. Obtain counseling on stimulus control, goal setting, self-monitoring, and contracts that reward behaviors.
      iii. Contraindications to exercise:
        1) There are several contraindications to beginning exercise: individuals with recent myocardial infarction (2 weeks), unstable angina, severe aortic stenosis, decompensated congestive heart failure (low ejection fraction), left ventricular outflow obstruction, uncontrolled dysrhythmias, uncontrolled diabetes or diabetic complications, and uncontrolled HTN. ▶

B. Client teaching:

1. Keep a food diary to identify food triggers.

2. Stress to the client the temporary nature of the weight-loss medications. Counsel about drug side effects and the lack of long-term safety data. Typical weight loss is modest, less than 5 kg (10–11 lbs) at 1 year.

C. Pharmaceutical therapy:

1. After an adequate trial (minimum of 6 months) of diet and exercise therapy, consider pharmaceutical therapy. Pharmacologic intervention is not allowed in some states. State statutes should be considered prior to prescribing weight control products.

2. Studies lack evidence to support whether one drug is more efficacious than another, nor does the literature support use of combination therapy for increased weight loss.

3. The choice of a pharmaceutical agent depends on the side effect profile of the drug and tolerance of the side effects.

4. The Food and Drug Administration (FDA) has not approved any weight-loss medication for use beyond 2 years in adults. Appetite suppressants that are FDA-approved:

a. Qsymia, phentermine, diethylpropion, benzphetamine, and phendimetrazine are approved for short-term (12 weeks) use.

b. Sibutramine (Reductil) is FDA-approved for 1-year use.

c. Orlistat (Xenical) is FDA-approved for 2-year use.

d. Naltrexone HCl/bupropion HCl (Contrave) is FDA-approved for 1-year use.

e. Liraglutide injection (Saxenda): a glucagon-like-peptide FDA-approved for weight loss.

5. Appetite suppressants:

a. Qsymia (phentermine and topiramate extended-release): Start with 3.75/23 mg extended-release per day for initial BMI greater than 30 kg/m^2, or BMI greater than 27 kg/m^2 in the presence of risk factors. May gradually increase dose to 15/92 mg:

i. Avoid evening dose.

ii. Monitor for hypersensitivity to phentermine and Topamax.

iii. These drugs are not recommended in the presence of HTN, hyperthyroidism, cardiovascular disease (CVD), and drug or alcohol abuse.

iv. Do not use if history of glaucoma, hyperthyroidism, or within 14 days of monoamine oxidase inhibitors (MAOIs).

v. Gradually discontinue medication if weight loss is not at least 5% of baseline.

b. Phentermine (Adipex-P) 37.5 mg orally once daily before or 1 to 2 hours after breakfast, or 18.75 mg one to two times per day for initial BMI greater than 30 kg/m^2, or BMI greater than 27 kg/m^2 in the presence of risk factors:

i. Avoid late-evening dosing.

ii. Not recommended in the presence of HTN, hyperthyroidism, CVD, and drug or alcohol abuse.

iii. Do not prescribe during or within 14 days of MAOIs.

c. Benzphetamine (Didrex) 25 to 50 mg orally initially once in the midmorning or midafternoon. Increase if needed to 25 to 50 mg one to three times a day:

i. Not recommended in the presence of HTN, hyperthyroidism, CVD, and drug or alcohol abuse.

ii. Not to be prescribed during or within 14 days of MAOIs.

iii. Pregnancy category X: known to cause fetal abnormalities or toxicity in animal and human studies.

d. Diethylpropion (Tenuate) 25 mg (immediate-release) one tablet Q8H, 1 hour before meals. May add one additional dose for night hunger. Half-life of 4 to 6 hours:

i. Avoid late-evening dosing.

ii. Not recommended in the presence of HTN, hyperthyroidism, CVD, and drug or alcohol abuse.

iii. Do not prescribe during or within 14 days of MAOIs.

e. Phendimetrazine (Bontril PDM) 35 mg orally two or three times daily 1 hour before meals. May reduce to 17.5 mg/dose (half a tablet). Maximum dose 70 mg TID. Also available in extended-release capsule 105 mg in the morning 30 to 60 minutes before breakfast:

i. Not recommended in the presence of HTN, hyperthyroidism, CVD, and drug or alcohol abuse.

ii. Not to be prescribed during or within 14 days of MAOIs.

f. Naltrexone HCl/bupropion HCl (Contrave) is a combination of an opiate antagonist and antidepressant. The synergistic effect reduces appetite:

i. Opioids should not be used 7 to 10 days before starting Contrave.

ii. Take medication with low-fat meal BID.

iii. Do not take close to bedtime to avoid insomnia side effect.

iv. Do not crush because a quick release would increase risk of seizure side effect.

g. Liraglutide (Saxenda) is FDA-approved for adults with a BMI ≥27 who also have weight-related medical problems:

i. Saxenda comes in a prefilled pen.

ii. It is not a treatment for type 2 diabetes.

iii. Should not be used with Victoza, insulin, or other glucagon-like peptide-1 receptor agonist medicines.

iv. Should not be prescribed to clients with thyroid tumors/cancer.

v. Monitor for potential hypoglycemia in type 2 diabetics.

vi. Recommended dose: Initiate at 0.6 mg subcutaneous once daily for 1 week and increase in increment of 0.6 mg every week until 3 mg daily is reached. Administer at any time without regard to meals.

h. Lorcaserin HCl (Belviq) was withdrawn in the United States in 2020 due to increased occurrence of cancer.

6. Lipase inhibitor:

a. Orlistat (Xenical) for use with a low-fat diet. Recommend 30% of calories spread over three main meals:

i. Take one 120 mg capsule orally during or up to 1 hour after main meals up to three times per day.

ii. If a meal is missed or had no fat, skip dosage.

iii. May decrease absorption of fat-soluble vitamins and beta carotene.

iv. Supplement diet with a multivitamin.

v. FDA-approved for up to 2 years of use in adults. ▶

vi. Gastrointestinal (GI) side effects include fatty/oily stools, oily spotting, flatus with discharge, fecal urgency, and fecal incontinence.
vii. Contraindicated in chronic malabsorption syndrome and cholestasis.
viii. May affect doses for antidiabetic medications.
ix. Monitor warfarin and cyclosporine levels.

b. Alli, a lipase inhibitor, is the only FDA-available OTC weight-loss product.

7. Off-label medications used for obesity:

a. Metformin (Glucophage) is used to decrease central adiposity in weight loss, lower insulin levels, and slow down the process of gluconeogenesis:
i. Start metformin 500 mg at the evening meal. The dosage can be increased by 500 mg/wk in divided doses up to the maximum of 2,000 mg/d.
ii. Titrate slowly due to the GI side effects.
iii. Check a metabolic panel before and every 3 to 6 months to evaluate for lactic acidosis.
iv. Metformin is contraindicated in clients with renal impairment; assess renal function prior to instituting metformin and monitor regularly.
v. Metformin must be stopped prior to any procedure with radiographic dye.
vi. Start on a client with a waist-to-hip ratio greater than 0.8%.

b. Topiramate (Topamax) is used to treat seizures and several types of headache. In small doses, it can be used alone or adjunct with phentermine to suppress appetite longer. Be familiar with the risks associated with the use of Topamax:
i. If used alone, may start at 25 mg every day in the morning. Increase up to two or three times a day. Topamax has a longer half-life of 19 to 25 hours.
ii. May be used as adjunct with phentermine. Start client at 18.75 mg of phentermine and 12.5 mg of Topamax. Then gradually increase the dose to 37.5 mg of phentermine and 25 mg of Topamax.
iii. Always give the phentermine in the morning, preferably 30 minutes before meals. Topamax should be dosed in the afternoon or evening.
iv. Phentermine (Adipex-P) 37.5 mg orally once daily before or 1 to 2 hours after breakfast, or 18.75 mg one to two times per day for initial BMI greater than 30 kg/m^2, or BMI greater than 27 kg/m^2 in the presence of risk factors.
v. Avoid late-evening dosing.
vi. Not recommended in the presence of HTN, hyperthyroidism, CVD, and drug or alcohol abuse.
vii. Do not prescribe during or within 14 days of MAOIs.

FOLLOW-UP

A. Reevaluate the client every week for 6 to 8 weeks; then monthly either with or without drug therapy until goal is achieved.

B. Maintain the recommended follow-up schedule for comorbid conditions.

C. If client is a candidate for bariatric surgery, follow recommended pretreatment/reauthorization guidelines required by the payer and bariatric center.

CONSULTATION/REFERRAL

A. Refer to a nutrition/registered dietitian consultation.

B. Consider a referral to a bariatric center/surgical consultation and evaluation of bariatric surgery.

C. Consider a psychology consultation (may be required prior to bariatric surgery).

D. If the family is eligible, refer to the Women, Infant, and Children (WIC) program.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Benzphetamine (Didrex) is a category X drug. Avoid use during pregnancy.
2. Counsel clients regarding appropriate weight gain with pregnancy.

B. Geriatrics:
1. All adults should avoid inactivity. Some exercise is better than none.
2. Create and monitor a diet plan specific to the clients that avoids poor food choices and promotes good choices for "cravings" and healthy eating. Studies suggest that the least restrictive diets customized to the client's preferences will foster the energy and motivation for mobility, respiratory improvement, and facilitate immunocompetence and wound healing.
3. In geriatrics, waist circumference is more appropriate than BMI for adiposity evaluation.
4. Qualitative studies reveal that many older adults desire to discuss weight-loss medications and elective corrective surgery. Must consider the benefits versus risks of weight-loss programs, medication regimens, and bariatric surgeries.

RESOURCES

President's Council on Sports, Fitness and Nutrition: www.hhs.gov/fitness/index.html.
Centers for Disease Control and Prevention: Overweight and obesity resources: www.cdc.gov/obesity.
Exercise: A Guide from the National Institute on Aging: www.nia.nih.gov/health/exercise-physical-activity.
National Institutes of Health: Exercise Videos: Exercise and Physical Activity for Older Adults: go4life.nia.nih.gov/exercise-videos.
American Heart Association: www.goredforwomen.org/live-healthy/betteru-nutrition-and-fitness-program-make-over-your-heart/nutrition-betteru-program-improves-health.
Apps: MyFitnessPal is a useful app to track calories, exercise activity, and weight loss. Establish goals and projected weight loss based on food intake and exercise activity: www.myfitnesspal.com.
World Health Organization Healthy Diet. The site has access to posters and templates including key facts, global strategies, malnutrition prevention, international foods standards, physical activity, and actions for health policies: www.who.int/mediacentre/factsheets/fs394/en.

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POLYCYSTIC OVARIAN SYNDROME

DEFINITION

A. Polycystic ovarian syndrome (PCOS), previously called Stein–Leventhal syndrome, is characterized by ovulatory dysfunction and hyperandrogenism. PCOS is a risk factor for metabolic syndrome, infertility, glucose intolerance, and type 2 diabetes mellitus (DM). PCOS itself is not considered a disease but a syndrome of coexisting conditions (see Table 24.5). ▶

TABLE 24.5 DIAGNOSIS OF PCOS FROM THE ANDROGEN EXCESS AND PCOS SOCIETY

Clinical hyperandrogenism	Hirsutism Acne vulgaris Alopecia
Biochemical hyperandrogenemia	Total testosterone or free testosterone with or without mild elevation of DHEA
Clinical ovulatory dysfunction	Menstrual irregularity, amenorrhea, anovulation
Morphologic ovulatory dysfunction	Polycystic ovaries (ultrasound or imaging)

DHEA, dehydroepiandrosterone; PCOS, polycystic ovarian syndrome.

Source: From Androgen Excess and Polycystic Ovarian Syndrome Society. (2021). Polycystic ovarian syndrome. https://ae-society.org/clinicians/.

B. The Androgen Excess and PCOS Society (AES) diagnostic criteria for PCOS:

1. Hyperandrogenism (clinical and/or biologic) and ovarian dysfunction (clinical and/or dysmorphology).

2. Exclusion of other androgen excess or related disorders.

3. Although obesity is one of the hallmarks of PCOS, lean females may also have insulin resistance/PCOS. The diagnosis of PCOS is based on medical history, physical examination, and laboratory tests. Other etiologies (thyroid dysfunction, nonclassic congenital adrenal hyperplasia [CAH], hyperprolactinemia) must be excluded. Lifestyle modification is the first-line approach in all females with PCOS.

INCIDENCE AND PREVALENCE

A. The prevalence of PCOS ranges from 5% to 15% worldwide. PCOS is one of the most common causes of infertility and endocrinopathy in reproductive females, affecting approximately 5 to 7 million females in the United States.

PATHOGENESIS

A. The exact etiology is unknown; however, PCOS is noted to have insulin resistance and abnormal pituitary function, as well as abnormal steroidogenesis.

PREDISPOSING FACTORS

A. Obesity and/or insulin resistance.
B. Genetic predisposition.
C. Metabolic syndrome.
D. Females with oligo-ovulatory infertility.
E. Type 1, type 2, or gestational diabetes.
F. History of premature adrenarche.
G. First-degree relatives with PCOS.
H. Antiepileptic medications.

COMMON COMPLAINTS

A. Hirsutism.
B. Menstrual problems.
C. Obesity.
D. Infertility.

OTHER SIGNS AND SYMPTOMS

A. Acne.
B. Alopecia (male pattern).
C. Hyperhidrosis.
D. Acanthosis nigricans.
E. Seborrhea.
F. Depressive symptoms.

SUBJECTIVE DATA

A. Review the client's menstruation history:

1. Premature puberty (younger than the age of 8).

2. Primary amenorrhea: lack of menses by age 15.

3. Oligomenorrhea: missing four periods per year.

4. Dysfunctional uterine bleeding (DUB): bleeding at irregular intervals, heavy cycles, periods longer than 7 days.

5. Subfertility or infertility.

B. Review the client's history of weight gain, increased waist circumference, and obesity.

C. Review the client's history of any skin/hair changes.

D. Psychological: depression, anxiety, psychosexual dysfunction, eating disorders.

E. Metabolic: diabetes, dyslipidemia, hypertension (HTN).

F. Review the family history for presence of diabetes, metabolic syndrome, obesity, and infertility.

PHYSICAL EXAMINATION

A. Check height, weight, waist circumference, blood pressure, pulse, and respirations.

B. Calculate the body mass index (BMI) and waist-to-hip measurement. The Centers for Disease Control and Prevention (CDC) and the National Institutes of Health (NIH) websites have BMI, body fat, and waist-to-hip ratio calculators.

C. Inspect:

1. Skin:

a. Evaluate for signs of hirsutism (upper lip, chin, nape of the neck, periareolar, abdomen-linea alba).

b. Pigmentation patterns for acanthosis nigricans (neck, axilla).

2. Observe fat distribution.

D. Pelvic examination:

1. Perform pelvic examination to evaluate enlarged ovaries and pelvic masses.

DIAGNOSTIC TESTS

A. Fasting glucose, oral glucose tolerance test (OGTT), or hemoglobin A1C to screen for hyperglycemia.

B. Thyroid function tests (thyroid-stimulating hormone [TSH], free T4).

C. Random serum cortisol to rule out Cushing syndrome.

D. 24-hour urine collection for free cortisol if there are clinical features suggestive of Cushing syndrome.

E. Serum luteinizing hormone (LH), follicle-stimulating hormone (FSH), and prolactin to rule out hypothalamic and pituitary diseases.

F. Ultrasound to rule out ovarian pathology (polycystic ovaries on ultrasound are not required for definitive diagnosis). Ultrasound diagnosis: when 10 small antral follicles are seen bilateral ovaries; unilateral is rare yet clinically significant. For adolescents and young females, ultrasound is not a reliable diagnostic test secondary to studies indicating that up to 70% of very young females may display polycystic ovaries on ultrasound.

G. Insulin-like growth factor (IGF-I).

H. Dehydroepiandrosterone sulfate (DHEA-S) to rule out adrenal hyperandrogenism. ▶

I. Free and total testosterone. Hormonal contraceptive use affects free testosterone; if appropriate, reassess after contraceptive has been discontinued for 3 months. Discuss alternate contraception if necessary.
J. 17-hydroxyprogesterone (17-OHP) for nonclassic CAH.
K. Lipid profile.
L. Endometrial biopsy if indicated for clients without menses for 1 year.
M. Pregnancy test prior to start of pharmaceutical therapy and history of an ovulation.
N. Serum 17-OHP.
O. Positive for anti-Müllerian hormone.

DIFFERENTIAL DIAGNOSES

A. Familial hirsutism.
B. Adrenal disorders:
  1. CAH.
  2. Cushing syndrome.
  3. Cortisol resistance.
C. Hyperprolactinemia.
D. Acromegaly.
E. Insulin resistance (types 1 and 2 diabetes).
F. Thyroid dysfunction.
G. Virilizing tumors.
H. Drug-induced:
  1. Anabolic steroids.
  2. Valproic acid.

PLAN

A. General interventions:
  1. Lifestyle modification focusing on increased physical activity and weight reduction, especially in overweight and obese individuals, is a first-line intervention.
B. Client teaching:
  1. Exercise recommendations include a minimum of 30 minutes of moderate-intensity physical activity (walking at a brisk pace) on most days. Start by using a pedometer, walking at breaks, or household work. In adolescents, a minimum of 60 minutes of moderate to vigorous physical activity a day.
  2. Weight loss of 5% to 10% or more. Gradual weight loss of 1 to 2 kg per month. Even small amounts of loss are associated with health benefits.
  3. Weight loss may cause a resumption of ovulation and the ability to get pregnant.
  4. High-fiber, low-fat diet, and reduction of refined sugar.
  5. Hair removal can be achieved with shaving, waxing, or use of depilatories. Electrolysis and laser treatment are more expensive therapies for hirsutism.
C. Pharmaceutical therapy:
  1. If pregnancy or ovulation induction is not desired, oral contraceptives (OCPs) are most often used for menstrual cycle regulation, endometrial hyperplasia prevention, and hirsutism:
    a. Due to sodium and water retention from the estrogen in OCP, weight reduction may be more difficult.
    b. OCPs that contain 20 to 35 mcg of ethinyl estradiol and progestins, such as norethindrone (Ortho Micronor), norgestimate (Ortho Tri-Cyclen), desogestrel (Desogen and Ortho-Cept), or drospirenone (Yaz and Yasmin) are prescribed for PCOS.
    c. The U.S. Medical Eligibility Criteria for Contraceptive Use is a useful tool to screen for contraindications.
  2. Progestin:
    a. Medroxyprogesterone acetate (Provera) 5 to 10 mg daily for 10 to 14 days every 1 to 3 months can be used to treat menstrual irregularity and induce withdrawal bleed for endometrial protection.
    b. Progestin-releasing intrauterine device (IUD) may be used to prevent endometrial hyperplasia and control abnormal uterine bleeding.
  3. For those with insulin resistance, metformin (Glucophage) is used to sensitize insulin, manage oligomenorrhea, promote weight loss, and induce ovulation in females with PCOS:
    a. Start metformin 500 mg at the evening meal. The dosage can be increased by 500 mg per week in divided doses up to the maximum of 2,000 mg/d.
    b. Titrate slowly due to gastrointestinal (G)I side effects (diarrhea).
    c. Check metabolic panel before and every 3 to 6 months to evaluate for lactic acidosis.
    d. Metformin is contraindicated in clients with late-stage renal impairment; assess renal function prior to instituting metformin and monitor regularly.
    e. Metformin must be stopped prior to any procedure with radiographic dye.
  4. Spironolactone (Aldactone) 100 to 200 mg given in two divided doses a day can be used as second-line antiandrogen therapy to treat hirsutism and acne if symptoms are not improved after a 6-month OCP trial.
    a. Spironolactone may be used alone or in combination with OCPs.
    b. For those who are sexually active, alternative methods of birth control should be used when spironolactone is used alone due to potential congenital defects such as abnormal development of the male fetus' external genitalia.
    c. Monitor for dose-dependent hyperkalemia during spironolactone therapy.
  5. Eflornithine (Vaniqa) topical may be prescribed to prevent facial hair regrowth.
  6. If pregnancy is desired, letrozole or clomiphene citrate (Clomid) is used to induce ovulation. Lifestyle modification including weight loss in overweight and obese individuals should be attempted before starting ovulation induction treatment.

FOLLOW-UP

A. Glucose tolerance needs to be evaluated regularly for clients diagnosed with type 2 DM and PCOS (Table 24.6).

CONSULTATION/REFERRAL

A. Refer to an obstetrician/gynecologist or a reproductive endocrinologist for consultation and management of the following:
  1. Infertility/pregnancy.
  2. Menstrual bleeding that is not controlled despite OCPs or progestin-releasing IUD.
B. An endocrinologist may be an appropriate consultation.
C. Consider a nutritional consultation.

INDIVIDUAL CONSIDERATIONS

A. Adults:
  1. Females who use OCPs are at increased risk for venous thromboembolism (VTE). Educate all clients regarding this risk factor and monitor the client closely for this risk. ▶

TABLE 24.6 ANDROGEN EXCESS SOCIETY SCREENING AND TREATMENT REQUIREMENTS FOR IGT

All clients with PCOS should be screened for IGT using OGTT, fasting plasma glucose, or hemoglobin A1C. OGTT is recommended in high-risk clients.
Clients with a normal glucose test should be rescreened at least once every 1–2 years or earlier if additional risk factors are identified.
Clients with IGT should be screened annually for the development of type 2 DM.
Adolescents with PCOS should be screened for IGT using a 2-hours OGTT every 2 years. If IGT develops, treatment with metformin should be considered.
The first-line treatment of PCOS and IGT is lifestyle modification (balanced diet, exercise, and weight loss in overweight and obese clients). Insulin-sensitizing agents (metformin) should be considered in clients with PCOS and IGT.

DM, diabetes mellitus; IGT, impaired glucose tolerance; OGTT, oral glucose tolerance test; PCOS, polycystic ovarian syndrome.

Sources: From Salley, K. E. S., Wickham, E. P., Cheang, K. I., Essah, P. A., Karjane, N. W., and Nestler. (2007). Glucose intolerance in polycystic ovary syndrome–a position statement of the Androgen Excess Society. *Journal of Clinical Endocrinology and Metabolism, 92*(12): 4546–4556. https://doi.org/10.1210/jc.2007-1549;

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2. Obese females older than 40 years of age are at greater risk for VTE. Precautions should be used when prescribing OCPs to this population.

3. Chronic conditions of the client should also be considered before prescribing OCPs for PCOS. Contraindications to the use of OCPs include clients with a history of VTE, HTN, females 35 years or older who smoke 15 or more cigarettes per day, cardiovascular disease (CVD), recent surgery, diabetes, and rheumatic diseases. The client's medical history should be thoroughly evaluated. The U.S. Medical Eligibility Criteria for Contraceptive Use is a useful tool to screen for contraindications.

B. Geriatrics:

1. Estrogen (oral/transdermal) with or without progestin increases risk for breast cancer, endometrial cancer, and worsening of incontinence, and lacks cognitive and cardiopulmonary protection.

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THYROID DISEASE

HYPERTHYROIDISM

DEFINITION

A. Hyperthyroidism, also known as overactive thyroid, is a condition in which the thyroid gland produces excess amounts of thyroid hormones. Hyperthyroidism may be subclinical and may not be easily recognized or exhibit overt symptoms. The most common hyperthyroid conditions are Graves disease and toxic multinodular goiter. The U.S. Preventive Services Task Force (USPSTF) currently concludes that there is insufficient evidence to recommend screening for thyroid dysfunction in asymptomatic adults who are not pregnant.

INCIDENCE AND PREVALENCE

A. Hyperthyroidism is more common in females and smokers. Overall prevalence of hyperthyroidism is approximately 1.3%. The most common cause of hyperthyroidism is Graves disease, which is responsible for 60% to 80% of hyperthyroid cases. The incidence of Graves disease is estimated to be 20/100,000 to 50/100,000.

B. Females:

1. The ratio of 5:1 is higher in females than males.

2. Graves disease is more common in younger females.

3. Toxic nodular goiter is more common in older females.

C. Older adults:

1. Toxic multinodular goiter, also referred to as toxic adenoma of the thyroid (called Plummer disease if single nodule), occurs in 15% to 20% of clients with thyrotoxicosis.

D. Symptomatology incidence:

1. Ophthalmopathy is more common in smokers.

2. Atrial fibrillation has 10% to 25% incidence and is more common in older adults.

3. Autoimmune thyroid diseases have a peak incidence in people aged 20 to 40 years.

PATHOGENESIS

A. Hyperthyroidism is one form of thyrotoxicosis in which an excess of hormone is excreted by the thyroid gland. The diseases that can cause hyperthyroidism include Graves disease, toxic multinodular goiter, thyroid cancer, and increased secretion of the thyroid-stimulating hormone (TSH). Thyrotoxicosis not related to hyperthyroidism may be subacute thyroiditis, ectopic thyroid tissue, and ingestion of excessive thyroid hormone. Postpartum thyroiditis can precipitate a short-term mild hyperthyroidism, which has an onset at 2 to 6 months postpartum. Severe thyrotoxicosis of any cause is called thyrotoxic crisis or storm.

B. In Graves disease, the normal feedback mechanisms that regulate thyroid hormone secretion are taken over by autoantibodies that stimulate the TSH receptor in an abnormal mechanism. Thyroid autoantibodies of the immunoglobulin G (IgG) class are present in more than 95% of clients with Graves disease. The hyperfunctioning of the thyroid gland increases circulating thyroid hormones that are responsible for thyrotoxic symptoms and promotes iodine uptake and thyroid hyperplasia.

C. Toxic multinodular goiter is another common cause of hyperthyroidism and increases in prevalence with age and in populations with iodine deficiency. The hyperfunctioning thyroid nodules from the toxic multinodular goiter can lead to clinical thyrotoxicosis.

D. Thyroiditis is a term used for hyperthyroid conditions that involves thyroid inflammation and the release of circulating thyroid hormones. Certain medications and health conditions requiring increased thyroid hormones are causes of thyroiditis and may result in transient hyperthyroidism.

PREDISPOSING FACTORS

A. Graves disease:
- **1.** Females in the second through fifth decades of life.
- **2.** Familial autoimmune thyroid disease.
- **3.** Concomitant disorders believed to be autoimmune.
- **4.** Increase in trisomy 21.
- **5.** Higher incidence in smokers.

B. Toxic multinodular goiter:
- **1.** Advanced age.
- **2.** Long-standing simple goiter.
- **3.** Dietary iodine deficiency.

C. Thyroiditis:
- **1.** Exposure to certain medications (amiodarone, lithium, interferon, antineoplastic agents).
- **2.** Conditions such as pregnancy, and immunologic, viral, or genetic disorders.

COMMON COMPLAINTS

A. Graves disease:
- **1.** Prominence/protrusion of the eye (exophthalmos).
- **2.** Prominent "stare."
- **3.** Visual changes:
  - a. Diplopia.
  - b. Photophobia.
  - c. Eye irritation: gritty feeling or pain.
  - d. Eyelid retraction, proptosis, periorbital edema, and conjunctival redness.

B. Weight loss with no change in diet or an increase in appetite.

C. Anorexia (may be prominent in older adults).

D. Weakness and fatigue.

E. Tachycardia.

F. Heat intolerance, diaphoresis.

G. Thinning scalp hair.

H. Onycholysis: fingernail separation from the nail bed.

I. Smooth, warm, moist, thin skin. In some adults and older adults, skin might have peau d'orange appearance and difficult to pinch.

J. Heart palpitations (atrial fibrillation).

K. Bowel symptoms:
- **1.** Increase in frequency and loose bowel movements (not diarrhea).
- **2.** Constipation (more frequent in older adults).

L. Swelling of feet and ankles

M. Neurologic: fine tremors extremities/digits, hyperkinesia, hyperreflexia.

OTHER SIGNS AND SYMPTOMS

A. Goiter: **Approximately 50% of clients will not have an enlargement of the thyroid gland**. Older adult clients are less likely to have a goiter.

B. Thyroid/neck tenderness with/without thyroid bruits.

C. Periorbital edema.

D. Flushing, warm skin; pruritus.

E. Fine hand tremors.

F. Dyspnea (especially older adults).

G. Exertional fatigue/exercise intolerance.

H. Insomnia.

I. Irritability.

J. Nervousness.

K. Mood swings.

L. Inability to concentrate.

M. Depression and apathy (older adults).

N. Menses (oligomenorrhea or amenorrhea).

O. Impotence and decreased libido in men.

P. Gynecomastia.

Q. Galactorrhea (TSH-mediated hyperthyroidism).

R. Atrial dysrhythmias (atrial fibrillation), left ventricular dilation (common in older adults).

S. Urinary frequency, polyuria and nocturia.

T. Osteopathy/thyroid acropachy: subperiosteal bone formation and swelling of metacarpal bones (most common in older adults).

U. A combination of these noted symptoms should lead to the assessment of hyperthyroidism.

SUBJECTIVE DATA

A. Identify when symptoms began, duration, and any change or progression.

B. Identify whether the client has noticed enlargement of the thyroid gland, difficulty swallowing, or change in voice.

C. Assess for change in weight over the past 3 months, past 6 months, and last year. Ask the client whether their appetite has changed.

D. Explore the client's family history of thyroid problems.

E. Obtain the client's medical history of associated diseases (especially those of autoimmune pathogenesis: pernicious anemia, type 1 diabetes mellitus [DM], myasthenia gravis, rheumatoid arthritis, ulcerative colitis).

F. Review the client's medication history, including amiodarone, interferon alpha, levothyroxine (overdose), expectorants, and health food supplements containing seaweed.

G. Ask the client to identify any changes in bowel habits, loose (nondiarrhea) stools, frequency of bowel movements, or constipation.

H. Ask about moods, changes in concentration, feelings of restlessness, nervousness, anxiety, and change in sleep habits.

I. Assess for any cardiac symptoms, such as palpitations, chest pain, shortness of breath, and decreased tolerance for past activities.

J. Ask whether the client has noticed any swelling or puffiness anywhere.

K. Determine whether the client has experienced changes in vision and/or eye irritation.

L. Assess for hand tremors, increase in the moistness and coolness of the skin, flushing, and blushing.

M. Ask the client to identify any menstrual changes or whether the client has had a recent pregnancy or is in the postpartum period.
N. Review for a recent history of a viral infection.
O. Review recent trauma to the neck (significant trauma can cause thyrotoxicosis).

PHYSICAL EXAMINATION

A. Check temperature, if indicated, pulse (tachycardia), respirations (dyspnea), blood pressure (systolic hypertension [HTN]), and weight.
B. Inspect:
1. Observe overall appearance: Does the client have any difficulty with breathing, including dyspnea, or difficulty swallowing from tracheal obstruction secondary to a large goiter?
2. Note eyelid retraction, lid lag, and exophthalmos. The clinician may see periorbital edema and an elevated upper eyelid, which leads to decreased blinking and a staring quality in Graves disease.
3. Note tremors that are best demonstrated from outstretched hands.
4. Inspect the skin for temperature and texture.
5. Inspect the fingernails for the following:
a. Onycholysis, also known as Plummer's nails (loosening of the nails from the nail beds).
b. Softening of the nails.
6. Inspect scalp/hair.
C. Auscultate:
1. Auscultate the thyroid for bruits.
2. Auscultate the heart and pulse rate. Clients with subclinical hyperthyroidism frequently present with atrial fibrillation.
3. Auscultate the carotid arteries for bruits.
4. Auscultate bowel sounds.
D. Palpate:
1. Palpate the neck and thyroid for nodules, thrills, and enlargement. Depending on the etiology of the hyperthyroidism, the thyroid may range from normal to massive (Graves disease or toxic multinodular goiter). Palpation of the thyroid can induce the gland to release increased hormone; be alert for signs and symptoms of thyroid storm.
2. If the thyroid is tender and painful to palpation, granulomatous thyroiditis may be the etiology of hyperthyroidism.
3. Palpate the heart for thrills.
4. Palpate extremities for edema. Pretibial myxedema is noted in Graves disease.
E. Neurologic examination:
1. Assess deep tendon reflexes (DTRs).
2. Tests for lid lag:
a. Have the client follow your finger as it moves up and down.
b. Have the client look down and observe if sclera can be seen above the iris.
3. Have the client stick out the tongue to observe for presence of tremors.

DIAGNOSTIC TESTS

A. TSH, free thyroxin (T4), triiodothyronine (T3; Table 24.7).
B. Radioactive iodine (131I) uptake (RAIU) to determine hyperthyroidism cause if needed. If the client has ophthalmopathy, clinical symptoms of hyperthyroidism, and a diffusely enlarged thyroid gland, the RAIU test is not necessary to confirm Graves disease. Pregnancy and breastfeeding are absolute contraindications to radionuclide imaging. When nodules have difficulty absorbing iodine, they will appear "cold" and biopsy is in order to rule out cancer. If nodules are overproducing hormones, it will appear darker and is called "hot." Thyroid nodules increase with age: Approximately 95% are benign and 5% are malignant.
C. Consider additional laboratory testing:
1. Complete blood count (look for possible elevated white blood cell count, thrombocytopenia, microcystic anemia).
2. Serum ferritin (may be high).
3. Thyroglobulin (Tg) cancer marker.
4. Thyroid peroxidase (TPO) antithyroid microsomal antibody to detect autoimmune disorder of thyroid.
D. If T4 and T3 are high, but TSH is normal or high, a pituitary MRI should be ordered to look for a pituitary mass.
E. An EKG should be obtained to evaluate palpitations and possible atrial fibrillation, and an echocardiogram should be considered if an irregular heart rate and signs of heart failure are noted on examination.
F. Microscopic for Plummer's/toxic multinodular goiter: hyperplastic nodules with discrete fibrous capsule; nonfunctioning nodules with degenerative changes of fibrosis, calcifications, and old/new hemorrhage.
G. Molecular/cryogenics: monoclonal or polyclonal.

DIFFERENTIAL DIAGNOSES

A. Hyperthyroidism:
1. Graves disease.
2. T3 toxicosis.
3. T4 toxicosis.
4. Thyroid adenoma.
5. Drug-induced hyperthyroidism, that is, iodine-rich amiodarone.
6. Subclinical hyperthyroidism.

TABLE 24.7 TEST RESULTS IN HYPERTHYROIDISM

Disorder	TSH	Free T4	T3	RAIU and Thyroid Scan
Overt hyperthyroidism	L	H	H	H
Graves disease	L	H	H	H
Multinodular goiter	L	H	–	H
T3 thyrotoxicosis (may be caused by antithyroid drug therapy)	L	N or H	H	N or H

H, high; L, low; N, normal; RAIU, radioactive iodine uptake; TSH, thyroid-stimulating hormone.

7. TSH-induced hyperthyroidism (TSH-secreting pituitary adenoma).
8. Hyperthyroidism during pregnancy.
9. Hashitoxicosis (combination of Hashimoto and thyrotoxicosis), rare autoimmune thyroid disease.
10. Dyshormonogenetic goiter of diffuse changes (compared with focal changes) in toxic multinodular goiter.

B. Cardiovascular disease (CVD) such as coronary artery disease (CAD) and heart failure.
C. Gastrointestinal (GI) disorders such as irritable bowel syndrome, ulcerative colitis, and Crohn's disease.
D. Cancer: testicular germ cell tumors.
E. Neurologic disorder.
F. Hydatidiform mole (molar pregnancy).
G. Psychological disorder.

PLAN

A. General interventions:
1. Carefully assess for complications of hyperthyroidism (cardiac, ophthalmologic, GI, musculoskeletal, and psychological); address each area identified.
2. Clients with tachycardia, palpitations, tremors, anxiety, and eyelid lag can be treated with a beta-adrenergic antagonist for some relief from those symptoms until they become euthyroid.
3. For infiltrative dermopathy over the lower extremities, occlusive wraps on the affected side are recommended.
4. Only 5% of clients with Graves' disease develop severe ophthalmopathy. An initial ophthalmologic exam is recommended as baseline, with follow-up determined by the ophthalmologist.

B. Client teaching:
1. Clients taking propylthiouracil (PTU) or methimazole (MMI) should be instructed to immediately report adverse effects, including rash, hives, fever, jaundice, abdominal pain, and clay-colored stools.
2. Less than 1% of clients develop agranulocytosis, but all clients should be instructed to call the provider immediately if a fever, sore throat, or joint ache develops because of their susceptibility to serious infection.
3. Clients receiving radioactive iodine (RAI) should be informed that they most likely will need to take lifelong hormone replacement after the RAI treatment is completed.
4. Teach safety measures to guard against the possibility of fractures due to bone density loss secondary to hyperthyroidism. Get a baseline bone density scan (dual-energy x-ray absorptiometry [DEXA] scan).
5. Inform clients that an episode of serious depression may follow successful treatment of hyperthyroidism.
6. No special diet is required; however, clients should be told to avoid herbal supplements and sushi that contains seaweed.

C. Medical-surgical management:
1. RAI is a common choice of therapy for adults. It is administered in capsule form or in water:
a. One dose is usually sufficient; however, a second dose may be given if necessary.
b. Permanent hypothyroidism requiring lifelong hormone replacement is the only notable complication.
2. A thyroidectomy is seldom used except in limited client conditions:
a. Pregnancy: if females are noncompliant or cannot tolerate thionamides due to allergies or agranulocytosis:
b. Clients who refuse RAI therapy.
c. Refractory amiodarone-induced hyperthyroidism.
d. Clients with unstable cardiac conditions that require quick normalization of thyroid function.

D. Pharmaceutical therapy:
1. Antithyroid drugs: The choice of drugs depends on clinician experience, the severity of the disease, client preference, and pregnancy status. Monitor thyroid function and titrate antithyroid drug dose every 4 weeks until thyroid function normalizes and to ensure the client does not become hypothyroid. Graves disease may go into remission after treatment for 12 to 18 months, and drug therapy can be discontinued.
a. PTU:
i. Initial dose 100 to 150 mg PO TID. Dosage reduction is almost always required at 4 to 8 weeks of start as hyperthyroidism symptoms improve.
ii. Thyroid storm: 150 to 200 mg orally every 4 to 6 hours.
iii. Except in thyroid storm, PTU is considered a second-line drug therapy. It is reserved for use in clients who are allergic to or intolerant of MMI and in clients who are in the first trimester of pregnancy or planning pregnancy.
iv. Switch to MMI for the remainder of the pregnancy (second and third trimesters).
v. PTU has a black box warning for acute liver failure
b. MMI (Tapazole):
i. Adults: 15 to 40 mg/d divided into three doses. Maintenance doses are 5 to 15 mg/d in three divided doses.
ii. MMI is more potent than PTU and has a longer duration of action.
iii. MMI is not recommended for use in the first trimester of pregnancy.
2. RAI therapy is one of the most common treatments in adults. It is administered orally as a single dose. RAI is contraindicated in pregnancy and lactation.
3. Beta-blockers such as propranolol (Inderal) are also used for hyperthyroidism until TSH levels are normalized.
4. Atrial fibrillation treatment is directed toward restoring a euthyroid state. Other therapies include the following:
a. Beta-blockers (unless contraindicated, caution use in those with chronic obstructive pulmonary disease [COPD] and asthma).
b. Calcium channel blockers if unable to utilize beta-blocker.
c. Oral anticoagulation (keeping the international normalized ratio [INR] between 2 and 3).
d. Antiarrhythmic drugs and cardioversion may be unsuccessful until euthyroid.

FOLLOW-UP

A. Depending on the experience of the clinician, an endocrinologist best prescribes therapy, including RAI and antithyroid medication.
B. Clients receiving PTU or MMI should be seen in the office every 4 to 6 weeks for evaluation and have blood drawn for serum TSH and free T4 measurements until euthyroid state is achieved and maintained.
C. Clients are usually maintained on these drugs for 1 to 2 years, with office visits every 3 months. The drug is then gradually withdrawn, and 25% to 90% of clients experience permanent remission. ▶

D. Clients with RAI therapy should be seen in the office to monitor thyroid levels. It is anticipated that most of them will require lifelong hormone replacement medication following treatment.

E. Due to the increased risk of bone loss, perform bone density screening (DEXA scan) to evaluate for osteopenia/osteoporosis.

F. Consider testing for impaired glucose tolerance (IGT) in untreated clients.

CONSULTATION/REFERRAL

A. Ophthalmology consultation is recommended for clients with ophthalmologic involvement.

B. If surgery is the choice of treatment, refer the client to an endocrinologist and a surgeon.

C. An experienced obstetrician or perinatologist should follow pregnant females.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Pregnant clients with mild hyperthyroidism may be followed with treatment.

2. If an antithyroid drug is necessary, PTU is the drug of choice in the first trimester. MMI has been associated with congenital anomalies such as tracheoesophageal fistula and choanal atresia. After the first trimester, mothers may be switched to MMI.

3. Monitor thyroid function every 4 weeks during pregnancy.

4. RAI therapy is contraindicated during pregnancy and breastfeeding.

5. Thyrotoxicosis may improve during pregnancy; however, symptoms may relapse during the postpartum period.

6. Hyperthyroidism in the third trimester may increase the risk of low birth weight.

7. Prolonged high-dose iodine therapy can cause fetal goiter.

8. Ultrasounds should be performed to evaluate for fetal hyperthyroidism, assessing for goiter, poor growth, cardiac failure, and hydrops fetalis.

B. Adults:

1. sympathetic activation (anxiety, hyperactivity, etc.) is seen in adult years more commonly than in older adults population.

C. Geriatrics:

1. Older clients exhibit fewer clinical signs, but may still have the following: tachycardia, atrial fibrillation, dyspnea, fatigue, moderate to severe ophthalmopathy, and weight loss.

2. Less frequent signs in this population include atrial fibrillation, hyperactive reflexes, increased sweating, heat intolerance, tremors, nervousness, polydipsia, new onset of hoarseness, difficulty swallowing, and increased appetite.

3. Older adult females with hyperthyroidism are at increased risk for accelerated bone loss.

4. Malignancy occurs most often in clients ≤20 years old and ≥70 years of age.

5. In older adults, if TSH is low but free T4 remains within the normal limits (subclinical hyperthyroid), retest in 4 to 6 weeks. If TSH remains low, test the T3. Treat subclinical hyperthyroidism if symptoms are present and the TSH remains less than 0.1 mIU/L after 3 to 6 months.

6. In older adults with CVD or low bone density, treat subclinical hyperthyroid if TSH is 0.1 to 0.5 mIU/L (Reubin et al., 2016).

7. Target is raised for geriatrics: Serum TSH is 4 to 6 mIU/L (for nonolder adults, it remains 0.5–4.5 mIU/L).

8. Angina pectoris and heart failure must be treated in conjunction with thyroid treatment. Medications implemented for geriatrics include beta-adrenergic blockers and MMI (Tapazole) only if a definitive diagnosis was made with radioactive iodine or from the discretionary decision of an endocrinologist specific to the unique history of a client.

9. Hyperthyroid medications may cause increases in liver enzymes in geriatrics. Discontinue medication if this occurs.

10. Beers Criteria considerations: See "Individual Considerations" in section "Thyrotoxicosis/Thyroid Storm."

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HYPOTHYROIDISM

DEFINITION

A. Hypothyroidism is a condition in which the body does not produce enough thyroid hormone and may present with a goiter. In general, hypothyroidism is considered permanent, requiring therapy to restore a euthyroid state. The most common worldwide cause of hypothyroidism is iodine deficiency, while the most common cause in the United States is Hashimoto thyroiditis, an autoimmune thyroid disease.

INCIDENCE AND PREVALENCE

A. Hypothyroidism occurs in approximately 5% of the population.

B. It occurs in females more than males (five to eight times higher).

C. It may present in up to 15% of people older than 65 years.

D. In adolescents, approximately 6% have acquired hypothyroidism.

E. Approximately 10% of clients with type 1 diabetes mellitus (DM) will develop chronic thyroiditis.

F. After pregnancy, up to 10% of females develop lymphocytic thyroiditis in the postpartum period (up to 10 months postpartum).

G. Approximately 1:4,000 newborns have congenital hypothyroidism (cretinism).

H. The incidence is higher in whites (5.1%) and Mexican Americans than in African Americans (1.7%).

PATHOGENESIS

Hypothyroidism is caused by an insufficient production of thyroid hormones by the thyroid gland, either by a primary or a secondary cause. ▶

A. Primary causes include decreased hormone production caused by autoimmune thyroiditis, endemic iodine deficiency, congenital defects, or decreased thyroid activity after treatment for hyperthyroidism.
B. Secondary causes are much less common but may include insufficient stimulation from the pituitary or hypothalamus and peripheral resistance to thyroid.
C. The most common cause of primary hypothyroidism in the United States is chronic autoimmune thyroiditis, called Hashimoto thyroiditis. In this disease, circulating thyroid antibodies and the infiltration of lymphocytes destroy thyroid tissue. Autoimmune thyroiditis may also be a result of an inherited immune defect.
D. Acute or infectious thyroiditis is a rare cause of hypothyroidism in which the cause is an acute bacterial infection. Subacute thyroiditis, a nonbacterial inflammation of the thyroid, is often preceded by a viral infection. Both conditions cause inflammation of the thyroid gland by lymphocytic and leukocytic infiltration into the thyroid tissue, resulting in hypothyroidism.

PREDISPOSING FACTORS

A. Iodine deficiency.
B. Females older than 40 years at highest risk.
C. Presence of other autoimmune disorders (diagnosed and previously undiagnosed).
D. Recent acute bacterial or viral infection.
E. Treatment with radioactive iodine (RAI) for thyroid gland problems.
F. Surgical removal of thyroid gland.
G. Exposure to external radiation.
H. Evidence of pituitary or hypothalamic disease.
I. Postpartum.
J. Type 1 (autoimmune) DM.
K. Chromosomal disorders:
  1. Down syndrome.
  2. Turner syndrome.
  3. Klinefelter syndrome.
L. Celiac disease.
M. Drug-induced:
  1. Amiodarone.
  2. Interferon alpha
  3. Thalidomide.
  4. Lithium.
  5. Stavudine.
  6. Dopamine.

COMMON COMPLAINTS

A. Weight gain/obesity.
B. Fatigue/sluggishness.
C. Cold intolerance.
D. Constipation.
E. Dry and flaky skin.
F. Coarseness or loss of hair, inability of hair to hold a curl, hair loss at eyebrows, and reduced growth of hair.
G. Reduced growth of nails.
H. Hoarseness.
I. Memory or mental impairment, difficulty concentrating, and slowed speech or thinking.
J. Periorbital edema and facial puffiness.
K. Irregular or heavy menses and infertility.
L. Muscle aching and stiffness.

OTHER SIGNS AND SYMPTOMS

A. Asymptomatic if subclinical hypothyroidism and have no overt symptoms.
B. Delayed reflexes.
C. Elevated blood pressure (BP).
D. Hyperlipidemia.
E. Jaundice.
F. Painful subacute thyroiditis:
  1. Sudden neck pain with sore throat, radiating to jaw and ears, and pain shifting to sides of the neck.
  2. Late stage: myxedema, thick scaly skin, muscle weakness/joint pain, enlarged tongue, hearing loss, bradycardia, cardiac hypertrophy, pleural effusion, and ascites.
G. Pituitary or hypothalamic failure:
  1. Loss of axillary and pubic hair.
  2. Cessation of menses.
  3. Postural hypotension.
H. Exercise intolerance:
I. Carpal tunnel syndrome is a common occurrence.
J. Depression.
K. Ataxia.
L. Decreased concentration/memory impairment.

SUBJECTIVE DATA

A. Note history of recent illness and/or pregnancy.
B. Evaluate the client's medical-surgical history for any treatment of hyperthyroidism, including radioactive treatment or thyroidectomy.
C. Review dietary and weight history, how much weight has been gained and over what period of time.
D. Review any changes in health status or symptoms associated with other body systems (thyroid symptoms usually involve multiple body systems).
E. Review any history of obstructive sleep apnea (OSA).
F. Assess for pain or swelling of the neck or difficulty swallowing.
G. Inquire as to the client's history of supervoltage x-ray therapy to the neck for nonthyroid cancer or for polio.
H. Identify family history:
  1. Does the client have a first-degree relative with thyroid disease?
  2. Is there a family history of any endocrine problems, including thyroid, type 1 DM, and/or receptor agonist (RA)?
I. Review the client's medication history for current medication, over-the-counter (OTC) medications, vitamins, or herbal supplements.
J. Review the client's menstrual history or history of infertility.

PHYSICAL EXAMINATION

A. Check pulse (bradycardia), respirations, BP (usually increased diastolic BP), and weight.
B. General observation:
  1. Gait problems such as ataxia or rigidity and spasticity of the trunk and proximal extremities.
  2. Quality of the client's voice (hoarseness).
  3. Signs of depression, decreased concentration, or memory impairment.
C. Inspect:
  1. Inspect the skin (dry/flaky) for presence of jaundice.
  2. Inspect the hair (coarse, thin, and brittle) and decrease in pubic/axillary hair pattern.
  3. Perform oral examination for evaluation of macroglossia (enlarged tongue).
  4. Inspect the face/eye for periorbital puffiness/edema.
  5. Inspect the neck for the presence of a goiter and surgical scar. ▶

D. Auscultate:
1. Auscultate the thyroid and carotids.
2. Auscultate the heart.
3. Auscultate the lungs.
4. Auscultate the abdomen for bowel sounds in each quadrant (hypoactive).
E. Palpate:
1. Palpate the neck, thyroid gland, and lymph nodes.
2. Palpate the abdomen for presence of abdominal distension, ascites, and hepatomegaly.
3. Palpate/evaluate extremities for edema.
F. Musculoskeletal examination:
1. Perform a detailed musculoskeletal examination:
G. Neurologic examination:
1. Check visual fields (restricted with hypothyroidism).
2. Test hearing:
a. Whisper words and have the client repeat.
b. Use a ticking watch.
c. Use a tuning fork:
i. Weber test: Place vibrating fork on top midline of head (sound hearing equally in both ears).
ii. Rinne test: Place vibrating tuning fork on mastoid, begin counting, and ask the client to tell you when they no longer hear; then quickly reposition 1/2 to 1 in. from the ear and ask the client when they no longer hear (hearing should be twice as long as bone conduction).
3. Check for loss or reduction of deep tendon reflexes (DTRs).
4. Evaluate proximal muscle weakness/strength.
5. Test for abnormal tandem gait: Have the client walk across the room in a heel-toe, heel-toe fashion.
6. Check for sensory loss:
a. Evaluate the first three fingers and one half of the fourth finger on testing loss of sensation from carpal tunnel syndrome.
b. Evaluate sensory loss of the feet/legs (generally symmetrical in a "stocking-glove" distribution).

DIAGNOSTIC TESTS

A. Thyroid-stimulating hormone (TSH).
B. When a high TSH is noted, repeat the test and add a free T4.
C. T3 resin uptake.
D. Thyroid antibodies (Table 24.8).
E. TSH assay (if TSH assay is elevated, it indicates hypothyroidism).
F. Thyroid scan.
G. Complete blood count (CBC; anemia).
H. Lipid profile.
I. Ultrasound of the neck and thyroid to detect nodules (not a first-line test).

DIFFERENTIAL DIAGNOSES

A. Hypothyroidism:
1. Hashimoto thyroiditis.
2. Subclinical hypothyroidism.
3. Hypothyroidism secondary to treatment/intervention for hyperthyroidism.
4. de Quervain thyroiditis.
5. If TSH and free T4 are both low, consider hypothyroidism secondary to pituitary or hypothalamic failure.
B. Obesity: Clients with elevated total cholesterol levels or triglyceride levels are often misdiagnosed by assuming that these symptoms are caused by obesity and high-fat diets.
C. Depression.
D. Ischemic heart disease.
E. Nephrotic syndrome.
F. Cirrhosis.
G. Side effects/adverse effects of medications.
H. Constipation.
I. Sleep apnea/sleep disorder.
J. Fibromyalgia.
K. Infectious mononucleosis.

PLAN

A. General interventions:
1. The American Thyroid Association clinical practice guideline for detection of both subclinical and symptomatic hypothyroidism recommends that all adults aged 35 and older be screened for hypothyroidism every 5 years to identify thyroid disease in the early stages. However, Medicare does not cover a screening TSH in an asymptomatic client.
2. Clients with underactive thyroids require lifelong treatment with levothyroxine.
3. In clients with subacute thyroiditis, relatively large doses of nonsteroidal anti-inflammatory drugs (NSAIDs) or prednisone may be prescribed.
4. For clients with enlarged thyroid glands, surgery may be recommended if the gland begins obstructing the airway.
5. The treatment of clients with malignant thyroid nodules depends on the type of cancer.
B. Client teaching:
1. Teach the client about the nature and course of the disease, as well as the signs and symptoms. Frequently, ▶

TABLE 24.8 TEST RESULTS IN HYPOTHYROIDISM

Disorder	TSH	Free T4	T3	RAIU and Thyroid Scan	Peroxidase Antibodies
Hypothyroid	H	L	Sometimes L	N or L	N/A
Hashimoto disease	H or variable	N or L	Not helpful	Variable	Positive
Subacute hypothyroidism	L	H	H or variable	L or absent	Usually thyroiditis
Silent lymphocytic	L when toxic	H when toxic	N/A	L when toxic	Positive
Thyroiditis (usually postpartum)	H when hypothyroid	L when hypothyroid			

H, high; L, low; N, normal; N/A, not applicable; RAIU, radioactive iodine uptake; TSH, thyroid-stimulating hormone.

clients are relieved that perceived symptoms are real and that there is treatment. This may also improve treatment compliance.

2. Teach the client to report any side effects of the drug:
- a. Tachycardia.
- b. Palpitations.
- c. Chest pain.

3. Emphasize the need for lifelong treatment with levothyroxine and the dangers of noncompliance.

4. Thyroid replacement should be taken on an empty stomach, ideally taken 30 to 60 minutes before breakfast.

5. The beneficial effects of thyroid replacement occur in about 3 days to 1 week; the client may not feel the clinical effects for several months.

6. When switching brands or using a generic version, the serum TSH should be checked in 6 weeks.

C. Pharmacologic therapy:

1. Clients requiring therapy with levothyroxine should be treated with the same brand/generic consistently since potency varies between brand and generics.

- a. Levothyroxine (Synthroid, Levoxyl, Levothroid, Unithroid):
  - i. Adults 1.6 mcg/kg/d PO is administered as a single dose in the morning on an empty stomach to achieve optimal absorption; adjust dosage by 12.5 to 25 mcg increments every 4 to 6 weeks until TSH is normalized. Overtreatment increases the risk of atrial fibrillation and loss in bone density.
  - ii. Older adults with comorbid coronary artery disease (CAD) or severe chronic obstructive pulmonary disease (COPD) should be started at lower dosages 12.5 to 25 mcg/d; adjust dosage every 6 to 8 weeks as necessary; normal TSH ranges may be higher for older clients.
  - iii. Maintenance dose is 50 to 200 mcg PO every morning.
  - iv. Do not give with medication/supplements that interfere with absorption: aluminum-containing antacids, calcium carbonate, ferrous sulfate, phosphate binders, proton pump inhibitors, and sucralfate.
  - v. Reassessment of TSH must be implemented during LT-4 levothyroxine therapy when starting/discontinuing the following: estrogen, androgen therapies, anticonvulsants, rifampin, sertraline, and tyrosine kinase inhibitors.
- b. Desiccated thyroid (Armor thyroid):
  - i. Adult initial dosing is 15 to 30 mg/d PO and increased by 15 to 30 mg/d every 4 weeks.
  - ii. Maintenance dose is 60 to 120 mg/d.

2. The medication should be titrated to the lowest dosage needed to maintain euthyroidism and a nonelevated serum TSH and a normal or slightly elevated T4.

3. Older adult clients and those with CVD should be started on very low doses (12.5 mcg/d) of levothyroxine; doses are increased very gradually over 8 to 12 weeks as tolerated. Close monitoring for the development of cardiac complications such as angina, arrhythmias, and myocardial infarction needs to be undertaken.

4. Thyroid hormones should be taken on an empty stomach in the mornings to avoid insomnia.

5. Other drugs can reduce the effectiveness or affect the absorption of thyroid hormone:
- a. Cholesterol-reducing drugs.
- b. Cholestyramine interferes with absorption in the gut.
- c. Calcium carbonate.
- d. Aluminum hydroxide.
- e. Sucralfate (Carafate).
- f. If taking one of these drugs and levothyroxine, they should be taken 4 to 6 hours apart.

6. Dietary fiber can interfere with levothyroxine absorption. Coffee reduces the absorption of levothyroxine.

7. Pharmacologic treatment of the client with subclinical hypothyroidism is controversial. If goiter is present, treatment may be considered.

FOLLOW-UP

A. Clients who are not treated with medication should be seen every 6 to 12 months for reevaluation.

B. When medication is instituted, monitor laboratory values and client well-being in the office every 4 to 6 weeks.

C. After the dosage is stabilized, the client with an elevated serum TSH level should be seen every 6 to 12 months:

1. Undetectable TSH levels are indicative of overmedication.

2. High TSH levels are indicative of insufficient medication or client noncompliance.

D. After the TSH is normalized, regular annual follow-up visits are required.

E. Order dual-energy x-ray absorptiometry scan as indicated for assessment of bone loss.

CONSULTATION/REFERRAL

A. Consultation with an endocrinologist is recommended for the following:

1. Clients unresponsive to therapy.

2. Pregnant clients.

3. Presence of goiter, nodule, or other structural changes in the thyroid.

4. Compression symptoms of dysphagia.

5. Any client with myxedema, significant cardiac disease, or involvement or hypothyroidism secondary to pituitary or hypothalamic failure should be managed in consultation with a physician or should be referred to an endocrinologist for continued care.

6. Refer to a radiologist/physician if fine-needle aspiration biopsy is required.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Pregnancy: Monitor TSH levels monthly during the first trimester, and if stable once during the second and third trimesters to monitor for development of hypothyroidism.

2. Small increases in medication dosage may be required.

3. Some females develop postpartum thyroiditis and hypothyroidism after taking hyperthyroid medication during pregnancy or immediately thereafter.

4. Postpartum: Monitor TSH level at 6 weeks postpartum examination.

5. A common cause of congenital hypothyroidism is maternal and infant iodine deficiency.

6. Monitor the TSH to avoid overtreatment in postpartum females because excess thyroid hormone levels increase the risk of osteoporosis.

7. Hypothyroidism in pregnancy is associated with preeclampsia, anemia, postpartum hemorrhage, cardiac ventricular dysfunction, spontaneous abortion, low birth weight, impaired cognitive development, and fetal mortality.

B. Geriatrics:

1. Some older adult clients who are actually hyperthyroid exhibit symptoms of hypothyroidism. Monitor these clients closely, especially when adjusting the medication ▶

dosage and/or adding medications that alter the absorption of the thyroid medication.

2. Target is raised for geriatrics: Serum TSH is 4 to 7 mIU/L (for nonolder adults, it remains 0.5–4.5 mIU/L). In adults aged 80 or older, TSH up to 7.5 mlU/L is normal. If the TSH is greater than 7.5 mlU/L, recheck TSH with a free T4 to confirm prior to treatment.

3. For older clients, starting dose of LT-4 levothyroxine is 25 to 50 mcg/d; recheck every 6 weeks. Increase dose in small increments until serum TSH stabilizes at 4 to 6 mIU/L. Clients without heart disease, stroke, or dementia may start at larger doses.

4. Geriatrics frequently take morning antacids, laxatives, and iron/calcium supplements. Be sure to instruct not to take LT-4 replacement therapy with those medications, and to wait approximately 2 hours after taking levothyroxine.

5. Symptoms of hypothyroidism such as memory loss, decreased cognitive function, constipation, hypersomnia, and dry skin are frequently attributed to advancing age. To further evaluate older adult clients, assess for positive family history of thyroid disease, history of hyperthyroid treatment, or history of surgery/radiotherapy to the neck.

6. CAD and heart failure must be treated in conjunction with thyroid replacement therapy.

7. LT-4 levothyroxine hormone replacement needs to begin slowly and not put stress on the heart and central nervous system. Educate to notify if these occur: increase of angina, dyspnea, confusion, changes in sleep patterns, or sudden changes in cognition.

8. Do not assess thyroid function in ill older adults as the TSH is often falsely low or high. If thyroid disease is suspected as part of the illness, recheck to verify. If it is a very low TSH (<0.1 mlU/L), or very high (>20 mlU/L), then thyroid disease is likely.

9. Beers Criteria: Synthetic form of desiccated thyroid is not recommended because it tends to cause cardiac adverse effects in geriatrics. An alternative to homeopathic desiccated thyroid extract/treatments is levothyroxine.

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THYROTOXICOSIS/THYROID STORM

DEFINITION

A. Thyrotoxicosis is the clinical syndrome of an excessive amount of circulating thyroid hormones to tissues. Severe thyrotoxicosis of any cause is called thyrotoxic crisis or storm and is a serious complication of hyperthyroidism.

INCIDENCE

A. It is rare and accounts for 1% to 2% of hyperthyroidism admissions; the incidence varies, depending on the cause of the thyrotoxicosis.

B. The mortality associated with thyrotoxicosis is estimated at 30% of documented cases.

C. The average age for thyrotoxicosis is 42 to 43 years and the male to female ratio is 1:3.

PATHOGENESIS

A. Thyrotoxic crisis or storm usually develops in clients with undiagnosed or severe hyperthyroidism, untreated or inadequately treated hyperthyroidism, and those subjected to excessive stress from other causes. Oversecretion of T3 and T4 is followed by a release of epinephrine. Metabolism is dramatically increased, and the adrenal glands produce excessive catecholamines, as a response to stress.

PREDISPOSING FACTORS

A. Severe, uncontrolled hyperthyroidism.

B. Noncompliance with antihyperthyroid medication.

C. Inadequate preparation for thyroid surgery or recent use of iodinated contrast medium.

D. Acute illnesses: infections, diabetic ketoacidosis, postmyocardial infarction (MI), drug reaction.

E. Parturition.

COMMON COMPLAINTS

A. Sudden onset of the following:

1. Hyperthermia with diaphoresis.

2. Tachycardia (usually atrial tachydysrhythmias).

3. High-output cardiac failure.

4. Altered sensorium (usually agitation, restlessness, delirium, psychosis).

5. Nausea, vomiting, and diarrhea.

SUBJECTIVE DATA

A. Elicit information regarding the onset, duration, and nature of symptoms.

B. Determine the presence of cardiac symptoms.

C. Take a complete drug history, including the client's adherence to antithyroid medication.

D. Rule out any excessive acute stressors such as infection, pulmonary or cardiac problems, dialysis, plasmapheresis, and/or emotional stressors.

E. Definitive diagnosis of thyroid storm in older adults:

1. Elevated FT3 and FT4 *plus* at least one central nervous system (CNS) manifestation (restlessness, delirium, psychosis, lethargy, somnolence) *plus* one or more of the following symptoms: fever, tachycardia (congestive heart failure [CHF]), nausea/vomiting/diarrhea, total bilirubin 3 mg/dL.

or

2. Elevated FT3 and FT4 *plus* a combination of at least three of the following symptoms: fever, tachycardia, CHF, N/V/D, total bilirubin 3 mg/dL.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, and blood pressure (BP).

B. Inspect:

1. Inspect the skin and eyes.

C. Auscultate:

1. Auscultate the heart and lungs.

D. Palpate:

1. Palpate the neck carefully for thyroid nodules and enlargement.

E. Additional assessments:

1. Deep tendon reflexes for hyperreflexia.

2. Presence of hand tremors.

DIAGNOSTIC TESTS

A. Serum T3 and free T4.

B. Thyroid-stimulating hormone (TSH).

DIFFERENTIAL DIAGNOSES

A. Thyroid storm.
B. Thyrotoxic crisis.
C. Sepsis.
D. Pheochromocytoma.
E. Hyperthermia.
F. Cocaine use.

PLAN

A. General interventions:
1. Closely monitor temperature, pulse, respirations, and BP.
2. Assess the need to hospitalize the client for supportive therapy: intravenous (IV) fluids, medication treatment, antipyretics, and oxygen.
3. Aggressive cooling with cooling blankets/ice packs. Look for signs of infection triggering thyroid storm and treat appropriately.
4. Monitor for acute disturbances of consciousness, psychosis, and convulsions. If necessary, refer to psychiatrist/neurologist for management.
5. Monitor hydration status if fever has occurred or gastrointestinal (GI) symptoms are severe.
6. Monitor for hepatotoxicity with/without jaundice in thyroid storm secondary to potential hepatocyte damage during a thyroid storm.
B. Pharmaceutical therapy:
1. See "Pharmaceutical Therapy" in section "Hyperthyroidism" (for propylthiouracil [PTU], oral methimazole, beta-blockers, calcium channel blockers, antiarrhythmic, and warfarin therapy).
2. IV methimazole recommended for severely ill clients with consciousness disturbances during thyroid storm.
3. Corticosteroids (300 mg/d hydrocortisone or 8 mg/d dexamethasone) recommended for thyroid storm regardless of origin.
4. Aggressive anticoagulation therapy should be used based on CHADS2 for clients ≥75 years old, and for adults with diabetes, CHF, hypertension (HTN), or stroke history. Thyroid storm clients are vulnerable to disseminated intravascular coagulation (DIC).

FOLLOW-UP

A. Following resolution of the crisis, see the client in the office every 3 to 4 weeks for evaluation and monitoring of serum TSH and free T4 levels.
B. See "Follow-Up" in section "Hyperthyroidism."

CONSULTATION/REFERRAL

A. Refer the client to a physician immediately for possible hospitalization.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. See "Individual Considerations" in section "Hyperthyroidism."
B. Geriatrics:
1. Mortality for geriatric thyrotoxicosis clients is 15% to 20% of documented cases. Confusion, consciousness disturbances, and dysrhythmias are common presentations. Geriatrics respond well to immediate hydration (IV fluids), metoprolol, IV hydrocortisone, and PTU. It is recommended to evaluate for hidden urinary tract infections (UTIs)/pulmonary/skin infections, which may be the triggering factor.
2. Beers Criteria:
a. Nifedipine (Norvasc) and clonidine (Catapres) have increased sensitivity with geriatrics for potential orthostatic hypotension, bradycardia, and CNS adverse effects. Consider long-acting calcium channel blocker or angiotensin-converting enzyme inhibitor (ACEI)/angiotensin II receptor blocker (ARB).
b. Oral anticoagulants (warfarin, aspirin, dabigatran, apixaban) have bleeding risks. Warfarin is recommended with tight international normalized ratio (INR) lab testing during thyroid storm. Consider unfractionated heparin and keep aspirin doses under 325 mg/d with clients who have a history of ulcers.
c. Antiarrhythmics: Digoxin may increase mortality in atrial fibrillation and heart failure and reduce renal clearance, which will increase the risk of toxicity. Amiodarone is more toxic than other treatments in the geriatric client. Consider propafenone (Rythmol). Diltiazem or verapamil may be used if ejection fraction is stable and/or HF is stable.
d. Corticosteroids may worsen delirium in geriatrics. Use lowest effective dose for shortest amount of time and avoid combining with aspirin/anti-inflammatory drug (NSAID) to prevent GI bleeds/ulcer formation.
e. If thyroid storm was triggered by an infection, a common prescribed antibiotic for UTI has been nitrofurantoin (Macrobid). Use this medication with caution in the geriatric population due to its risk of pulmonary toxicity, neuropathy, hepatotoxicity, and concerns with creatinine clearance.

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CLIENT TEACHING GUIDE

ADDISON DISEASE

PROBLEM Addison disease has many symptoms, including weakness and fatigue, fainting or dizziness, poor appetite, weight loss, nausea, vomiting, diarrhea, abdominal discomfort, skin discoloration, and low blood pressure, as well as cravings for salty foods.

CAUSE Addison is most commonly caused by the body's unusual ability to attack its own tissues, called autoimmunity. This results in inadequate amounts of cortisol and/or aldosterone produced by the adrenal glands.

TREATMENT PLAN/CARE

A. Wear your identification band stating that you have Addison disease and that lists treatment, as well as your healthcare provider's name and phone number.

B. Increased medication dosage will be required during any significant illness, especially with vomiting and/or diarrhea, before dental extractions, and before major surgical procedures. **These issues need to be discussed with your healthcare provider so you will know the correct dosage of medication to take.**

C. Have injectable cortisol available for emergencies when you are unconscious or otherwise unable to take pills.

Activity: As tolerated; avoid overexertion.

Diet: Eat a high-sodium, low-potassium, high-protein diet.

Medications: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:

A. You have a reaction to your medications.
B. You cannot tolerate the prescribed medications.
C. You are having dental procedures or surgery, to discuss adjusting your medicines.
D. You are sick with nausea and vomiting, to discuss adjusting your medicines or coming into the office.
E. Other: _____

Phone: _____

RESOURCES MedlinePlus (National Library of Medicine): https://medlineplus.gov/addisondisease.html

National Adrenal Diseases Foundation: https://www.nadf.us/adrenal-diseases/addisons-disease

National Endocrine and Metabolic Diseases Information Service: http://endocrine.niddk.nih.gov/ pubs/addison/addison.aspx

You can build your own identification bracelets or neck chains from American Medical ID: https://www.americanmedical-id.com/customized-ids/customized-medical-id-jewelry.html

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CLIENT TEACHING GUIDE

CUSHING SYNDROME

PROBLEM Cushing syndrome has many symptoms, including weight gain, obesity, moon-shaped face, excessive hair growth, easy bruising, thin skin, muscle weakness, decreased or no menstrual periods, increased blood pressure, osteoporosis, and impaired wound healing.

CAUSE Cushing syndrome is caused by excessive cortisol production.

TREATMENT/CARE

A. Take medications precisely as directed.
B. Avoid excessive alcohol.
C. You may be instructed to purchase a MedicAlert bracelet or necklace for any emergencies. This can be used to list your treatment and healthcare provider's name and phone number.

Activity: Exercise is encouraged.

Diet: Eat a low-sodium, high-potassium, high-calcium diet.

Medications:_____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office if You:
A. Have a reaction to the medications.
B. Cannot tolerate the prescribed medicines.
C. Other: _____

Phone: _____

RESOURCES National Adrenal Disease Foundation. Cushing Patient Education:

http://www.nadf.us/adrenal-diseases/cushings-syndrome/

The Pituitary Society. Cushing Syndrome and Cushing Disease Patient Education:

https://pituitarysociety.org/patient-education/pituitary-disorders/cushings/what-are-cushings-disease-and-cushings-syndrome

You can build your own identification bracelets or neck chains from American Medical ID:

https://www.americanmedical-id.com/customized-ids/customized-medical-id-jewelry.html

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CLIENT TEACHING GUIDE

DIABETES

PROBLEM You have been diagnosed as having diabetes. Diabetes does not go away; you have to manage it every day. It is a condition in which your body cannot use the glucose from food properly. Some signs that your blood sugar is too high and too low are listed here:

A. *Hyperglycemia* (high blood sugar) signs/symptoms: fruity breath odor; abnormal breathing pattern; rapid, weak pulse; confusion or stupor.

B. *Hypoglycemia* (low blood sugar) signs/symptoms: hunger, weakness, sweating, headache, shaking, rapid heartbeat, paleness, fainting, seizures, or coma.

CAUSE Your body has an organ close to the stomach called a pancreas. Your pancreas is not making enough insulin, or your body is not using the insulin it is making properly.

TREATMENT/CARE

A. Lifestyle changes—exercise, stopping smoking, following your diet, and losing some weight—are necessary to control your diabetes.

1. Stop smoking. Smoking doubles your risk of developing heart or blood vessel disease. **It's never too late to quit smoking.**
2. Weight loss of even 5 to 10 lbs helps to control your diabetes.
3. You need to wear an identification tag that tells others you have diabetes in case of an emergency.

B. Foot care is very important.

1. Check feet every day.
2. Wash with mild soap and lukewarm water. Avoid soaking the feet.
3. Apply lotion but do not apply lotion between the toes.
4. File or clip nails after washing and drying.
5. Do not tear skin around calluses.
6. Wear clean socks daily.
7. Wear well-fitted shoes at all times: no bare feet. You may not be able to feel or detect damage.
8. Avoid crossing your legs.
9. Take your shoes off at every office visit.

C. Blood glucose monitoring: You will be instructed on how to check your blood sugar at home.

Goals of glycemia control:

1. Fasting blood sugar: 80 to 130 mg/dL.
2. 2-hour postprandial (after meal) glucose: less than 180 mg/dL.
3. Bedtime glucose: 90 to 150 mg/dL.
4. Hemoglobin A1C: less than 7% for most.

D. For low blood sugar you need to follow the 15:15 rule:

1. After having low blood sugar, you need to check your blood every 4 hours for the next 24 hours.
2. 15:15 rule: Choose one to follow:
   a. Take three glucose tablets.
   b. Drink 1/2 cup orange juice.
   c. Drink 1/2 cup apple juice.
   d. Drink 1/3 cup grape juice.
   e. Drink 6 oz of regular Coke.
3. If your blood sugar drops to less than 59, you need to follow the 15:15 rule, drinking juice or taking glucose tablets, followed with 1/2 cup of milk, and a starch and 1 oz of protein.
4. **If you have severe low blood sugar, you could pass out and go into a coma. You need to have glucagon for emergencies**:
   a. Glucagon is not glucose, but it helps the liver raise your blood glucose.
   b. Glucagon is a prescription drug and is given by injection; it usually works within 15 minutes.
   c. If you do not respond with the first shot of glucagon, your family needs to call 911.
   d. A second dose of glucagon should be given by your family if you do not awaken in 15 minutes.
   e. After receiving glucagon and you respond, you need to eat a snack.

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CLIENT TEACHING GUIDE

DIABETES (*CONTINUED*)

REGULAR CARE

A. You will need to come to the office at regular times to have your diabetes checked and make sure you do not have any complications. Your provider will follow national standards of care, including the following:

1. Order a dilated eye examination every year.
2. Check your blood pressure and keep it less than 140/90 or less than 130/80 if you are at high risk.
3. Check your cholesterol at least once a year.
4. Check your feet daily (and the bottom of your feet) for redness, swelling, cuts, or any changes, and have your healthcare provider perform a comprehensive foot examination at least once a year. Wear well-fitted and closed-toe shoes in your home to protect your feet.
5. Check your A1C every 3 months to see if your blood sugar is under control.
6. Check your urine for protein/kidney problems.
7. Update your flu vaccine every year.
8. Update your pneumonia vaccine based on the Centers for Disease Control and Prevention (CDC) recommendations.
9. Update other vaccines you may need as an adult (shingles, tetanus, etc.).

SICK-DAY RULES You will also need a special plan in the event you are sick or have a special occasion.

A. Test blood sugar more often, up to every 2 to 4 hours.
B. Increase your fluids, even if you do not feel like drinking.
C. Follow a meal plan if you can.
D. Call your healthcare provider if your blood sugar is less than 70 or greater than 240 for two readings in a row that cannot be explained; if you are unable to retain food/fluids; and if you are spilling urine ketones.
E. Check your ketones if your blood sugar is greater than 240. If your ketones are negative, keep testing if your blood sugar stays up.
F. Continue to take your usual insulin dose.
G. Do not take metformin (Glucophage) if you are dehydrated.

Activity: Exercise is very important in managing your diabetes. It not only improves blood sugar by helping your insulin to work, but it also reduces your risk of heart attack and stroke, and helps with losing weight.

Talk with your healthcare provider before starting an exercise plan. If you are currently doing some form of exercise, please continue; however, avoid any strenuous exercise.

Moderate exercise (walking, cycling, and swimming) is the best exercise. Your goal will be to develop a consistent exercise activity three to four times a week for 20 to 45 minutes. Drink plenty of fluids before and after you exercise to prevent dehydration.

Exercise causes a decrease in your blood sugar for up to 24 hours. Do not exercise if your fasting blood sugar is greater than 250 or your sugar at any time is over 300. Exercise is not recommended if you have ketones (burning fat instead of sugar).

Diet:

A. You will be seeing a dietitian to develop a nutritional plan that is suited for you.
B. Consistency with meal times and amounts and food from all the six major food groups is important.
C. Even though we stress carbohydrate counting, diabetes has abnormalities in carbohydrate, fat, and protein metabolism that cause hyperglycemia.
D. Dietary control includes limiting fats such as cholesterol and saturated fat to help control blood lipid levels and prevent cardiovascular disease.
E. Eat a balanced diet, eat at regular times, and try not to skip meals; eat about the same amount of food at meal/snack times.
F. Use portion control, decrease the fats that you eat, and decrease fast simple sugars.
G. Decrease your alcohol consumption.

Medications:

A. You may need a combination of medications or insulin to help control your blood sugar to prevent complications.
B. If you use insulin injections or the insulin pump, the best place to give it is your stomach.

(*continued*)

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CLIENT TEACHING GUIDE

DIABETES (*CONTINUED*)

C. The American Diabetes Association recommends that you take an angiotensin-converting enzyme (ACE) inhibitor (blood pressure medicine) to help protect your kidneys.
D. You may be told to take an aspirin every day (if you are not allergic).

You Have Been Prescribed: _____

You Need to Take: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office If:
A. You are sick and unable to keep foods/fluids down (vomiting) or have severe diarrhea.
B. You intend to take over-the-counter medications; they could react with your diabetes medication.
C. You are using metformin (Glucophage) and are going to have x-rays using any dyes. You must stop your medicine.

Phone: _____

Remember to carry your ID and a carbohydrate source. Glucagon should be available for severe low blood glucose caused by risk of aspiration and/or inability to swallow.

RESOURCES American Diabetes Association (ADA): http://www.diabetes.org/research-and-practice/we-support-your-doctor/patient-education-materials.html

Handouts from Academy of Nutrition and Dietetics: Diabetes Care and Education. https://www.dce.org/publications/education-handouts

You can build your own identification bracelets or neck chains from American Medical ID: https://www.americanmedical-id.com/customized-ids/customized-medical-id-jewelry.html

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CHAPTER 25

PSYCHIATRIC GUIDELINES

Kristina Potts and Doncy J. Eapen

ANXIETY

DEFINITION

A. Anxiety disorders are differentiated from transient fear and normal anxiety by persistence for 6 months or longer. Anxiety disorders are often comorbid with other psychiatric disorders, most often depression. They are characterized by excessive worry, tension, apprehension, and uneasiness from anticipated danger. Hypervigilance and avoidant behaviors are often features of anxiety disorder. It is the "fight-or-flight" response that is part of the survival instinct. The source of anxiety is largely unknown or unrecognized.

B. Anxiety in its chronic form is maladaptive and is considered a psychiatric disorder. Many cases of anxiety disorder in late life are chronic, having persisted from younger years. In its pathologic form, it interferes with developmental learning because it infers significant distress. Anxiety disorders include generalized anxiety disorder (GAD), panic disorder, social anxiety disorder, agoraphobia, and specific phobias.

INCIDENCE

A. Anxiety disorders affect approximately 40 million adults yearly. The lifetime prevalence of anxiety disorders is estimated to be from 5.7% to 28%. Prevalence of diagnosis peaks in middle age and tends to decline in later life. Increased prevalence is noted among persons who are widowed, separated, or divorced. Although highly treatable, only 36.9% of individuals suffering from one or more anxiety disorders receive treatment.

B. Anxiety is present in many medical illnesses and must be distinguished to treat it appropriately. GAD and panic disorder are also associated with frequent suicide attempts.

PATHOGENESIS

A. Some degree of familial transmission of GAD, as well as panic disorder, has been noted. Unconscious conflict is thought to be the underlying cause of anxiety, which signals the ego to be careful expressing unacceptable impulses. Behavioral anxiety is considered a conditioned response to a stimulus associated with danger.

B. Clinically, however, identifying specific anxiogenic stimuli is difficult. The onset of GAD is also thought to be the cumulative effect of several stressful life events. Many studies have found that phobic/anxiety symptoms predated clinical alcoholism by a number of years. The gamma-aminobutyric acid (GABA)–benzodiazepine receptor complex, the locus coeruleus-norepinephrine system, and serotonin are three neurotransmitter systems implicated in the biologic basis of anxiety. These systems are thought to mediate "normal" anxiety and pathologic anxiety.

PREDISPOSING FACTORS

A. Young to middle-aged females, onset usually at 20 to 30 years of age.
B. Single.
C. Lower socioeconomic status/poverty.
D. Chronic physical illness (respiratory, cardiovascular, metabolic, cognitive). Chronic mental disorder (depression, phobia, and past GAD).
E. Parental loss or separation.
F. Low affective support during childhood.
G. History of mental problems in parents.

COMMON COMPLAINTS

A. Inability to control worrying.
B. Motor tension.
C. Autonomic hyperactivity vigilance.
D. Sleep disturbance.

Statements concerning self-medication with alcohol to help with sleep may indicate a coexistent alcohol abuse/dependence diagnosis that must be treated concomitantly.

E. Shortness of breath.
F. Increased heart rate and respirations.
G. Feelings of apprehension.
H. Dizziness.
I. Abdominal disturbances/nausea.
J. Increased perspiration.
K. Trembling.

OTHER SIGNS AND SYMPTOMS

A. According to the *Diagnostic and Statistical Manual of Mental Disorders,* Fifth Edition *(DSM-5)*, anxiety is excessive worry out of proportion to the likelihood or impact of the feared events that occurs for a period of 6 months or longer, during which the person has been bothered more days than not by these concerns.

B. At least three of the following six symptoms:
  1. Muscle tension.
  2. Restlessness or feeling keyed up or on edge.
  3. Easy fatigability.
  4. Difficulty concentrating or "mind going blank" due to anxiety.
  5. Trouble falling or staying asleep.
  6. Irritability. ▶

The contributions of Karen M. Kress, Cheryl A. Glass, and Alyson Wolz to this chapter in prior editions are acknowledged here.

C. Impaired social or occupational function. The anxiety, worry, or physical symptoms significantly interfere with the person's normal routine or usual activities or cause marked distress.
D. The anxiety is not attributable to another medical condition or medication or substance effect.

SUBJECTIVE DATA

A. Review the onset, course, and duration of symptoms. How often does the anxiety occur (e.g., every day, week, or month)?
B. Review any history of anxiety and age of onset. If treated, how was the previous anxiety treated and what was the success of the treatment?
C. Determine whether there is a history of suicide attempts. Does the client have a current plan or vague ideas of suicide? Ask the client: "Have you ever thought of hurting yourself or others?" If there is any concern regarding suicide/homicide, immediately refer the client to a psychiatrist.
D. Review drug history for prescription, over-the-counter (OTC), and recreational/illicit drug use, and the client's use of caffeine, which precipitates anxiety symptoms.
E. Review the client's history of alcohol consumption. Mild or moderate alcohol withdrawal presents primarily with anxiety symptoms. In clients who have developed tolerance to the effects of alcohol or benzodiazepines, abrupt cessation of these agents may produce heightened anxiety over baseline, as well as a risk of seizure.
F. Review the client's history for major stressors. Are these stressors new or chronic? If chronic problems, ask what made the client come in today.
G. Determine how the client has been coping with stress up until today (exercise, medication).
H. Review the client's history of other medical problems.
I. Does anyone else in the family have the same problem? How are they treated?

PHYSICAL EXAMINATION

A. Check pulse, respirations, blood pressure, and weight.
B. Inspect:
1. Observe general appearance; note grooming, dress, ability to communicate, body movements, nail biting, playing with hair, and inability to sit still.
C. Administer mental exam of choice:
1. *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision* (*DSM-5-TR)* Diagnostic Criteria for Generalized Anxiety Disorder, available from the American Psychiatric Association (APA; *DSM-5*, 2013).
2. Beck Anxiety Scale.
3. The GAD Seven-Item Scale (GAD-7).
4. Elder-specific self-report measures of anxiety: Abbreviated Math Anxiety Scale (AMAS), Geriatric Anxiety Inventory (GAI), Goal Attainment Scale (GAS), Worry Scale (WS).
D. Physical examination as indicated by somatic complaints.

DIAGNOSTIC TESTS

A. Blood alcohol.
B. Thyroid profile.
C. Blood glucose.
D. Medication level (theophylline, etc.) if applicable.
E. Urine drug screen.
F. Additional testing related to suspected physical pathology.

DIFFERENTIAL DIAGNOSES

A. Anxiety.
B. Psychiatric syndrome:
1. Mood disorders, such as depression or bipolar disorder.
2. Psychotic disorders.
3. Somatoform disorders (characterized by physical complaints lacking known medical basis or demonstrable physical finding in the presence of psychological factors judged to be etiologic or important in the initiation, exacerbation, or maintenance of the disturbance).
4. Personality disorders.
5. Alcoholism and drug abuse/dependence.
6. Adjustment disorders (posttraumatic stress disorder [PTSD]).
C. Medical conditions. Anxiety syndromes mimic many medical illnesses, including intracranial tumors, menstrual irregularities, hypothyroidism, hyperparathyroidism and hypoparathyroidism, postconcussion syndrome, psychomotor epilepsy, and Cushing disease:
1. Hypoglycemia if anxiety is chronic.
2. Hypothyroidism.
3. Hyperthyroidism: Rapid-onset anxiety could be a symptomof hyperthyroidism.
4. Tumor.
5. Cushing disease.

PLAN

A. General interventions:
1. Treat medical conditions as appropriate.
2. Refer the client for cognitive behavioral therapy. Counseling is effective for learning new techniques to help with alleviating symptoms. Cognitive behavioral therapy may be effective alone and/or may also be used as adjunct to medication treatment.
3. Encourage the client to perform self-calming techniques such as deep breathing/relaxation techniques and exercise.
B. Client teaching: *See Client Teaching Guides for this chapter, "Sleep Disorders/Insomnia" and "Alcohol and Drug Dependence."*
C. Pharmaceutical therapy:
1. Selective serotonin reuptake inhibitors (SSRIs) or selective serotonin-norepinephrine reuptake inhibitors (SNRIs):
a. Fluoxetine (Prozac), 5 to 10 mg/d orally initially; usual dose 20 to 80 mg/d; long half-life; alters metabolism of cytochrome P-450 2D6-cleared agents; use caution.
b. Paroxetine (Paxil), 10 mg/d orally initially; usual dose 25 to 50 mg/d.
c. Sertraline (Zoloft), 25 to 50 mg/d orally initially; usual dose 50 to 200 mg/d.
d. Venlafaxine (Effexor), 37.5 mg/d initially; usual dose 75 to 300 mg/d.
e. Citalopram (Celexa), 5 to 10 mg/d orally initially; usual dose 20 to 40 mg/d. Caution: risk for QT prolongation; contraindicated in clients with congenital long QT syndrome and should not exceed 20 mg every day if prescribed to clients also taking CYP2C19 inhibitors (cimetidine, fluconazole, and omeprazole).
f. Escitalopram (Lexapro) 10 mg/d orally initially; usual dose 10 to 20 mg/d.
g. Duloxetine (Cymbalta), 20 mg/d orally initially; titrate to 30 to 60 mg/d.
h. These medications can take 4 to 6 weeks to take full effect.
i. Warn clients that they should not stop these medications abruptly; they should taper off gradually.

2. Nonbenzodiazepine anxiolytic:
   a. Buspirone (Buspar), 7.5 mg BID.
   b. May increase by 5 mg/d orally every 2 to 3 days.
   c. Usual range: 20 to 30 mg orally every day, maximum 60 mg orally every day.
   d. Therapeutic effects may be delayed from 1 to 4 weeks.
3. Short-acting benzodiazepines:
   a. Alprazolam (Xanax), 0.25 to 0.5 mg orally two to three times daily.
   b. Clonazepam, 0.25 to 0.5 mg orally one or two times daily, titrated up to 1 mg two to three times daily as needed
   c. Lorazepam (Ativan), 0.5 to 2 mg, up to 6 mg orally every day in divided doses; maximum dose of 10 mg/d in divided doses:
      i. Use for initial short-term stabilization while simultaneously prescribing buspirone or SSRI/SNRI because therapeutic effects are delayed from 1 to 4 weeks.
      ii. Limit use to several weeks to a few months to prevent dependence.

FOLLOW-UP

A. Follow up in 1 to 2 weeks to assess the client's status.
B. Follow up every 2 to 4 weeks after to evaluate the client's progress.
C. Assess suicide potential with every office visit.

CONSULTATION/REFERRAL

A. Refer to a psychiatric clinician for complex medication management and psychotherapy after initial assessment.
B. If the client expresses suicidal thoughts, immediately refer for an emergency mental health evaluation; this may be through the local ED or mental health center.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
   1. Caution should be used in prescribing medications for anxiety during pregnancy; the benefits must be weighed against the risks.
   2. If the client becomes pregnant while taking these medications, taper medication dose instead of ceasing abruptly.
B. Geriatrics:
   1. Anxiety is often unrecognized and inadequately treated in this population because of concomitant medical illness, overlap with cognitive disorders, comorbid depression, ageism, and cohort effects.
   2. Older adult clients may be hesitant to discuss mental health concerns and needs due to the history of cultural stigma associated with mental illness.
   3. Start with the lowest dose of medication and increase slowly.
   4. Older adults may have increased sensitivities to benzodiazepines and decreased metabolism of long-acting agents. Benzodiazepines increase risk of cognitive impairment, delirium, falls, and fractures. Avoid short- and intermediate-acting benzodiazepines.
   5. SSRIs and SNRIs are to be used with caution because they may exacerbate or cause syndrome of inappropriate antidiuretic hormone excretion or hyponatremia: Monitor sodium level closely when starting or changing doses in older adults.
C. Partners:
   1. If available in the community, provide resources for partners, such as the National Alliance for the Mentally Ill.
   2. Psychotherapy for the client and partner is often helpful.

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ATTENTION DEFICIT HYPERACTIVITY DISORDER

DEFINITION

A. Attention deficit hyperactivity disorder (ADHD) is a syndrome that consists of a cluster of behaviors that emerge early in a child's life and persist over time. Excessively high levels of motor activity and problems with attention span, concentration, and/or impulsivity characterize ADHD. Up to 50% of clients with ADHD also have other psychosocial disorders such as oppositional defiant disorder (ODD), depression or bipolar disorder (develops in 25% of children with ADHD), posttraumatic stress disorder (PTSD), or Tourette syndrome. Diagnosis of ADHD in adulthood requires childhood diagnosis, whether treated or not.
B. The diagnostic criteria for ADHD include symptoms of inattention and symptoms of hyperactivity/impulsivity, as published by the American Psychiatric Association (APA), in the *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition (*DSM-5;* 2013). These criteria are available at www.cdc.gov/ncbddd/adhd/diagnosis.html.

INCIDENCE

A. 3% to 5% of all school-age children and 2.5% in adults.
B. Occurs in all races and socioeconomic groups.
C. Boys are more likely than girls to have symptoms by a ratio of 2:1.
D. Not uncommon for this disorder to persist into adulthood.
E. Approximately 33% of children with learning disabilities also have ADHD.

PATHOGENESIS

A. Unknown, but several studies suggest a biochemical basis involving deficits in the availability of neurotransmitters to the frontal–orbital circuits of the neurobehavioral regulatory systems of the brain. ADHD may also be inherited.
B. Associated problems with ADHD include academic, social, and emotional problems.

PREDISPOSING FACTORS

A. A close relative with a mood disorder, anxiety, or ADHD.
B. Brain trauma.
C. Selective therapeutic regimens, such as intrathecal chemotherapy.
D. Recent studies have suggested a possible link to maternal smoking, alcohol abuse, or other toxins during pregnancy.
E. Some perinatal influences have been theorized to be connected to ADHD, including fetal distress, prolonged labor, prematurity, and perinatal asphyxia.

COMMON COMPLAINTS

A. Hyperactivity, impulsivity, and/or inattentiveness.
B. Poor work performance.
C. Difficulty paying attention to something that bores them.

OTHER SIGNS AND SYMPTOMS

A. Inattentive and easily distracted when completing tasks: daydreams, does not finish work, loses things, has difficulty concentrating.
B. Impulsive: risk taking, impatient, very emotional.
C. Hyperactivity/over activity: speech and motor skills overactive.
D. Difficulty with learning, poor performance in school/work.
E. Difficulty controlling emotions.

SUBJECTIVE DATA

A. Determine the presenting symptoms and ascertain when symptoms first began (childhood, adolescence).
B. Note duration of symptoms:
  1. Have they been present for at least 6 months?
  2. Ask regarding specific behaviors as listed in the diagnostic criteria.
C. Review settings in which symptoms are present (home, school, etc.).
D. Discuss the medical history. Were symptoms present during childhood treated or not?
E. Review the family, social, and school histories.
F. Review all current medications, including any over-the-counter (OTC) medications and herbals. Specifically review medication history for theophylline, prednisone, and albuterol.
G. Review history of substance use/abuse.
H. Review dietary, eating, and sleeping habits.
I. Review daily routines and habits: Stimulation may come from television, video, and computer viewing.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure, weight, and height.
B. Inspect:
  1. Observe overall appearance. Note behavior and interactions with others.
  2. Inspect the skin, eyes, ears, nose, and throat.
C. Auscultate:
  1. Heart.
  2. Lungs.
  3. Abdomen.
D. Palpate:
  1. Neck.
  2. Thyroid.
  3. Chest.
  4. Abdomen.
E. Neurologic examination:
  1. Hearing/vision evaluation: Evaluate constant, involuntary movement of the eyes (nystagmus).
  2. Evaluate coordination difficulties/impaired motor skills.
  3. Evaluate visual-motor control problems (hand–eye coordination).

DIAGNOSTIC TESTS

A. Complete blood count (CBC) to rule out iron-deficiency anemia.
B. Lead level.
C. Thyroid studies to rule out other organic problems.
D. CBC with differential (if indicated).
E. Administer the Adult ADHD Screening Tool. This tool is a symptom checklist instrument consisting of the 18 *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, Text Revision (*DSM-IV-TR*) criteria for ADHD. This can be located at add.org/wp-content/uploads/2015/03/adhd-questionnaire-ASRS111.pdf.
  1. Recommend referring to mental health professional for neuropsychological testing for diagnosis.
F. EEG may be considered.
G. MRI may be considered to rule out organic diagnoses.
H. Consider urine drug screen.

DIFFERENTIAL DIAGNOSES

A. Attention deficit disorder (ADD)/ADHD.
B. Anxiety.
C. Depression.
D. Obsessive-compulsive disorder.
E. Hyperthyroidism/hypothyroidism.
F. Substance use/abuse disorder (caffeine, tobacco, alcohol, drugs).

PLAN

A. General interventions:
  1. Administer assessment tool for ADD.
  2. Effective behavioral management and family and/or individual therapy are strongly encouraged.
  3. Counseling and behavior therapy are helpful to manage day-to-day problems and routines.
B. Client teaching: *See Client Teaching Guide for this chapter, "ADHD: Coping Strategies for Teens and Adults."*
  1. Provide ADHD resources for client.
  2. Counseling/life coaching encouraged to assist client in learning new skills to handle daily situations and create new routines/habits for controlling behavior.
  3. Encourage client to learn organizational skills to assist in daily routines.
  4. Diet high in protein and complex carbohydrates is encouraged to improve concentration and attempt to prevent mood swings.
C. Pharmaceutical therapy:
  1. Begin with short-acting stimulants. Advance dose as needed for desired result.
  2. Consider adding a long-acting agent to a short-acting agent if needed.
  3. Long-acting agent used alone.
  4. First-line agents: rapid onset with short duration:
    a. Methylphenidate (Ritalin, Methylin, Metadate, Concerta).
    b. Dextroamphetamine (Dexedrine, ProCentra, Zenzedi).
    c. Amphetamine-dextroamphetamine (Adderall).
  5. Rapid onset with long duration:
    a. Methylphenidate (Concerta, Ritalin LA, Ritalin SR, Daytrana patch, Metadate CD, Metadate ER, Methylin ER, Quillivant XR).
    b. Amphetamine-dextroamphetamine (Adderall XR): duration 8 to 12 hours.
    c. Lisdexamfetamine (Vyvanse): duration 10 hours.
  6. Medications to use if substance abuse is a concern:
    a. Atomoxetine (Strattera).
    b. Lisdexamfetamine dimesylate (Vyvanse).
    c. Bupropion (Wellbutrin).
  7. Medications recommended for comorbidity of depression:
    a. Bupropion (Wellbutrin, Wellbutrin SR, Wellbutrin XL).
    b. Venlafaxine (Effexor, Effexor XR).

8. Medications recommended for comorbidity of anxiety.
   a. Use combination of a stimulant and selective serotonin reuptake inhibitor (SSRI)/serotonin-norepinephrine reuptake inhibitor (SNRI).
9. Medication doses:
   a. Methylphenidate (Ritalin): starting dose in adults: 10 mg/d; may increase up to 60 mg in two or three divided doses before meals.
   b. Methylphenidate HCl (Ritalin LA): 20 mg in the morning; may increase dose 10 mg weekly; maximum dose 60 mg/d.
   c. Methylphenidate HCl liquid (Quillivant XR): starting dose: 10 to 20 mg orally in the morning; increase the dose weekly by 10 to 20 mg; maximum daily dose 60 mg/d.
   d. Methylphenidate HCl (Concerta): adolescents/adults: 18 mg/d with maximum 72 mg every day; increase by 18-mg increments weekly as needed; contraindicated with severe anxiety.
   e. Dexmethylphenidate HCl (Focalin XR): for adults: 40 mg every day in the morning.
   f. Lisdexamfetamine dimesylate (Vyvanse): 30 mg every day in a single dose in the morning; may be increased by 10 mg/d; maximum dose of 70 mg every day in the morning.
   g. Atomoxetine (Strattera): not a controlled substance; may take a few weeks to see the full effect. Start 40 mg /d, advance to 80 mg after 1 to 4 weeks, maximum of 100 mg/d.
      i. Should be taken daily at the same time every day to avoid SNRI effects. Food and Drug Administration (FDA)-approved indication for adults.
   h. Side effects of these medications include anorexia, insomnia, stomach pain, growth suppression, and tics. If weight loss occurs, give medication after meals.
   i. If psychostimulants are not effective, other medications used include antihistamines, guanfacine (Tenex), clonidine, carbamazepine (Tegretol), divalproex (Depakote), beta-blockers, and antidepressants.
   j. Be cautious when prescribing other medications concurrently.

FOLLOW-UP

A. At 1 month, inquire about improvements in each area of life and duration of the medication's actions.

B. Medication dose and/or frequency may need to be altered.

C. When medication schedule is stable, subsequent visits can be every 3 months.

CONSULTATION/REFERRAL

A. Refer to specialist/psychiatrist for evaluation/workup for ADHD.

B. Consult with a psychiatric provider to help initiate and/or coordinate medications.

C. Refer the client for individual and/or family therapy if indicated.

INDIVIDUAL CONSIDERATIONS

A. Adults:

1. Up to half of the affected children have some symptoms of ADHD that persist into adulthood. Adults tend to outgrow the "hyperactivity" aspect of ADD; however, they may still require treatment for the ADD.
2. Teens and adults benefit from specific coping strategies for ADHD.

RESOURCES

Adult ADHD Self-Report Scale (ASRS-v1.1) Symptom Checklist Instructions: https://add.org/wp-content/uploads/2015/03/adhd-questionnaire-ASRS111.pdf

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BIPOLAR DISORDER

DEFINITION

A. Bipolar disorder, commonly called manic-depressive disorder, is a psychiatric disorder that causes changes in moods, thoughts, and behaviors. These changes may range from mania (excessive energy, euphoria, racing thoughts, decreased need for sleep, grandiosity, pressured speech, and impulsive behavior) to depression (low energy, sadness, diminished interest and pleasure in most activities, recurrent thoughts of death, changes in sleep patterns, cognitive impairment, and difficulty carrying out daily activities).

B. There are six types of bipolar disorders that affect adult and geriatric clients.

1. Bipolar I disorder is diagnosed when the individual experiences a manic episode, which may have been preceded or followed by a hypomanic or depressive episode.
2. Individuals with bipolar II disorder have a pattern of depressive episodes and hypomanic episodes (no mania). Often symptoms of depression and hypomania, especially agitation, irritability, and verbal impulsivity are displayed in the same episode (mixed).
3. Cyclothymic disorder is a chronic pattern of hypomanic and depressive symptoms that do not meet the full criteria for hypomania or depressive episodes. Symptoms occur for at least 2 years (1 year in children and adolescents).
4. The unspecified bipolar and related disorder category applies to situations in which there are symptoms that are characteristic of bipolar disorder, but they do not meet the full criteria. There may be insufficient information to make a diagnosis or underlying medical conditions or substance use contributing to the condition. Specific criteria for bipolar disorder as published by the American Psychiatric Association (APA), *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition (2013) are available at the National Institute for Mental Health website: www.nimh.nih.gov/health/topics/bipolar-disorder/index.shtml.
5. Early-onset bipolar disorder (EOBD) is common in elderly clients with long-standing clinical history. Due to the onset of the disorder in early life, EOBD clients usually experience more severe and atypical symptoms in their first manic episode, as well as higher rates of psychotic symptoms and rapid cycling.
6. Late-onset bipolar disorder (LOBD) is common in elderly clients with first occurrence of mania in old age. LOBD mania is usually characterized by fewer and rather milder manic symptoms compared with EOBD. However, ▶

LOBD clients show more frequent irritable behaviors, more often develop treatment resistance, and have higher mortality rates.

INCIDENCE

A. 5.7 million or 2.6% of adults ages 18 or older in the United States.
B. Median age of onset is 25, although can range from childhood to late onset (50s).
C. Bipolar disorders occur in all races and socioeconomic groups.
D. Males may present with symptoms of mania earlier than females. Females are more likely to present with symptoms of depression and experience more rapid cycling.
E. Bipolar disorder is the sixth leading cause of disability in the world.
F. One in five individuals diagnosed with bipolar disorder will die by suicide.
G. The general population aged 65 years and older: The 1-year prevalence of bipolar I disorder is approximately 0.4% and the lifetime rate is 0.8%. These rates were less than rates in younger individuals. Approximately 3% of nursing home residents are diagnosed with some form of bipolar disorder.

PREDISPOSING FACTORS

A. A family history of bipolar disorder or schizophrenia.
B. Periods of high stress.
C. Drug or alcohol abuse.
D. Major life changes such as the death of a loved one or a traumatic experience.

COMMON COMPLAINTS

A. Individuals most frequently seek treatment for depressive episodes.
B. Poor work performance, unfinished tasks.
C. Mood swings, irritability, or anger episodes.
D. Social problems.
E. Sleep disturbance.

SIGNS AND SYMPTOMS

A. Mania:
  1. Symptoms last at least 1 week.
  2. Inflated self-esteem, grandiosity.
  3. Decreased need for sleep, talkative, racing thoughts, distractibility.
  4. Increase in goal-directed activity.
  5. Impulsive behavior (buying sprees, sexual indiscretions, poor business decisions).
  6. Symptoms cause marked impairment in functioning and may require hospitalization.
B. Hypomania:
  1. Symptoms last at least 4 days.
  2. Symptoms are the same as mania but are not severe enough to cause marked impairment.
  3. Frequently displays irritability and agitation.
C. Depression:
  1. Symptoms last for at least 2 weeks.
  2. Symptoms are like major depressive disorder: depressed mood most of the day, diminished interest in pleasure, changes in weight, sleep disturbance, restlessness or low energy, fatigue, feelings of worthlessness, guilt, and burdensomeness, cognitive changes, thoughts of death.
D. Impulse control:
  1. Client may present themselves as extremely happy and sociable.
  2. May have rapid mood changes, such as irritability or aggression when wishes are denied, especially if using substances.
E. Suicidal or homicidal thoughts or acts. Any statements made by the client, such as "Life isn't worth living, I wish I were dead, I don't deserve to be alive, I can't deal with this," should be taken seriously.
  1. Refer the client for immediate mental health assessment and treatment.

SUBJECTIVE DATA

A. Review the onset, duration, and course of presenting symptoms.
B. Review any history of depression, mania, or mood disorders.
C. Determine how the previous mood disorder was treated, if applicable.
D. Evaluate the client's suicide potential. Ask: "Have you ever thought of hurting yourself or others?" Does the client have a current suicide plan or vague ideas of suicide? Has the client had any history of suicide attempts? If so, evaluate how life-threatening they were.
E. Review the client's medical history.
F. Review the client's drug history for prescription, over-the-counter (OTC), and recreational/illicit drug use (how much, how long, how often), and review their history of alcohol consumption (how much, how long, how often).
G. Assess client's compliance with prescribed medications.
H. Review the client's history for recent major life changes, such as pregnancy, death, divorce, or any loss that may be normal throughout the stages of life. The client's *perception* of the loss is what is important.
I. Review dietary intake since the symptoms have begun.
J. Establish usual weight, review weight gain/loss, and in what time span.
K. Review the client's activities of daily living (ADLs). Does the client get up and dress daily, perform daily hygiene, put on makeup?
L. Review how many hours of sleep and quality of sleep per day.
M. Review the disruption of usual activities: return to work, return to school, exercise. Has the client been engaging in activities outside their norm?
N. Assess mood patterns, rate of cycling, seasonal changes.
O. Review occupational/home exposure to neurodegenerative products.
P. Review any exposures to infectious diseases, including Lyme disease. Does anyone else such as family, friends, or coworkers have similar symptoms?
Q. If female, review for symptoms of menopause (sleep disturbances, irregular menses/amenorrhea, hot flashes, vaginal dryness, dyspareunia).

PHYSICAL EXAMINATION

A. Check, pulse, respirations, blood pressure, and weight.
B. Inspect:
  1. Observe overall appearance; note grooming, tone of voice, eye contact, conduct of client during communication, and breath (smell of alcohol).
  2. Complete neurologic examination with screening tool of choice.

3. Complete dermal examination for signs of substance use (refer to "Substance Use Disorders" section of this chapter).

C. Palpate:

1. Palpate the neck and thyroid; note the goiter.

2. Palpate the axilla and groin for lymphadenopathy (infectious etiology).

3. Check the joints for swelling and arthritis and range of motion (ROM; rule out musculoskeletal cause).

D. Auscultate:

1. Heart, lungs, and abdomen (as applies to physical complaints).

E. Mental status exam:

1. Complete Mental Status Exam (MSE), including appearance, affect/mood, thought content, perception, suicide/self-destruction, homicide/aggression, judgment/insight, and cognition.

DIAGNOSTIC TESTS

A. Complete blood count (CBC) to rule out iron-deficiency anemia.

B. Urine/serum drug screen.

C. Thyroid studies to rule out other organic problems.

D. Comprehensive metabolic panel (CMP).

E. Erythrocyte sedimentation rate (ESR).

F. Liver and lipid panel.

G. Vitamin B_{12} and folic acid level.

H. Tests for syphilis including rapid plasma reagin.

I. Test for HIV.

J. Refer for psychological testing to confirm diagnosis, to rule out other mental health disorders, and to assess for social/emotional adjustment and presence of learning disabilities.

K. EKG.

L. EEG may be considered.

M. CT scan or MRI may be considered to rule out organic diagnoses.

DIFFERENTIAL DIAGNOSES

A. Bipolar disorder.

B. Substance-induced mood disorder, including caffeine, alcohol/illicit drugs.

C. Head trauma.

D. Hyperthyroidism/hypothyroidism.

E. Lead poisoning.

F. Other behavioral/psychological disorders (pervasive developmental delay, oppositional defiant disorder, seasonal affective disorder [SAD], anxiety disorder, schizoaffective disorder, schizophrenia, and personality disorder).

G. Posttraumatic stress disorder (PTSD).

H. Attention deficit hyperactivity disorder (ADHD).

I. Seizure disorder, nonconvulsive.

J. Medical conditions (menopause, neurosyphilis, multiple sclerosis, Lyme disease).

K. Adverse reactions to medications (theophylline, prednisone, albuterol, Levaquin, and antidepressant medications; may induce mania).

PLAN

A. General interventions:

1. Keep the client safe from self-harm.

2. Treat physical/laboratory findings. Recommend dietary change, iron supplements, hormone replacement therapy per findings (see related chapters).

B. Client teaching:

1. Encourage the client to take medications as prescribed. Educate the client that some medications may take time to get into the system to work and time should be allowed to see the effects of the medication. Review side effects.

2. Encourage the client to express feelings or worsening of symptoms if this occurs prior to next appointment. Have the client make a client contract with you that they will not hurt themselves or others and that if they begin having these thoughts they will contact you or go to the nearest ED.

3. Encourage exercise on a daily basis for 20 to 30 minutes to increase energy and enhance a feeling of well-being.

4. Encourage the client to get at least 7 to 8 hours of sleep each night. If sleep is a problem, address this issue with the client.

5. Avoid caffeine at night and/or watching television late at night.

6. Encourage the client to seek counseling with a professional counselor. Refer to appropriate site (psychologist, psychiatrist, group therapy, etc.). Offer local resources to the client.

7. Advise client to participate in activities to enhance interpersonal relationships and build self-esteem. Include family and friends in recommended therapies and advise them to encourage the client to participate in activities to enhance self-esteem.

8. Once the client is feeling better, encourage continued use of medication, activities, and resources.

Bipolar disorder is a chronic condition requiring long-term, continuous treatment. Untreated episodes of mania and depression may become more severe and more difficult to treat over time.

C. Pharmaceutical therapy. Medication choice depends on the current episode: mania/depression.

1. Mood stabilizers:

a. Divalproex sodium (Depakote, Depakote ER):

i. Indicated for mania.

ii. Initial dose of 25 mg/kg/d mg every day in divided doses.

iii. Increase as quickly as possible to achieve target plasma concentrations of 50 to 125 mcg/mL.

iv. Maximum concentration achieved within 14 days.

v. Maximum recommended dose is 60 mg/kg/d.

vi. Drug–drug interactions: may increase concentrations of clonazepam, diazepam, lamotrigine, and carbamazepine. Aspirin increases Depakote blood levels.

vii. Labs: Check valproic acid level, liver function test (LFT), and CBC after 1 week, then at 1 to 2 months, then every 6 to 12 months thereafter.

viii. Side effects: dizziness, sedation, nausea, tremor, thrombocytopenia, elevated liver enzymes, polycystic ovarian syndrome, hepatotoxicity (rare).

ix. Monitoring: serum drug levels during treatment initiation; monitor weight and menstrual history every 3 months for the first year, then annually.

b. Carbamazepine ER (Equetro):

i. Indicated for mania and mixed episode.

ii. Initial dose of 200 mg BID.

iii. Adjust dose in 200-mg increments. Maximum dose: 1,600 mg/d.

iv. Capsules may be taken whole or opened and sprinkled on food.

v. Do not use with monoamine inhibitors oxidases (MOAIs) or within 14 days of using an MOAI.
vi. Side effects: dizziness, somnolence, dry mouth, constipation, aplastic anemia, agranulocytosis, rash, toxic epidermal necrolysis (TEN), and Stevens–Johnson syndrome (SJS; clients of Asian ancestry have a 10-fold greater risk of TEN/SJS).
vii. Monitoring: serum drug levels during treatment initiation and as clinically indicated; CBC, LFT, electrolytes, blood urea nitrogen (BUN), and creatinine monthly for 3 months, then annually.
viii. Medications that are discontinued should be gradually tapered off to avoid side effects.

c. Lithium:
i. Indicated for mania and maintenance.
ii. **Lithium workup** prior to initiating the medication: EKG, BUN/creatinine, urinalysis, CMP, and thyroid-stimulating hormone (TSH).
iii. Initial dose of 300 mg BID.
iv. Lithium levels drawn 2x per week with dose adjustments in 300-mg increments until target serum levels are reached: target serum levels 0.6 to 1.2 mEq/L. Levels should be drawn 12 hours after last dose.
v. Lithium toxicity: nausea, vomiting, diarrhea, muscular weakness, lack of coordination, giddiness, and large output of dilute urine; may involve multiple organs and organ systems.
vi. Monitoring: serum drug levels every 3 to 6 months once stable; electrolytes, BUN, and creatinine every 3 to 6 months to exclude renal impairment; TSH, calcium, and weight after 6 months, then annually.

d. Lamotrigine (Lamictal):
i. Indicated for maintenance therapy.
ii. Must titrate slowly due to risk of rash/SJS.
iii. Target dose of 200 mg to 400 mg.
iv. Titration dose adjustments required if client is on Depakote (slower titration) or carbamazepine (faster titration).
v. Side effects: nausea, insomnia, somnolence, rash, leg cramps, aphasia, and SJS.

2. Atypical antipsychotics:

Potential side effects for all atypical antipsychotics

- Extrapyramidal and/or withdrawal symptoms in neonates with third trimester exposure
- Lower seizure threshold
- Leukopenia, neutropenia, and agranulocytosis
- Metabolic changes including hyperglycemia/ diabetes, dyslipidemia, weight gain
- Orthostatic hypotension.
- Tardive dyskinesia
- Cognitive and motor impairment
- Increased risk of stroke in older adult clients with dementia-related psychosis; older adult clients with a history of cerebrovascular accident (CVA) treated with antipsychotic
- Medications are at an increased risk of death
- Suicidal thinking
- Neuroleptic malignant syndrome (NMS): high fever, stiff muscles, confusion, sweating, changes in pulse, heart rate, and elevated blood pressure
- Labs: Monitor body mass index (BMI), waist circumference, hemoglobin A1C (HbA1C), fasting plasma glucose, fasting lipid panel at baseline, 3 months, and then annually.

a. Aripiprazole (Abilify):
i. Indicated for mania, mixed episodes, and maintenance.
ii. Initial dose: 15 mg every day (10–15 mg if used in addition to a mood stabilizer). Once daily with or without meals.
iii. Available in tablets, orally disintegrating tablets, oral solution, and injectable 9.75 mg/1.3 mL single-dose vial.
iv. Maximum recommended dose: 30 mg every day.
v. Drug–drug interactions: known CYP2D6 poor metabolizers, strong CYP2D6 *or* CYP3A4 inhibitors: ½ dose. Known CYP2D6 poor metabolizers *and* strong CYP3A4 inhibitors: ¼ dose.
vi. Common side effects: akathisia (restlessness), weight gain, nausea, constipation, headache, dizziness, and stuffy nose.

b. Ziprasidone (Geodon):
i. Indicated as monotherapy for mania and mixed episodes and as an adjunct to lithium or valproate for maintenance.
ii. Initial dose: mania: 40 mg BID, with increase to 60 mg to 80 mg BID on day 2 of treatment. **Giving oral doses with food increases absorption up to twofold.**
iii. Available in capsules and injectable 20 mg/mL single-dose vial.
iv. Maximum recommended dose: 60 mg to 80 mg BID with food. Some client doses up to 240 mg/d may be necessary.
v. Drug–drug interactions: Do not give with other medications known to cause QT prolongation.
vi. Side effects: QT prolongation. Do not use in clients with recent heart failure, recent heart attack, or arrhythmias. Other more common side effects include akathisia (restlessness), sleepiness, weight gain, nausea, constipation, headache, dizziness, stuffy nose. Less common includes severe cutaneous adverse reactions (SCAR) such as SJS.

c. Risperidone (Risperdal/Risperdal Consta):
i. Indicated as monotherapy for mania and mixed episodes, and as an adjunct to lithium or valproate for maintenance therapy.
ii. Dosing:
1) Risperidone: 2 to 3 mg every day with titration in 1-mg increments at intervals of 24 hours or greater. Target dose of 1 to 6 mg every day. Maximum dosing 6 mg/d. Once or twice daily with or without meals. Available in tablets, orally disintegrating tablets, and oral solution 1 mg/mL.
2) Risperdal Consta: 25 mg intramuscularly (IM) every 2 weeks with dose increase in 4-week intervals. Maximum dosing 37.5 to 50 mg IM every 2 weeks. Oral dose of risperidone should be used along with the first injection of Risperdal Consta and continued for 3 weeks. Available in dose strengths of 12.5 mg, 25 mg, 37.5 mg, or 50 mg.
iii. Drug–drug interactions: CYP2D6 enzyme inducers (carbamazepine), may need to increase dose up to double the client's usual dose. CYP2D6 enzyme inhibitors (paroxetine, fluoxetine), dose of ▶

risperidone should be titrated slowly and dose may need to be decreased.

iv. Side effects: dizziness, drooling, nausea, tiredness, weight gain, akathisia, tremor, blurred vision, QT prolongation, and increased prolactin levels. Dose adjustment recommended for clients with severe renal or hepatic impairment. Monitor prolactin levels along with other recommended labs.

d. Asenapine (Saphris): dissolvable; available as sublingual tabs only.

i. Indicated as monotherapy for mania and mixed episodes, and as an adjunct to lithium or valproate for maintenance therapy.

ii. Initial dose: 5 to 10 mg sublingually BID.

iii. Available in sublingual tablets only.

iv. Maximum recommended dose: 10 mg BID.

v. Drug–drug interactions: antihypertensive drugs: Saphris may cause hypotension. Paroxetine (CYP2D6 substrate and inhibitor): Reduce paroxetine dose when used in combination with Saphris.

vi. Common side effects: orthostatic hypotension, syncope, akathisia (restlessness), weight gain, nausea, constipation, headache dizziness, stuffy nose. Contraindicated in clients with severe hepatic impairment.

e. Quetiapine (Seroquel/Seroquel XR):

i. Indicated as monotherapy for bipolar mania, mixed, and depressive episodes, and as an adjunct to lithium or valproate for maintenance therapy.

ii. Dosing: Mania*:* 50 mg BID. Dose adjustments in increments of 100 mg every day up to total of 400 mg. Further dose adjustments up to 800 mg/d should be in increments of no greater than 200 mg/d. Target dose: 400 to 800 mg every day. Maximum recommended dose: 800 mg/d. Depression*:* Administer once daily at bedtime. Initial dose of 50 mg at bedtime. Increase to 100 mg on day 2 with further increases of 100 mg every day up to 300 mg/d. Target dose: 300 mg/d. Maximum daily dose: 300 mg.

iii. Quetiapine extended-release: Bipolar mania/mixed episode: initial dose of 300 mg every day in the evening without food or with light meal. Target dose 400 to 600 mg every day. Maximum dosing 600 mg every day. Bipolar depression: 50 mg every day in the evening without food or with a light meal. Target dose: 300 mg every day. Maximum recommended dose: 300 mg every day.

iv. Drug–drug interactions: Dose should be increased up to fivefold of the original dose when used in combination with a potent CYP3A4 inducer. Titrate dose based on clinical response and tolerability. Dose should be reduced to 1/6 of the original dose when used in combination with a potent CYP3A4 inhibitor. Quetiapine may cause hypotension, thus enhancing the effects of antihypertensive medications.

v. Side effects: somnolence, dry mouth, dysarthria, increased appetite, weight gain, nausea, constipation, dizziness, stuffy nose, joint pain, QT prolongation, hypotension.

f. Olanzapine (Zyprexa, Zydis):

i. Indicated for mania, mixed episodes, and maintenance monotherapy.

ii. Initial dose: 10 to 15 mg once daily with or without meals, with dose adjustments of 5 mg every day.

iii. Available in tablets, orally disintegrating tablets, and injectable 10 mg/2.1 mL single-dose vial.

iv. Maximum recommended dose: 20 mg every day.

v. Drug–drug interactions: Coadministration with diazepam may potentiate orthostatic hypotension. Known CYP1A2 inducers, such as carbamazepine, increase the clearance of olanzapine. Titrate dose based on clinical response and tolerability. When used in combination with CYP1A2 inhibitors, such as fluvoxamine, lower doses of olanzapine should be considered.

vi. Side effects: postural hypotension, akathisia (restlessness), increased appetite, weight gain, somnolence, nausea, constipation, headache, dizziness, stuffy nose, and tremors.

g. Olanzapine/fluoxetine combination (Symbyax):

i. Indicated for bipolar depression.

ii. Initial dose: 6 mg/25 mg capsule daily in the evening (mg equivalent olanzapine/mg equivalent fluoxetine). Dose adjustments as needed and tolerated. Target dose: within the approved dosing range of 6/25, 6/50, 12/25, 12/50 mg.

iii. Maximum recommended dose: 18 mg/75 mg every day in the evening.

iv. Drug–drug interactions: same as with olanzapine and fluoxetine individually.

v. Common side effects: akathisia (restlessness), increased appetite, fatigue, somnolence, peripheral edema, blurred vision, weight gain, nausea, constipation, headache, dizziness, stuffy nose.

h. Chlorpromazine (Thorazine):

i. Indicated for mania/mixed episodes.

ii. Initial dose: 25 mg orally TID with gradual increase until effective dose is reached. Target dose: 400 to 500 mg every day in divided doses.

iii. Available in tablets, spansule/sustained-release, single and multidose vials for IM injection, and suppositories.

iv. Maximum recommended dose: 1,000 mg every day.

v. Drug–drug interactions: multiple drug–drug interactions, alpha-blockers, anticholinergic/antispasmodic drugs. Avoid use with other medications that may cause QT prolongation or respiratory depression.

vi. Side effects: drowsiness, postural hypotension, akathisia (restlessness), dystonia, tremor, weight gain, nausea, constipation, headache, dizziness, stuffy nose, QT prolongation.

vii. Other better tolerated agents are generally preferred. May be considered if preferred agents are unavailable.

3. Antidepressants:

a. Use of traditional antidepressants to treat bipolar depression is controversial and not approved by the Food and Drug Administration (FDA).

b. Using antidepressants alone to treat bipolar depression is not recommended due to the potential to cause hypomania, mania, and rapid cycling.

c. Do not prescribe an antidepressant during manic or mixed episodes. ▶

4. Electroconvulsive therapy (ECT):
   a. Short-term treatment for severe manic or depressive episodes, especially when suicidal or psychotic symptoms are present, or when medication intervention is ineffective or not well-tolerated.
   b. ECT is among the safest treatments for severe mood disorders and is effective in up to 75% of clients.

FOLLOW-UP

A. Initially, monitor clients closely to assess for response to treatment, need for medication dose adjustment, side effects, and changes in level of functioning.

B. Monitor for emergence of suicidal thoughts, psychosis, mania, or depression.

C. As appropriate, order lab work to monitor serum blood levels of medications and assess for potential side effects, such as metabolic changes, elevated liver enzymes, decreased kidney functioning, cardiac changes, and blood dyscrasias.

D. After initial stabilization, follow-up appointments should be made every 1 to 3 months.

E. Client teaching related to importance of self-care and medication compliance.

CONSULTATION/REFERRAL

A. If client is experiencing suicidal/homicidal ideations, refer immediately to the nearest ED to be evaluated by a mental health professional for possible emergency admission.

B. Consult with the psychiatric provider to help coordinate care.

C. Refer the client for individual and/or family therapy if indicated.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Some of the medications used to treat bipolar disorder can potentially lower the effectiveness of hormone contraception. Clients should be counseled on the use of additional methods of pregnancy prevention.

2. Medications such as mood stabilizers cause an increased risk of birth defects in the first trimester. Individuals of childbearing age should be counseled on planned pregnancy to allow for medication adjustment and initiation of supportive therapy/close monitoring.

3. Stopping bipolar medications during pregnancy puts a client at high risk of relapse during the pregnancy and development of postpartum depression. Some bipolar medications pass through the breast milk. Risk versus benefits of medication use during pregnancy and breastfeeding should be discussed with client and significant other, if appropriate. Refer to a psychiatric specialist for consultation.

B. Partners/family:

1. Provide educational materials and information on local support resources to partners and family members. The National Alliance on Mental Illness provides family support groups and online resources in many rural and urban areas. Information may be accessed at www.nami.org.

C. Geriatrics:

1. Dementia and delirium are common in the older adults. Rule out underlying medical conditions and possible medication-induced mood/cognitive changes.

2. Caution should be used in the treatment of bipolar mania/mixed symptoms in geriatrics. Atypical antipsychotics increase the risk of stroke and death in older adult clients with a history of cerebrovascular accident and dementia-related psychosis.

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DEPRESSION

DEFINITION

A. Depression is a mental health disorder that interferes with a person's daily life. Depressive disorders are characterized by sad, empty, or irritable mood lasting at least 2 weeks in duration. Depression may be mild or severe, depending on signs and symptoms expressed, as well as the length of time symptoms are present. Depression affects multiple body systems and may impact one emotionally, cognitively, or physically, as well as behaviorally. Symptoms of depression may include difficulty sleeping, depressed mood, inability to function at work, change in appetite, and inability to enjoy activities that bring one pleasure. There are many forms of depression, and treatment varies depending on the specific diagnosis. Types of depression include major depressive disorder, persistent depressive disorder (dysthymia), premenstrual dysphoric disorder, disruptive mood dysregulation disorder, depressive disorder due to another medical condition, and other specified depressive disorders.

B. Diagnostic criteria include depressed mood most of the day, decreased pleasure in activities, weight loss or weight gain, insomnia or hypersomnolence, psychomotor agitation or retardation, fatigue or loss of energy, feelings of worthlessness or guilt, decreased concentration, thoughts of death, suicidal ideation, and suicide attempt or plan for self-harm.

INCIDENCE

A. One in 10 adults experience one or more episodes of depression during their lifetime. Depression can occur at any age. In the United States, the incidence of depression peaks in the 20s, but first onset in late life can occur. The lifetime risk is estimated to be as high as 30%. Females have a twofold to threefold higher rate of reported depression than males. Estimated rates of major depression in older adults are 3% to 5% for community-dwelling, 15% to 30% for those living in an institutional setting, and 13% for those living in nursing homes.

B. Only 10% to 25% of people with depressive disorders seek treatment. There is a high mortality from suicide if untreated (see "Suicide" section of this chapter).

C. No single causal factor has been identified. Depressive syndromes are so varied in course and symptomatology that a single cause is unlikely. Several factors appear to contribute, including genetics, neurochemical abnormalities (reductions in adrenergic or serotonergic neurotransmission), electrolyte disturbances, and neuroendocrine abnormalities such as hypothalamic, pituitary, adrenal cortical, thyroid, and gonadal functions. Higher concentrations of proinflammatory cytokines, tumor necrosis factor (TNF)-alpha, and interleukin (IL)-6 have been found in depressed subjects, compared with control subjects.

D. Depression is frequently a concomitant diagnosis with other physical or mental disorders. Personality and psychodynamic factors of depression include low self-esteem, self-criticism, and interpersonal loss. A childhood history of emotional, physical, and/or sexual abuse can also contribute to adult-onset depression.

PREDISPOSING FACTORS

A. Age (peak onset in 20s and older adults).
B. Lack of social support/living alone.
C. A history of early parental loss.
D. Female sex:
  1. Most common in childbearing years from ages 25 to 45 years.
  2. Premenstrual.
  3. Perimenopausal.
  4. Postpartum.
E. Family history of depression.
F. Frequent exposure to stressful events.
G. Nutritional disorders:
  1. Vitamin B_{12} deficiency.
  2. Vitamin D deficiency.
  3. Magnesium deficiency.
  4. Folate deficiency (may result in poor response to antidepressants).
  5. Iron deficiency.
H. Personality characteristics that include absence of hardiness factors in response to stress (lack of resilience, flexibility, and optimism).
I. Anger not dealt with and turned in on the self.
J. Negative interpretation of one's life experiences.
K. Poor physical health.
L. Postsurgical diagnosis of cancer.
M. Chronic pain.
N. Chronic medical problems such as hypothyroidism and hyperthyroidism, Cushing syndrome, hypercalcemia, hyponatremia, diabetes mellitus, lupus erythematosus, fibromyalgia, rheumatoid disease, and chronic fatigue syndrome.
O. Neurologic disorders such as stroke, subdural hematoma, multiple sclerosis, brain tumor, Parkinson disease, epilepsy, dementias, and Huntington disease.
P. Alcoholism/drug abuse or dependence/withdrawal.
Q. Infectious etiology such as mononucleosis and other viral infections, syphilis, HIV, and Lyme disease.
R. Side effect of prescription drugs, such as alpha-methyldopa, antiarrhythmics, benzodiazepines, barbiturates/central nervous system (CNS) depressants, beta-blockers, cholinergic drugs, corticosteroids, digoxin, H2 blockers, and reserpine.

COMMON COMPLAINTS

A. Lack of interest in pleasurable activities.
B. Digestive problems.
C. Chronic aches and pains that are not otherwise explained.
D. Always tired, fatigued.

OTHER SIGNS AND SYMPTOMS

A. Vegetative:
  1. Changes (increased or decreased) in sleep, appetite, and weight.
  2. Changes in appearance: poor grooming and hygiene.
  3. Poor eye contact, staring downward, flat affect.
  4. Loss of energy.
  5. Decreased interest in sex.
  6. Psychomotor retardation or agitation.
B. Cognitive:
  1. Sense of guilt, worthlessness, low self-esteem.
  2. Problems with attention span, concentration or memory, frustration tolerance, negative distortions, mild paranoia, and psychosis.
C. Impulse control:
  1. Suicidal or homicidal thoughts or acts. **Any statements made by the client, such as "Life isn't worth living, I wish I were dead, I don't deserve to be alive, I can't deal with this," should be taken seriously. Refer the client for emergency mental health assessment and treatment.**
D. Behavioral:
  1. Depressed mood, anxiety, and irritability.
  2. Isolation, decreased motivation, fatigability, and anhedonia (inability to derive gratification from pleasurable activities).
E. Physical symptoms:
  1. Digestion problems, nausea, constipation, diarrhea (less common), and dry mouth.
  2. Fatigue, but difficulty sleeping.
  3. Physical pain, chronic aches, and pains that cannot be explained.
  4. Recurrent headaches, backaches, or stomach aches that have no cause.
  5. Migrating pain that disappears when depression lifts.
  6. Increased muscle tension.

SUBJECTIVE DATA

A. Review the onset, duration, and course of presenting symptoms.
B. Review any history of depression (such as postpartum depression).
C. Determine how the previous depression was treated.
D. Evaluate the client's suicide potential. Ask: "Have you ever thought of hurting yourself or others?" Does the client have a current suicide plan or vague ideas of suicide? Has the client had any history of suicide attempts? If so, evaluate how life-threatening they were.
E. Review the client's medical history (see "Predisposing Factors").
F. Review the client's drug history for prescription, over-the-counter (OTC), and recreational/illicit drug use (how much, how long, how often), and review their history of alcohol consumption (how much, how long, how often).
G. Review the client's history for recent major life changes, such as pregnancy, death, divorce, or any loss that may be normal throughout the stages of life. The *client's perception* of the loss is what is important.
H. Review dietary intake since the symptoms have begun.
I. Establish usual weight, review weight gain/loss, and in what time span.
J. Review the client's activities of daily living (ADLs). Does the client get up and dress daily, perform daily hygiene, put on makeup?
K. Review how many hours of sleep and the quality of sleep per day.
L. Review the disruption of usual activities: return to work, return to school, exercise.
M. Review the amount of crying per day and for what length of time (days, weeks).
N. Assess whether the depression is cyclic/seasonal (starts in the fall, ends in the spring).
O. Review occupational/home exposure to lead and lead-based products. ▶

P. Review any exposures to infectious diseases, including Lyme disease (refer to Chapter 19, "Infectious Disease Guidelines," for specific questions). Does anyone else such as family, friends, or coworkers have similar symptoms?
Q. If female, review for symptoms of menopause (sleep disturbances, irregular menses/amenorrhea, hot flashes, vaginal dryness, dyspareunia).

PHYSICAL EXAMINATION

A. Check pulse, respirations, blood pressure, weight, and temperature.
B. Inspect:
  1. Observe overall appearance; note grooming, tone of voice, conduct of client during communication, and breath (smell of alcohol).
  2. Complete neurologic examination with screening tool of choice.
  3. Complete dermal examination for signs of substance use (refer to "Substance Use Disorders" section of this chapter) or areas of self-harm.
C. Palpate:
  1. Palpate the neck and thyroid; evaluate for presence of goiter.
  2. Palpate the axilla and groin for lymphadenopathy (infectious etiology).
  3. Check the joints for swelling, arthritis, and range of motion (ROM; rule out musculoskeletal cause).
  4. Abdomen to check for hepatosplenomegaly; could be indicative of an underlying disease.
D. Auscultate:
  1. Auscultate heart, lungs, and abdomen (as applies to physical complaints).

DIAGNOSTIC TESTS

A. Complete blood count (CBC) with differential.
B. Electrolytes, serum calcium, and phosphorus.
C. Thyroid profile.
D. Liver profile.
E. Lead level.
F. Follicle-stimulating hormone (FSH)/luteinizing hormone (LH).
G. Viral cultures.
H. Blood alcohol.
I. Urine drug screen and culture.
J. Monospot.
K. CT and MRI scans.
L. Dexamethasone suppression test.
M. Screening for vitamin/nutritional deficiencies.
N. Perform mental state examination with depression rating scale of choice:
  1. Beck Depression Inventory Scale, available at beck-institute.org/get-informed/tools-and-resources/professionals/patient-assessment-tools.
  2. Geriatric Depression Scale (GDS), available at www.healthcare.uiowa.edu/familymedicine/fpinfo//geridepr.htm.
  3. Patient Health Questionnaire (PHQ-9), available online at www.phqscreeners.com.

DIFFERENTIAL DIAGNOSES

A. Depression.
B. Bipolar disorder.
C. Chronic untreated anxiety disorders such as generalized anxiety disorder (GAD), posttraumatic stress disorder (PTSD), or obsessive-compulsive disorder (OCD).
D. Personality disorders.
E. Schizoaffective disorder.
F. Seasonal affective disorder (SAD):
  1. Seasonal pattern: starts in the fall, linked to lack of light exposure.
  2. Females more than males.
  3. Age typically in the 20s.
G. Alcoholism and drug abuse/dependence.
H. Early dementia.
I. Endocrine etiologies (see section "Predisposing Factors").
J. Infectious etiologies (see section "Predisposing Factors").
K. Menopause.
L. Side effect of medication (see section "Predisposing Factors").
M. Cancer: 50% of individuals with tumors, particularly of the brain and lung, and carcinoma of the pancreas develop symptoms of depression before the diagnosis of tumor is made.
N. Heavy metal poisoning.
O. Nutritional deficit (see section "Predisposing Factors").

PLAN

A. General interventions:
  1. Keep the client safe from self-harm.
  2. Treat physical/laboratory findings. Recommend dietary change, iron supplements, or hormone replacement therapy per findings (see related chapters).
B. Client teaching:
  1. Encourage the client to take medications as prescribed. Educate the client that some medications may take time to get into the system to work and time should be allowed to see the effects of the medication. Review side effects.
  2. Encourage the client to express feelings or worsening of symptoms if this occurs prior to the next appointment. Have the client make a client contract with you that they will not hurt themselves or others; if they begin having these thoughts, the client agrees to contact you or go to the nearest ED.
  3. Encourage exercise on a daily basis for 20 to 30 minutes to increase energy and enhance a feeling of well-being.
  4. Encourage the client to get at least 7 to 8 hours of sleep each night. If sleep is a problem, address this issue with the client.
  5. Avoid caffeine at night and/or watching television late at night.
  6. Encourage the client to seek counseling with a professional counselor. Refer to appropriate site (psychologist, psychiatrist, group therapy, etc.). Offer local resources to the client.
  7. Advise client to participate in activities to enhance interpersonal relationships and build self-esteem. Include family and friends in recommended therapies and advise them to encourage the client to participate in activities to enhance self-esteem.
  8. Once the client is feeling better, encourage continued use of medication, activities, and resources for at least 6 months after the client has started feeling better to prevent relapse.
C. Pharmaceutical therapy: Table 25.1 presents dosage information:
  1. Drugs of choice: selective serotonin reuptake inhibitor (SSRI) antidepressants. Caution should be used when coadministering SSRIs and drugs with a narrow therapeutic window such as carbamazepine (Tegretol), warfarin, ▶

tricyclic antidepressants (TCAs), antiarrhythmics, and some antipsychotic medications (risperidone, haloperidol, and phenothiazine), and other drugs including diazepam (Valium) and monoamine oxidase (MAO) inhibitors. There are numerous SSRIs on the market today. Some of the newer agents, such as Viibryd and Trintellix, may have fewer side effects:

a. Most antidepressant therapy takes 3 to 4 weeks for onset of action to elicit visible changes.
b. Never prescribe more than a week's supply or a total of 2 g of a TCA if there is a risk of suicide.
c. Medication should not be changed until it has been given a trial of 6 to 8 weeks to measure progress.
d. Before concluding that the antidepressant is ineffective, verify that the client is taking the medication correctly.
e. Although to be used with caution, first-line medication therapy in older adults are the SSRIs. The SSRIs have significantly fewer side effects than the traditional TCAs. Paroxetine is highly anticholinergic and sedating and may cause orthostatic hypotension.
f. Antidepressant therapy should be continued for 6 months to 1 year (up to 5 years if necessary) due to the risk of recurrence of depression.
g. Taper the medication off instead of abruptly withdrawing.

2. TCAs: Avoid administration after acute myocardial infarction. Males with prostatic hypertrophy do best with a nonsedating TCA that has a mild anticholinergic activity, such as desipramine (Norpramin) or nortriptyline (Pamelor).

3. Adrenergic modulators: a weak inhibitor of norepinephrine, dopamine, and serotonin reuptake: Bupropion hydrochloride (Wellbutrin):

a. For clients older than age 18.
b. No dosage established for older adults.
c. Therapeutic onset in 1 to 3 weeks.
d. Serum level peaks within 2 hours.
e. Initially 100 mg BID for at least 3 days.
f. If well-tolerated, increase to 375 or 400 mg every day.
g. After 3 days more, 450 mg every day in four divided doses at least 4 hours apart.
h. Maximum single dosing 150 mg, maximum 450 mg every day:
   i. Avoid bedtime dosing.
   ii. Do not give to clients with predisposition to seizures.
   iii. Give reduced dose to clients with renal or hepatic impairment.

4. Dual reuptake inhibitors: blocks the reabsorption of the norepinephrine and serotonin into the neurons in the CNS. When this occurs, higher levels of norepinephrine and serotonin remain and improve the nerve transmission in the brain, which then improves mood:

a. Venlafaxine hydrochloride (Effexor):
   i. For clients older than age 18.
   ii. No dosage established for older adults.
   iii. Therapeutic onset unknown.
   iv. Serum level peak and duration unknown.
   v. Initially 75 mg every day in two to three divided doses with food.

TABLE 25.1 DOSAGES FOR COMMON ANTIDEPRESSANT DRUGS

Drug	Adults Aged 18–60 Years	Older Adults >60 Years or Renal or Hepatic Clients
Fluoxetine hydrochloride (Prozac) Therapeutic onset occurs in 1–4 weeks. Serum levels peak in 4.5–8.5 hours.	20 mg qd in a.m., dosage increased according to client patient response May be given BID—in the morning and at noon Maximum dosage 80 mg/d	10–20 mg qd in a.m. with clinical response monitored q 1–2 weeks Gradual dose increases q 6 weeks until optimal therapeutic response obtained
Sertraline hydrochloride (Zoloft) Therapeutic onset occurs in 2–4 weeks. Serum levels peak in 4.5–8.5 hours.	50 mg qd in a.m. May increase to 100 mg Dosage adjustments to be made at intervals of no <1 week Maximum dosage 200 mg/d	25–50 mg qd in a.m. with clinical response monitored q 1–2 weeks and gradual increase
Paroxetine hydrochloride (Paxil) Therapeutic onset occurs in 1–4 weeks. Serum levels peak in 2–8 hours.	10 mg qd in a.m., p.m., or at HS May increase 10 mg/d increments at weekly intervals Maximum dosage 50 mg/d	10–20 mg qd in a.m., p.m., or at HS with clinical response monitored q 1–2 weeks and gradual increase
Nortriptyline hydrochloride (Pamelor) Therapeutic onset occurs in 2–4 weeks or longer. Serum levels peak in 7–8.5 hours.	25 mg TID or QID initially, gradually increase to maximum dose 150 mg qd	30–50 mg qd TID or QID or in 1 day's dose
Desipramine hydrochloride (Norpramin) Therapeutic onset occurs in 2–4 weeks or longer. Serum levels peak in 4–6 hours.	100–200 mg PO in single or divided dose	25–100 mg qd in divided doses, increase gradually to maximum of 150 mg qd
Amitriptyline (Elavil) Therapeutic onset unknown—thought to take several weeks. Serum levels peak 2–12 hours.	75 mg PO in divided doses initially or 50–100 mg at HS Dose not to exceed 150 mg/d	10–20 mg PO TID and 20 mg at HS qd

Note: Paxil works well for clients who have a component of anxiety to their depression. Monitor prothrombin time for clients taking warfarin, sertraline, and paroxetine.

HS, at bedtime; q, every; qd, every.

vi. May increase at 4-day intervals in 75 mg/d increments to 150 mg every day. Maximum dosage is 375 mg every day in three divided doses.
vii. With hepatic impairment, reduce dose by 50%.
viii. Withdraw gradually over 2 weeks when discontinuing medication.
ix. Interacts with MAO inhibitors. Do not start venlafaxine within 14 days of discontinuing an MAO inhibitor.

b. Duloxetine (Cymbalta) also blocks reuptake of serotonin and norepinephrine:
i. Usual starting dose is 30 mg/d increasing to 60 mg/d. Maximum dose is 120 mg/d.
ii. Clients should be warned about stopping the medication abruptly; have them taper down slowly.
iii. Duloxetine is also helpful in treating the physical pain that accompanies some depression symptoms.

5. Tetracyclic antidepressant: mirtazapine (Remeron). Potentiates noradrenergic and serotonergic activity:
a. Starting dose is 15 mg at bedtime, with maximum dose of 45 mg.
b. Remeron stimulates appetite and may be used in clients with symptoms of weight loss.
c. Remeron is administered at bedtime; side effect of somnolence may be beneficial for clients with insomnia.
d. Contraindicated within 14 days of taking MAO inhibitor.
e. Contraindicated with concomitant linezolid use.

6. MAO inhibitors should only be prescribed by a psychiatrist.

FOLLOW-UP

A. Follow up in 1 to 2 weeks to assess client's status, drug effectiveness, and adverse reactions.

B. Clients can become suicidal after the depression is treated and they begin to have more energy to act on the suicidal ideation. **Assess suicidality with every office visit.**

C. Follow up every 2 to 4 weeks afterward to check client's progress.

D. Once positive change is seen, the client can be seen monthly.

E. Refer to other applicable medical diagnosis for follow-up recommendations.

CONSULTATION/REFERRAL

A. If there is any potential for suicidal/homicidal behavior, refer client immediately for an emergency mental health assessment and treatment.

B. Refer for psychotherapy: Combined treatment with psychotherapy and antidepressant medication is more effective than treatment with medication alone.

C. Clients who fail to respond to antidepressants after 1 to 2 months of appropriate antidepressant therapy should have a psychiatric consultation. Psychiatry may prescribe adjunctive medications including psychotropics and mood stabilizers.

D. Refractory depression should be managed by a psychiatrist. Other treatment options may include electroconvulsive therapy (ECT), transcranial magnetic stimulation, eye movement desensitization and reprocessing (EMDR), and deep brain stimulation (DBS).

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. A female with a history of depression or previous postpartum depression is at high risk for postpartum depression (recurrent). Caution should be used in prescribing antidepressants during pregnancy. Review the benefits versus risks.
2. Refer to the section "Postpartum Depression" in Chapter 16, "Obstetrics Guidelines."

B. Partners:
1. If available in the community, provide resources for partners, such as the National Alliance for the Mentally Ill (listed in most telephone books).
2. Frequently, partners will take too much responsibility for the depressed client's state of mind and over time also become depressed. Relating to other people with depressed partners will assist them in dealing with their significant other's depression.

C. Geriatrics:
1. Dementia masked as "pseudodepression" is common in older adults. Check for memory impairment and disorientation because delirium can often be mistaken for depression (see section "Neurocognitive Disorders (Dementia)" in Chapter 23, "Neurologic Guidelines").
2. Older adult clients who are depressed may experience agitation rather than retardation in psychomotor function.
3. Persons aged 65 and older account for 25% of all suicides. White males, age 80, have the highest rate of suicide of any age group in the United States.
4. Assess for depression in caretakers of clients with Alzheimer disease.
5. Avoid use of Elavil and other highly anticholinergic medications in older adults due to the high risk of sedation and orthostatic hypotension.
6. Many older adults do not have the resources required to deal with depression. This may include material (e.g., lack of access to smart technology), social (e.g., few family members or friends), or cognitive or biologic (e.g., inability to engage in physical exercise or participate in activities or routines) resources. Clinicians and caregivers must estimate resource availability and consider how the absence of resources can be mitigated for a given individual and family. It is also important to understand the importance in the role of technology, which has emerged as an important factor for maintaining social connection as well as accessing mental health services.

Older adult clients who are suicidal may present with atypical symptoms of depression and may not express their distress directly. Three identified behaviors include impaired ability to communicate, intractable tinnitus, and feelings of helplessness.

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FAILURE TO THRIVE

DEFINITION

A. Failure to thrive (FTT) is an abnormality in which an individual fails to maintain nutritional health. FTT is a manifestation of an underlying problem, whether the problem is mental, physical, or psychological. ▶

1. Adults: FTT is seen in adults who have a weight less than 80% of the ideal average body weight for the adult.
2. Geriatrics: FTT in the geriatric population is defined as a deterioration in functional status disproportional to disease burden. Signs are decreased appetite, weight loss of greater than 5% of their weight, and decreased physical activity, along with dehydration, depression, and compromised immune status.
   a. Weight loss.
   b. Loss of muscle mass.
   c. Loss of subcutaneous fat.
   d. Localized or generalized fluid accumulation that may mask weight loss.
   e. Diminished functional status as measured by handgrip strength.

INCIDENCE

A. It is estimated to occur in 5% to 35% of older adult population, with nursing home residents having an occurrence rate of 25% to 40%.

PATHOGENESIS

A. Organic causes of FTT:
1. Gastrointestinal (reflux, celiac disease, Hirschsprung disease, and malabsorption).
2. Cardiopulmonary (cardiac diseases, congestive heart failure).
3. Pulmonary (asthma, bronchopulmonary dysplasia, cystic fibrosis).
4. Renal (diabetes insipidus, renal insufficiency, urinary tract infections).
5. Endocrine (hypothyroidism, adrenal diseases, parathyroid disorders, thyroid disorders, pituitary disorders).
6. Neurologic (mental retardation, cerebral hemorrhages).
7. Metabolic disorders (inborn errors of metabolism).
8. Congenital (congenital syndromes such as fetal alcohol syndrome, chromosomal abnormalities, perinatal infections).
9. Infectious (gastrointestinal infections, tuberculosis, HIV).

B. Inorganic (or psychosocial) causes pertain to family and social dynamics. It is common to see both organic and inorganic problems as causative factors for FTT.

C. Geriatric population:
1. Feel fuller with less food; this may be an endorphin response that decreases the adaptive relaxation of the fundus of the stomach.
2. Increased number of cytokines, which contributes to anorexia.
3. Diminished sense of smell or taste.
4. Dysphagia.
5. Medications.
6. Depression, delirium, dementia.
7. Alcohol or substance abuse.

PREDISPOSING FACTORS

A. Geriatrics: dementia, comorbidities (cancer, chronic infections, malabsorption syndromes, psychiatric disorders), limited mobility, despair.
B. Poverty.
C. Organic conditions with the major organs noted earlier.
D. Parents with psychosocial disorders.
E. Altered family processes.
F. Previous hospitalization.
G. No health insurance.
H. Living in a rural area.

COMMON COMPLAINTS

A. Weight loss that is unintentional.
B. Fatigue.
C. Impaired thought process.

Clients do not always present for this problem. Many clients are diagnosed at a routine examination in the ambulatory setting.

OTHER SIGNS AND SYMPTOMS

A. Loss of subcutaneous fat tissue.
B. Muscle atrophy.
C. Alopecia.
D. Dermatitis.
E. Marasmus.
F. Kwashiorkor.

SUBJECTIVE DATA

A. Obtain detailed history of the client's diet. Note the differences between foods offered and foods eaten. If dietary supplements are used, note type, frequency, and amount taken.
B. Assess quality of nutrients offered to the client. Consider knowledge deficit of care provider if inadequate:
1. Geriatrics: Is client able to chew and swallow food offered? Are dentures well-fitting? Are supplements being offered?

C. Query regarding financial resources, and if needed does the family participate in low-income opportunities (food stamp programs, meals on wheels, etc.)?
D. Evaluate whether religious or unusual dietary beliefs/habits contribute to the food preparation, if inadequate.
E. Rule out any difficulty in swallowing or retaining ingested food.
F. Note regular bowel/bladder habits.
G. Note any recent illness, chronic or acute.
H. Rule out family history of cystic fibrosis or lactose intolerance.
I. Rule out neglect/mistreatment. May need to interview the client privately.
J. Geriatrics: Evaluate nutritional screening using the Mini Nutritional Assessment. This tool is available at www.mna-elderly.com/forms/mini/mna_mini_english.pdf.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure, height, and weight.
B. Inspect:
1. Observe overall appearance.
2. Observe oral pathology: Check for ill-fitting dentures, and dental and gum condition.
3. Note muscle tone, strength, and movement.
4. Note social interactions among family members.
5. Note social skills of the client.

C. Palpate:
1. Palpate abdomen, back, and extremities.

D. Percuss:
1. Percuss the abdomen.

E. Auscultate:
1. Auscultate the heart and lungs.

DIAGNOSTIC TESTS

A. Complete blood count (CBC), urinalysis, electrolytes.
B. Thyroid panel, if indicated.
C. X-rays, if appropriate.
D. Serum albumin in geriatrics.

DIFFERENTIAL DIAGNOSES

A. FTT: inorganic versus organic etiology.
B. Weight loss.
C. Depression.
D. Deconditioning.

PLAN

A. General interventions:
1. The plan is based on the cause of FTT.
2. Severe malnutrition requires hospitalization.
3. Obtain nutritional consult to evaluate the dietary needs for protein, iron, and other nutrients according to age and size.
4. Social work consult if suspected neglect.
B. Client teaching:
1. Reinforce positive eating habits and encourage dietary meal planning.
2. Offer nutrition counseling with dietitian.
3. Educate the client and family about the importance of meeting the dietary requirements for protein, iron, calcium, and other nutrients to prevent weight loss and loss of muscle and bone mass, and to prevent infection and other complications that can stress the body.
C. Dietary management:
1. Meal suggestions: Offer adequate time for meals (20–30 minutes), offer solid food before drinks/juices, and provide a pleasant environment for eating.
2. Socialization enhances mealtime. Encourage all family members to sit down and eat at least one meal a day together.
3. Provide handout for high-calorie foods (peanut butter, cheese, whole milk, etc.).
4. Consider exercise sessions for geriatrics to stimulate appetite.
5. Encourage clients to attend centers where meals are served as a group or have meals delivered to the home.
6. Encourage small, frequent meals with snacks between meals and before bedtime.

Weight gain with high-calorie supplements is commonly seen with clients who have a psychosocial etiology of FTT.

D. Pharmaceutical therapy:
1. High-calorie supplements are recommended for some clients (Polycose, Carnation Instant Breakfast, Ensure, Boost, Thrive).
2. Geriatrics: Short-term aggressive caloric replacement has been shown to be effective in reversing FTT. Severe malnutrition may require hospitalization with total parenteral nutrition. Medications used to increase appetite:
a. Megestrol 400 or 800 mg every day with meal.
b. Selective serotonin reuptake inhibitors (SSRIs) have been shown to be beneficial to help stimulate appetite and increase weight.
c. Mirtazapine (Remeron) 15 mg at bedtime.

FOLLOW-UP

A. Two-week evaluation for weight measurements and to evaluate compliance with regimen at home.
B. Routine visits recommended every 2 to 4 weeks to monitor progress.
C. Reevaluate client in 1 to 2 months. After 2 months, if no improvement or further loss is noted, refer to a specialist.

CONSULTATION/REFERRAL

A. A nutritional consult is helpful to assist the client or food provider in providing adequate resources/calories for the client.
B. Consider social services and visiting nurses for outpatient assistance in the home.
C. Occupational therapy may be useful to evaluate the client's capacity to plan and prepare meals.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. FTT in older adults may lead to a decline in physical and mental function. Aggressive treatment should be employed to improve the nutritional status of these clients.
2. FTT increases the risk of morbidity and mortality.
3. FTT increases the risk of depression and social isolation in older adults.

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GRIEF

DEFINITION

A. Bereavement, grief (acute and integrated), mourning, prolonged grief disorder, anticipatory grief, disenfranchised grief, and Takotsubo cardiomyopathy (broken heart syndrome) all describe different aspects related to the death or loss of a loved one.
1. Bereavement is the situation in which someone who is close dies (rather than the reaction to that loss). Bereavement does not always lead to grief.
2. Grief is defined as the normal, appropriate emotional response caused by a loss. This feeling of loss is a response that has been caused by a particular event in one's life. It is unique to the individual experiencing it, and there is no general timetable for completing it. Grief is commonly seen following the death of a loved one, but grief also follows other losses (e.g., loss of independence, loss of affection, or loss of body parts, pain, and distress).
3. Mourning is defined as the process by which grief is resolved. Mourning is individual and helps in reaching acceptance of a loss.
4. Prolonged grief disorder is a form of grief that is unusually protracted, intense, and disabling. The disorder is characterized by maladaptive thoughts, dysfunctional behaviors, dysregulated emotions, and/or serious psychosocial problems that impede adaptation to the loss.
5. Anticipatory grief is a response to an expected loss. It affects both the person diagnosed with a terminal illness as well as their families.
6. Disenfranchised grief is defined by grief that someone experiences when they incur a loss that is not or cannot be openly acknowledged, publicly mourned, or socially supported. Physicians regularly encounter losses in the form of client deaths.
7. Takotsubo cardiomyopathy (broken heart syndrome) is a weakening of the left ventricle caused by severe ▶

emotional or physical stress such as loss of a loved one, sudden illness, a serious accident, or a natural disaster (e.g., earthquake). It almost exclusively occurs in females and resolves within a month.

B. The process through which one resolves grief usually follows a typical course that can be viewed in five stages:

1. Denial: Denial occurs when one refuses to accept the circumstance that has occurred. It is a natural defense mechanism that occurs to protect the body.

2. Anger: Pain, tears, anxiousness, anger, and feelings of guilt may be seen.

3. Bargaining: In this stage, the person tries to negotiate alternatives that will make their feel better.

4. Depression: One begins to understand what has happened and may show feelings of sadness and fear.

5. Acceptance: One begins to rebuild life and think about the past with pleasure. In this phase, one regains interest in activities and forms new relationships.

C. It is important to distinguish between the normal grief reaction to pathologic grief and major depression. Often, depressive symptoms are a pervasive part of the grief response, and a clear delineation of grief versus depression is not always possible. (Based on the Grief Cycle model first published in *On Death and Dying*, Elisabeth Kübler-Ross, 1969, Interpretation by Alan Chapman 2006–2009. www.ekrfoundation.org/5-stages-of-grief.)

INCIDENCE

A. Grief is a universal emotional response. Approximately 5% to 9% of the population will lose a close family member or friend each year. Grief is a normal emotional response that will follow this loss for an individual.

B. Older adults experience grief at a higher rate than younger adults or children. Spousal loss is common in older adults, as well as the death of friends, siblings, and cousins.

PATHOGENESIS

A. Grief is a normal emotional response to the loss of a loved one, pain, and/or distress. Abnormal, pathologic grief can occur if the mourner is not encouraged to grieve losses. Normal grief resolution begins to subside at approximately 6 months but may sometimes take longer.

PREDISPOSING FACTORS

A. Sudden or terrible deaths.

B. Excessive dependency on the deceased and feelings of ambivalence.

C. Traumatic losses earlier in life.

D. Social isolation.

E. Actual or imagined responsibility for "causing" the death.

F. Avoidance of grief and denial of loss.

G. Survived a traumatic experience that killed the deceased.

H. Large-scale emergency event such as earthquake, flood, or pandemic (COVID-19).

COMMON COMPLAINTS

A. Angry feelings at God or medical personnel for not doing more, anger at oneself for not seeing the warning signs, anger at the deceased for not taking better care of themselves. Common feelings of being left alone and not making proper financial/legal preparations may also occur.

B. Sleeping all the time or inability to sleep without medication.

C. Change in eating habits with significant weight loss or gain.

D. Fatigue, lethargy, or lack of motivation.

E. Decreased concentration and memory, forgetfulness.

F. Increased irritability.

G. Unpredictable bouts of crying.

H. Fears:

1. Of being alone or with people.

2. Of leaving the house.

3. Of staying in the house.

OTHER SIGNS AND SYMPTOMS

A. Normal grief:

1. Protest, disbelief, shock, and denial.

2. Profound sadness and survivor guilt.

3. Multiple somatic symptoms without actual organic disease.

4. Sense of unreality and withdrawal from others.

5. Disruption of normal patterns of conduct, with restlessness and aimlessness.

6. Preoccupation with memories of the deceased, dreams of the deceased, hallucinations, fear of going crazy, and transient psychotic symptoms.

7. Response to support and ventilation improves over time.

B. Complicated or prolonged grief:

1. Persistence of denial with delayed or absent grief.

2. Depression with impaired self-esteem, suicidal thoughts, and impulses with self-destructive behavior.

3. Actual organic disease and medical illness.

4. Progressive social isolation.

5. Persistent anger and hostility, leading to paranoid reactions, especially against those involved in medical care of the deceased, or suppression of any expression of anger and hostility.

6. Continued disruption of normal patterns of conduct, often with a persistent hyperactivity unaccompanied by a sense of loss or grieving.

7. Continued preoccupation with memories of the deceased to the point of searching for reunion (sustained depressive delusions).

8. Conversion symptoms similar to the symptoms of the deceased.

9. Self-blame.

10. Prolonged grief longer than 6 months is commonly linked to complications and impairment for the next 1 to 2 years.

SUBJECTIVE DATA

A. Review onset, duration, and course of presenting symptoms. Review the mourner's grief symptoms.

B. Obtain an in-depth personal history of the mourner and their relationship to the identified loss or with the deceased.

Understanding the bereaved's history is critical to understanding the individual's loss.

C. Identify anniversary dates pertinent to the mourner's relationship with the deceased/loss.

D. Determine whether the mourner has suicidal ideation (especially with a plan). **Be sure to ask: "Have you ever thought of hurting yourself or others?"**

E. Assess whether the mourner experiences self-blame.

F. Review the mourner's appetite.

G. Establish usual weight, review weight gain/loss, and in what time span.

H. Review the mourner's activities of daily living (ADLs). Does the mourner get up and dress daily and perform daily hygiene?
I. Review the mourner's sleep quality.
J. Review the mourner's daily routines: return to work, return to school, and exercise.
K. Review the mourner's amount of crying per day, and note for what length of time (days, weeks).
L. Review the mourner's drug and alcohol consumption since the loss.

Statements suggesting self-medication with alcohol to facilitate sleep could indicate a coexistent alcohol abuse dependence diagnosis.

M. Review the mourner's usual medical problems and how the loss/grief has affected these problems.
N. Review social support system: friends or family.

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure, weight.
B. Inspect:
1. Observe overall appearance. Note grooming habits, dress, appearance.
2. Note social interactions among family members.
3. Note social skills of the client.
C. Auscultate:
1. Auscultate the heart and lungs.

DIAGNOSTIC TESTS

A. As indicated to rule out other pathology.
B. Blood glucose.
C. Thyroid studies.
D. If depression is suspected, perform depression screening such as Beck's Depression Inventory Scale questionnaire (see "Depression" section of this chapter).

DIFFERENTIAL DIAGNOSES

A. Grief.
B. Depressive disorder.
C. Posttraumatic stress disorder (PTSD).
D. Somatoform disorders (characterized by physical complaints lacking known medical basis or demonstrable physical findings in the presence of psychological factors).
E. Alcoholism and drug abuse/dependence.

PLAN

A. General interventions:
1. Evaluate the nature of the grief and any accompanying psychiatric symptoms.
2. Treat physical/laboratory findings as indicated.
3. Encourage the client to eat a healthy diet, exercise daily, and maintain normal sleep habits and activities.
4. Encourage family and friend support.
5. Offer counseling with professional psychologist or group sessions.
6. Assess for depression at each office visit and treat accordingly.
B. Client teaching**:** *See Client Teaching Guide for this chapter, "Grief."*
C. Pharmaceutical therapy: Antidepressants should not be prescribed for acute grief but reserved for a possible subsequent major depression. Clinical data suggest that selective serotonin reuptake inhibitors (SSRIs) may assist the client with mobilizing the energy necessary to assist them through the grieving process.

Resist sedation of individuals suffering from acute grief because this tends to delay and prolong the mourning process. Refer to the "Depression" section for pharmaceutical therapy.

1. Drug of choice: sedative to help sleep:
a. Sedative anxiolytic hypnotics may be prescribed for **no more than 2 weeks at a time.**
b. Try initially for 1 week to establish a sleep pattern. If insomnia continues, refer the client to a specialist.
c. Temazepam (Restoril) 7.5 to 30 mg at bedtime *or* flurazepam (Dalmane) 15 to 30 mg at bedtime.
d. Zolpidem (Ambien) 5 to 10 mg at bedtime (not to be used for more than 1 month).
2. Sedating antihistamines:
a. Hydroxyzine HCl (Atarax) 50 to 100 mg at bedtime.
b. Hydroxyzine pamoate (Vistaril) 50 to 100 mg at bedtime **(not to be used for more than 4 months).**
3. Antidepressant with sedating properties:
a. Trazodone HCl (Desyrel) 50 to 100 mg at bedtime.
b. Paroxetine (Paxil) 10 to 20 mg at bedtime.
c. Mirtazapine (Remeron) 15 mg every day at bedtime. Increase at 1 to 2 weeks: Usual range is 15 to 45 mg every day at bedtime.

FOLLOW-UP

A. Follow up in 1 week to assess the client's status and symptoms.
B. Then follow up every 2 weeks to assess the client's progress.
C. Assess for depression and suicide at every office visit.
D. Once positive change is seen, the client can be seen monthly.

CONSULTATION/REFERRAL

A. Provide immediate referral/consult for continuing psychotherapy for severe depression and/or suicidal threats.
B. Consult with a physician for evaluation of pharmacologic agents versus referral.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:
1. Miscarriage, stillbirth, and neonatal death should be considered a major loss and treated as a grief reaction.
2. Grief is also seen in pregnancy termination. The client who terminates a pregnancy (regardless of gestational age and reason for termination) may exhibit a major response to this loss.
3. Hospitals often provide photographs, footprints, and identification bracelets and connect families with perinatal grief support groups.
4. Use the baby's name when discussing feelings about the loss of a child.
5. Suggest that friends and family not put away the clothes and bedroom furniture. The baby couple should do this as part of closure.
B. Adults:
1. Grief responses vary from among individuals. Look for behaviors outside the norm.
C. Geriatrics:
1. Grief in older adults should be closely assessed to rule out medical diagnoses.

2. Grief can be a very complicated and prolonged process for individuals who have dementia.

3. Avoid use of sedating antihistamines and hypnotics in older adults due to potential for delirium, falls, fractures, and car accidents, as well as minimal improvement in sleep latency and duration.

D. Partners:

1. Involvement in grief/loss psychotherapy groups is extremely helpful.

E. End-of-life care:

1. It is important to prepare spouses and family members of terminally ill clients.

a. Preparedness for death and coping with bereavement are an important part of the grieving process.

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INTIMATE PARTNER VIOLENCE

DEFINITION

A. Intimate partner violence (IPV) is defined as intentional control or victimization of a person with whom the abuser has or is currently in an intimate, romantic, or spousal relationship. Domestic IPV violence crosses all cultures and economic boundaries; it encompasses violence between both genders, including gay and lesbian relationships. Abusive behaviors can occur in a single event or sporadically, or can be continual. The Duluth Model Power and Control Wheel from the Domestic Abuse Intervention Project illustrates the abuser's power and control over the victim (Figure 25.1).

B. Physical abuse, sexual assault, coercion, social isolation, emotional abuse, economic control, and deprivation are associated with IPV. There is no typical abuser, although all abusers tend to be violent in the home setting and their behavior at work is normal.

C. Forms of physical violence include threatening or assaulting with weapons, pushing, shoving, slapping, punching, choking, kicking, holding, throwing objects, and binding.

D. Psychological abuse includes threats of physical harm to the victim or others, humiliations, intimidation, degradation, ridicule, false accusations, isolation, and deprivation of food, money, access to healthcare, and transportation.

E. Psychological abuse in LGBT relationships includes the threat to "out" their partner as well as threats related to custody of coparented children.

F. Stalking as a form of IPV. Cyberstalking is psychological abuse via the internet or texting. Intimate partner stalking occurs during a relationship or after the relationship ends:

1. Monitoring cell phone and internet activity.

2. Posting photographs or other types of humiliation on social media.

3. Putting tracker on the victim's car or hidden in the house.

G. Sexual abuse is nonconsensual (unwanted kissing or touching) or painful sexual acts.

H. Reproductive coercion is another form of IPV.

1. Partner sabotage of safe sex practices (e.g., refusal to use condoms, thus exposing the client to sexually transmitted infections).

2. Refusal/control of contraception.

3. Forcing the female to have an abortion, or utilizing physical violence to endanger a pregnancy.

4. Controlling access to healthcare.

INCIDENCE

The exact incidence of IPV is unknown due to the lack of reporting. The United Nations estimates that more than 600 million females live in countries where domestic violence is not considered a crime. The most significant reason for missing the diagnosis of IPV is failure to ask the client.

A. Girls and young women between the ages of 16 and 24 experience the highest rate of IPV.

B. Domestic violence is the leading cause of homicide death in females globally.

C. One in three females and one in four males have experienced physical violence by an intimate partner.

D. Up to 75% of domestic assaults occur after separation; females are most likely to be murdered when reporting abuse or attempting to leave an abusive relationship. Presence of a firearm in a domestic violence situation increases the risk of homicide by 500%.

E. An estimated 81% of females stalked by an intimate partner also suffer physical assault. Stalking by an intimate partner is estimated to occur among 1 million females and 317,000 males per year.

F. An estimated 4% to 15% of females presenting to EDs have situations related to domestic violence.

G. Females who separate have a risk of violence approximately three times that of divorced females:

1. Over half of the children who witness domestic violence intervene in some way, including yelling to the abuser to stop, calling for help, and trying to get away.

H. The incidence of abused males is estimated as one in three. Males are also victims of attempted or complete rape, at approximately 3% during their lifetime.

I. Pregnancy has an increased incidence of violence:

1. One in five young females and 35% of women have experienced pregnancy coercion.

2. 53% of young females have experienced birth control sabotage.

3. It is estimated that 5% to 20% of intimate partner abuse occurs against pregnant females.

J. Sexual violence, rape, physical assault, or stalking by an intimate partner occurs for 43.8% of lesbians, 61.1% of bisexual females, and 35% of heterosexual females, and for 26% of male with male partners, 37.3% of bisexual males, and 29% of heterosexual males.

K. Among college females, 20% to 30% report violence during a date:

1. Approximately 70% of college students say they have been sexually coerced.

L. Females in the military are recognized as a vulnerable population susceptible to abuse due to their geographic location away from family and friends and the social isolation within the military culture:

1. In 2017 the Department of Defense (DoD) estimated that 6,769 reports of military assaults occurred.

2. One in three convicted military sex offenders remain in the military.

PATHOGENESIS

A. IPV is not associated with an underlying medical condition. The cycle of abuse has three phases: ▶



FIGURE 25.1 Abuser's power and control wheel.

Source: From Domestic Abuse Intervention Programs. (n.d.). https://www.theduluthmodel.org/wp-content/uploads/2017/03/PowerandControl.pdf.

1. Tension building: The victim tries to avoid violence and is described as "walking on egg shells," unsure what will trigger an abusive incident.
2. Explosion and acute battering.
3. "Honeymoon phase," noted for the absence of tension and reconciliation.

B. Victims stay with their partners for multiple reasons, including fear, shame, denial, religious reasons, lack of resources, custody and other legal issues, fear of being "outed," and family pressures.

PREDISPOSING FACTORS

A. Sex/gender: Females and female-identifying individuals are predominantly the victims.
B. Race: African Americans, American Indians, Hispanic women, and Alaskan Natives.
C. Higher incidence in interracial couples.
D. Pregnancy.
E. History of violence:
1. Family domestic violence.
2. Abuse as a child: 50% report abuse as an adult.
F. History of drug use.
G. Posttraumatic stress disorder (PTSD).
H. Lack of social support systems.
I. Impulse control disorders.
J. Poor economic status.
K. LGBT.

COMMON COMPLAINTS

A. Vague complaints.
B. Sexual problems.
C. Depression.
D. Chronic pain inconsistent with organic disease.
E. Chronic headaches/migraines.

F. Stress:
  1. Anxiety.
  2. Panic attacks.
G. Alcohol or drug abuse (the batterer, victim, or both).
H. Current or past self-mutilation.
I. Gynecologic and obstetric complaints:
  1. Dyspareunia.
  2. Frequent vaginal or urinary tract infections.
  3. Pelvic pain/infection.
  4. Recurrent sexually transmitted infections.
  5. Unintended pregnancy.
  6. Late prenatal care.
  7. Miscarriage.
  8. Preterm bleeding/delivery.
J. Complaints of falls and other recurrent accidents.
K. Eating disorders.
L. Gastrointestinal complaints/irritable bowel syndrome.
M. Musculoskeletal complaints.

OTHER SIGNS AND SYMPTOMS

A. Multiple prior visits to the ED for traumatic and nontraumatic complaints.
B. A delay between injury and office visits (may result from lack of transportation or inability to leave the house).
C. Noncompliance with the treatment or missed appointments (lack of access to money or telephones)
D. Suicide attempt (25% higher in females with IPV).
E. The partner accompanies the client at all visits.

SUBJECTIVE DATA

A. The "gold standard" research method to document the prevalence of female exposure to violence includes conducting the interview one on one, in private, and asking specific direct questions.
B. The batterer often refuses to leave the client alone and may answer questions for the client. Translators should not be members of the client's or suspected abuser's family.
C. Use direct questions: female-validated Partner Violence Screen (PVS):
  1. Have you been hit, punched, kicked, or otherwise hurt by someone in the past year? If yes, by whom and were you injured?
  2. Do you feel safe in your current relationship?
  3. Is a partner from a previous relationship making you feel unsafe now?
  4. Are you here today due to injuries from a partner?
  5. Are you here today because of illness or stress related to threats, violent behavior, or fears due to a partner?
D. Assess if the client has ever told family or friends, called hotlines, or attempted to leave the abuser.
E. Has the client sought help with law enforcement or legal help, that is, filed a criminal complaint or got an order of protection?
F. Are there any weapons in the home?
  1. Has the abuser ever threatened or tried to kill you?
  2. Are you thinking of suicide? Have you ever considered or attempted to commit suicide because of problems in your relationship?
  3. Have you ever considered or attempted killing your batterer?
  4. Do you have a plan?

PHYSICAL EXAMINATION

A. Enforce the need to interview and do physical examinations in private. Do a full body examination, including the head/scalp:
  1. Most injuries are to the central (breast, chest, and abdomen) area, which is easily concealed by clothing.
  2. Other frequent sites of injury include the head, face, throat, and genitals.
  3. Explain the physical examination and touch with permission.
  4. Forensic exams need thorough documentation of injuries:
    a. Use color photographs before any treatment is started.
    b. Photograph damaged clothing.
    c. Take at least one full body photograph and a facial photograph.
    d. Take closeup photographs of all injuries.
    e. Use a ruler to identify/document the size of injuries.
    f. Documentation on the back of the photographs should include the client's name, date, photographer's name, as well as any witnesses to the examination. The photographer should also sign each photograph.
    g. Use direct quotes of the client's history of the violence.
B. Check blood pressure, pulse, and respirations.
C. General observation: Observe for depression/withdrawal, flat affect, anxiousness, fear, evasiveness, poor eye contact, and wearing of heavy makeup or clothing to conceal signs of abuse. Evaluate voice changes: dysphonia and aphonia. Observe for difficulty breathing.
D. Inspect:
  1. Dermal exam for presence of cigarette burns, impression marks, rope burns, welts, abrasions, scratch marks, claw marks, bite marks, ligature marks, petechiae, and contusions at multiple sites (e.g., back, legs, buttocks). Inspect skin for healing injuries that do not fit recent injury scenario. The client may not always be able to be evaluated at the time of injury.
  2. Eye exam:
    a. Observe subconjunctival hemorrhages from strangulation/struggle.
    b. Perform a funduscopic examination (if indicated secondary to trauma).
  3. Evaluate the genitals for lacerations and hematomas of the vagina or labia.
E. Auscultate:
  1. Auscultate all lung fields.
  2. Auscultate heart.
  3. Auscultate the bowel sounds in all four quadrants of the abdomen.
F. Palpate:
  1. Evaluate skull/facial trauma to the maxillofacial area, eye orbits, mandible, and nasal bones. Facial injuries are reported in 94% of victims.
  2. Evaluate for dislocations, fractures (including spiral fractures), sprains, and contusions to the wrists, forearms, and shoulders.
  3. Palpate abdomen, liver, and spleen for tenderness.
G. Percuss:
  1. Percuss abdomen, chest, and areas of injury (if indicated secondary to trauma).
H. Neurologic examination (if indicated secondary to trauma).
I. Genital/rectal examination:
  1. Evaluate genitals/anal area for redness, swelling, bruising, hematomas, abrasions, or lacerations.
  2. Perform bimanual examination (females). ▶

3. Perform an anoscopy (if indicated).
4. Evaluate for evidence of sperm (recto/vaginal).
5. Evaluate for presence of condyloma (perineum, rectum, vagina).
6. Evaluate for presence of foreign bodies (recto/ vaginal).

DIAGNOSTIC TESTS

A. Diagnostic tests and x-rays are ordered dependent on the type of presenting complaints and physical examination.
B. Administer a domestic abuse assessment screening tool and have the victim mark a body map of injuries (Figure 25.2).
C. Complete blood count (CBC) with differential and peripheral smear, bleeding evaluation, including prothrombin time/partial prothrombin time (PT/PTT), alanine aminotransferase (ALT), and aspartate aminotransferase (AST) to evaluate injury to the liver, serum amylase, or lipase to rule out pancreatic injury.
D. Urinalysis.
E. Drug/toxicology screen (urine and blood).
F. Obtain forensic DNA samples from the skin, under nails, and within the vagina and rectum, as well as saliva from bite marks using sterile cotton-tipped applicators that have been moistened with sterile saline. These should be sent to a crime laboratory as soon as possible.
G. Test for sexually transmitted infections/HIV.
H. Pregnancy test (if indicated).
I. Radiographs: facial injury, anteroposterior (AP) and lateral radiograph for any areas of bone tenderness, swelling, deformity, or limited range of motion (ROM).
J. Ultrasounds as indicated.
K. Neuroimaging CT/MRI for any suspected nonaccidental head injury (e.g., head trauma or scalp hematoma).

DIFFERENTIAL DIAGNOSES

A. Domestic violence:
  1. Intimate partner abuse.
  2. Elder abuse.
  3. Human trafficking.
B. Rape.
C. Other: related to presenting symptoms.

PLAN

A. General interventions:
  1. Provide a safe environment. Assess for immediate danger.
  2. Clearly document the history, physical findings, and interventions. Document specific details about the abuse.
  3. Determine the risk to the victim and any children.
  4. Evaluate the need for ED/hospital admission.
  5. Battery is a crime; assess the victim's readiness for police intervention and need for a court order of protection.
  6. Help develop a safety plan.
  7. Assess readiness to leave: collection of important papers (e.g., birth certificates, custody papers, divorce papers, legal agreements, address book, and copies of restraining orders), access to money/credit cards, and telling family and friends.
  8. Familiarize yourself with the local resources such as hospital programs, shelters, social welfare services, and support groups.
  9. Provide contact numbers for shelters. Have the client hide information in their shoes.
  10. Counsel that violence may escalate.
B. Client teaching:
  1. Reinforce the fact that the violence is not the victim's fault. IPV is very common and the victims do not deserve to be abused. Discuss the cycle of abuse.
  2. Violence increases in frequency and severity.
  3. Help is available.
  4. The DoD has a self-help phone app created by the Rape, Abuse & Incest National Network (RAINN) for sexual assault survivors to create a customized self-care plan. This app is available through the iTunes store. The app is a resource for Active Duty, National Guard, and Reserve service members:
    a. **RAINN has adapted the National Sexual Online Hotline to provide specialized live help online at SafeHelpline.org to members of the DoD community who have been sexually assaulted.**
    b. **The Safe Helpline is also available by calling 877-995-5247.**

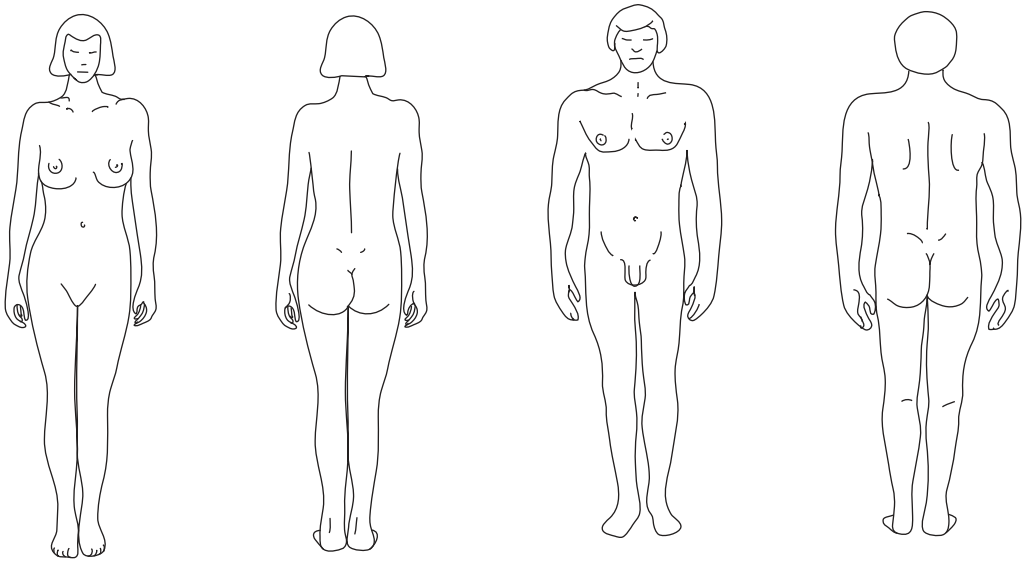


FIGURE 25.2 Abuse assessment screening tool with body map.

Source: Reprinted with permission from Parker, B., McFarlane, J., Soeken, K., Torres, S., & Campbell, D. (1993). Physical and emotional abuse in pregnancy: A comparison of adult and teenage women. *Nursing Research*, 42(3), 173–78. https://doi.org/10.1097/00006199-199305000-00009.

c. **All Safe Helpline staff members have been trained to answer questions related to military-specific topics such as restricted and unrestricted reporting and how to contact relevant military resources such as the installation or base's Sexual Assault Response Coordinator (SARC) if their services are requested.**

C. Pharmaceutical therapy:

1. Prescriptions are related to physical injuries.

2. Treatment for sexually transmitted infections in the oral, anal, and genital areas.

3. Tranquilizers may impair the victim's ability to flee or defend themselves and should not be prescribed.

FOLLOW-UP

A. Develop a follow-up plan:

1. What type of help does the client want?

2. Does the client have a plan for returning? Is the batterer home? Does the client think it is safe?

3. Does the client have a place to stay with family or friends; or do they want to go to a shelter?

4. Give the telephone numbers for shelters and crisis hotlines.

B. Screen the client for abuse at all subsequent visits.

C. Mandatory reporting:

1. States require reporting when domestic violence involves a child under the age of 18 and abuse or neglect of the child is suspected.

2. Abuse of a disabled person must be reported to the Disabled Persons Protection Commission.

3. Reporting elder abuse may be mandatory in your state.

4. If weapon was used in abuse such as gun, club, or sword.

D. Your state may mandate reporting and intervention with law enforcement. Refer to the Domestic Violence, Sexual Assault, and Stalking Data Resource Center: www.jrsa.org/projects/domviol.html.

E. The 2013 National Protocol for Sexual Assault Medical Forensic Examination for Adults and Adolescents is available at ncjrs.gov/pdffiles1/ovw/241903.pdf.

F. Protection order. Clients may request a Domestic Violence Protection Order (DVPO), preventing perpetrators from contacting them. Refer to a community advocate or legal advisor for assistance.

CONSULTATION/REFERRAL

A. Facilitate referrals to a shelter, counseling, and legal services.

B. Contact a sexual assault nurse examiner (SANE) qualified healthcare provider if indicated.

C. Refer to community or private support groups and agencies.

D. Refer for a consultation with a psychiatrist if the victim is homicidal or suicidal.

E. Refer for a neurologic or neurosurgical consultation for intracranial injuries or focal neurologic findings.

F. Refer for an orthopedic consultation for fractures.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Pregnancy is a known period of increased risk of violence.

2. The genitals, breast, and abdomen are common sites targeted for trauma.

3. Females may present with a miscarriage or premature labor.

4. Blunt trauma is a common injury in pregnancy.

5. Perform universal screening at each trimester and postpartum since abuse often begins during pregnancy.

6. Question during OB visits, in a nonjudgmental way, all bruises and abrasions.

RESOURCES

Dating Abuse Stops Here: www.datingabusestopshere.com

Domestic Violence, Sexual Assault and Stalking Data Resource Center: www.jrsa.org/projects/domviol.html

Futures Without Violence *(formerly Family Violence Prevention Fund):* www.futureswithoutviolence.org

National Domestic Violence Hotline: 1-800-799-7233

National TEEN Dating Abuse Helpline: 1-866-311-9474

Rape Abuse & Incest National Network (RAINN) Hotline: 1-800-656-4693

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SLEEP DISORDERS

DEFINITION

A. In normal sleepers, transient insomnia occurs in those who have traveled to another time zone (i.e., "jet lag"), are under situational stress, or are sleeping in unfamiliar surroundings. Treatment is not required in these situations because time takes care of the problem. With short-term insomnia, the normal sleeper experiences difficulty sleeping that does not resolve within a few days. This can be the result of stress, such as financial difficulty and divorce. These clients may require short-term symptomatic relief of insomnia.

B. Long-term insomnia is persistent and disabling. Studies suggest that almost all have an associated psychiatric disorder, especially depression; an associated drug use/abuse/withdrawal problem; or an associated medical disorder.

INCIDENCE

A. Difficulties with sleep are among the most common complaints of medical clients and affect a large percentage of the population. From 10% to 20% of all adults express sleep-related complaints they consider to be serious. Sleep disorders also can affect proper mental functioning (53% of chronic insomniacs complain of memory difficulties). Sleep disorders are also implicated in decreased work efficiency, impaired industrial productivity, and increased risk of traffic accidents. They also seem to enhance the propensity for cardiovascular disease and increase the risk of death. Insomniacs are also at increased risk for the development of depression and anxiety disorders. Clients with obstructive sleep apnea (OSA) syndrome have significant performance impairments on complex motor tasks.

The risk of depression increases with time if insomnia is left untreated.

B. COVID-19: 56.2% of people believed that their sleep cycle had changed due to lockdown.

PATHOGENESIS

A. Other than situational stress, jet lag, and sleeping in unfamiliar surroundings, difficulty with sleep can be related to psychiatric illness or medical problems. It is most frequently due to chronic depression and/or anxiety. Antisocial and obsessive-compulsive features are also common among these clients.

1. Clients may self-medicate, which produces more insomnia.

PREDISPOSING FACTORS

A. Alcohol use: initially assists with sleep but produces fragmented sleep.

B. Hypnotic medications can produce tolerance, which causes sleep disruption and rebound insomnia with withdrawal from the medication.

C. Substances such as caffeine, nicotine cigarettes, amphetamines, steroids, methylphenidate, hallucinogens, aminophylline, ephedrine, decongestants, bronchodilators, weight loss/diet pills, thyroid preparations, monoamine oxidase (MAO) inhibitors, and anticancer agents.

D. Persons with fibromyalgia syndrome.

E. Individuals experiencing menopausal symptoms.

F. Upper respiratory symptoms.

G. OSA disorders such as nasal obstruction, large uvula, low-lying soft palate, craniofacial abnormalities, excessive pharyngeal tissue, pharyngeal masses (tumors, cysts), macroglossia, tonsillar hypertrophy, and vocal cord paralysis.

H. Obesity.

I. Hypothyroidism.

J. Acromegaly.

K. Chronic pain.

L. Urinary frequency and nocturia due to prostatism, diabetes, diuretics, and infection.

M. Anxiety or depression.

N. Inhaled glucocorticoids.

COMMON COMPLAINTS

A. Statements regarding impaired sleep pattern:

1. Inability to fall asleep.

2. Restless throughout the night.

3. Early morning awakening with inability to fall back to sleep.

4. Difficulty concentrating during the daytime hours.

5. Feels fatigued after 8 hours of sleep, no energy.

6. Partner complains of the client's snoring.

OTHER SIGNS AND SYMPTOMS

A. Excessive daytime sleepiness.

B. Feels tense, irritable, and agitated.

C. Heightened anxiety and aggressiveness (occasionally).

D. Reports of prolonged pauses in respiration during sleep.

E. Weight gain.

F. Frontal headaches on awakening.

G. Difficulty with short-term recall.

SUBJECTIVE DATA

A. Review the onset, course, and duration of problems and symptoms.

B. Take a thorough history of the sleep problem, including the 24-hour sleep–wake cycle: sleep–wake habit history, sleep hygiene history, meal and exercise times, ambient noise, and light and temperature.

C. Identify the pattern: trouble falling asleep, trouble staying asleep (frequent awakenings), and early morning awakenings.

D. Inquire about life stresses, drug and alcohol use, and marital and family problems.

Statements suggesting self-medication with alcohol to facilitate sleep could indicate a coexistent alcohol abuse/dependence diagnosis. In clients who have developed tolerance to alcohol or sleep medications, abrupt cessation of these agents may produce increased insomnia and anxiety.

E. Determine whether the insomnia is simply normal sleep. Some "insomniacs" get ample sleep (pseudoinsomnia), and the problems are psychological.

F. Review the client's smoking and caffeine intake history.

G. Review all medications, including prescribed and over-the-counter (OTC) medications, recreational drug use, and weight loss medications.

H. Review the client's medical history: thyroid problems, hypertension, steroid use, diabetes, and cancer.

I. Conduct an interview with the client's bed partner to provide information about snoring, breathing pauses, and unusual body positions or movements. Is the partner concerned/frightened about the apneic pauses?

J. Review cardiopulmonary dysfunction: orthopnea, paroxysmal nocturnal dyspnea, or nocturnal angina.

K. If female, establish last menses; rule out pregnancy or menopause. Are there regular menses, vaginal dryness, and/or hot flashes?

L. If male, review for signs of prostatism (>age 50, hesitancy, dribbling, nocturia, frequency, incomplete emptying, etc; see the section "Benign Prostatic Hypertrophy" in Chapter 15, "Genitourinary Guidelines").

PHYSICAL EXAMINATION

A. Check pulse, respirations, blood pressure, and weight.

B. Inspect:

1. Observe general overall appearance; note grooming and behaviors during interview.

2. Evaluate eyes: pupil dilation/constriction (may indicate recent medication/nonprescription drug use).

3. Inspect nasal mucosa for erythema, edema, discharge, and nasal patency; look for septal deviation and polyps. Transilluminate sinus (if indicated).

4. Inspect the mouth for erythema, the teeth for uneven surfaces (grinding), and the retropharynx for abnormality.

C. Auscultate:

1. Heart.

2. Lungs.

3. Abdomen.

D. Palpate:

1. Conduct a neurologic examination.

2. Palpate the neck and thyroid; evaluate for goiter.

3. Check the joints for swelling and arthritis, and range of motion (ROM; rule out musculoskeletal cause).

4. Perform rectal examination if indicated for males with prostate symptoms.

5. Perform speculum/bimanual examination if indicated to evaluate menopausal atrophy and bladder complaints.

DIAGNOSTIC TESTS

A. Complete blood count (CBC) with differential.

B. Electrolytes.

C. Thyroid-stimulating hormone (TSH) or full thyroid profile, follicle-stimulating hormone (FSH), luteinizing hormone (LH).

D. Prostate-specific antigen (PSA) for males. ▶

E. Serum creatinine and blood urea nitrogen (BUN).
F. Urinalysis: Check hematuria.
G. Urine culture (if indicated).
H. Glucose tolerance test.
I. Urine drug screen.
J. Sinus x-rays.
K. Urodynamics tests if bladder issues suspected.
L. Postvoid residual (catheterization or ultrasound).
M. Nocturnal polysomnography or actigraphy.
N. Administer psychiatric evaluation (if indicated).

DIFFERENTIAL DIAGNOSES

A. Insomnia/sleep disorder:
1. Inadequate sleep hygiene: habitual behaviors that harm sleep, such as delaying morning awakening time or napping.
2. Insufficient sleep syndrome: curtailing time in bed in response to social and occupational demands, over long periods of time (shift work, circadian rhythm sleep–wake disorder).
3. Adjustment sleep disorder: acute emotional stressors (job loss or hospitalization) resulting in difficulty falling asleep due to tension and anxiety.
4. Psychophysiologic insomnia: anticipatory anxiety over the prospect of another night of sleeplessness and the next day of fatigue.
5. Narcolepsy: persistent daytime sleepiness with brief naps accompanied by vivid dreams:
a. Cataplexy or abrupt paralysis or paresis of skeletal muscles following anger, surprise, laughter, or physical exercise.
b. Hypnagogic hallucinations (vivid and often frightening dreams that occur shortly after falling asleep or on awakening).
c. Sleep paralysis, a transient global paralysis of voluntary muscles that occurs shortly after falling asleep and lasts a few seconds or minutes.
d. Disturbed and restless sleep.
B. Alcoholism and drug abuse/dependence.
C. Major depressive disorder.
D. Acute psychosis, mania, and hypomania.
E. Medical problems such as chronic pain, anxiety, depression, hyperthyroidism, epilepsy, general paresis, diabetes, benign prostatic hypertrophy, urinary problems related to age/diuretic use, cardiopulmonary dysfunction, and menopause.

PLAN

A. General interventions:
1. Identify cause of insomnia.
2. Treat physical/laboratory findings if underlying conditions exist. Treat conditions according to diagnosis made, that is, hormone replacement therapy, thyroid medications, diabetes, and so forth, as indicated. See related chapters for plans of care.
B. Client teaching: *See Client Teaching Guide for this chapter, "Sleep Disorders/Insomnia."*
1. Have the client record a 2-week log for sleep–wake habits. A sleep diary is available from the National Sleep Foundation at sleepfoundation.org/sleep-diary/sleepdiaryv6.pdf.
2. Advise the client to avoid alcohol, caffeine, and stimulating agents during the evening hours.
3. Avoid exercising prior to going to bed.
4. Encourage smoking cessation. Avoid smoking in the evening hours (*see Client Teaching Guide for this chapter, "Nicotine Dependence"*).
5. Encourage regular sleep habit/hygiene. Recommend going to bed the same time every night and waking up the same time every day.
6. Recommend keeping the bedroom cool, quiet, and dark while sleeping.
7. Encourage relaxation exercises prior to going to bed.
8. If stress/anxiety contributes to sleeping disorder, recommend counseling with a psychologist or counselor to identify and deal with issues.
9. The bed should be used only for sex and sleep. Encourage other activities or relaxation time to happen out of bed. The brain needs to associate the bed with sleeping.
10. Educate client that they need at least a 7-hour window to sleep to prevent feeling groggy the next day.
C. Pharmaceutical therapy:
1. Eliminate prescription medications (when possible) and OTC products as part of your management plan *before writing another prescription.*
a. Melatonin: 5 to 10 mg about 1 hour before bed.
b. Benadryl: 25 to 50 mg about 30 minutes before bed.
2. Only after making the assumption that the insomnia cannot be adequately treated by addressing the underlying medical problem responsible for causing the insomnia should medications for sleep be prescribed.

Do not prescribe medications for insomnia to clients with alcohol/drug or depressive disorders.

3. Use short-term pharmaceutical therapy.
4. Drug of choice: s*edative anxiolytic hypnotics:* Try initially for 1 week to establish a sleep pattern:

If insomnia continues for more than 1 month, refer the client to a physician or specialist.

a. Temazepam (Restoril) 7.5 to 30 mg at bedtime (1–2 weeks).
b. Zolpidem (Ambien) 5 to 10 mg at bedtime (4 weeks maximum).
c. Eszopiclone (Lunesta) 2 to 3 mg at bedtime. Start with 1 mg in older adults.
d. Ramelteon (Rozerem) 8 mg PO 30 minutes before bedtime. Do not take with meals.
e. Suvorexant (Belsomra) 5 to 20 mg within 30 minutes of bedtime.
f. Alprazolam (Xanax) 0.25 to 1 mg at bedtime (shorter acting so may prevent fatigue the following day).

Anxiolytic agents such as diazepam (Valium) and alprazolam (Xanax) tend to increase the duration and frequency of sleep apneas and are contraindicated in clients with possible/undiagnosed apnea spells.

5. Sedating antihistamines are not recommended for use over 4 months. Side effects in older adult clients outweigh benefit.
a. Hydroxyzine HCl (Atarax) 50 to 100 mg at bedtime.
b. Hydroxyzine pamoate (Vistaril) 50 to 100 mg at bedtime.
6. Antidepressants with sedating properties:
a. Trazodone HCl (Desyrel) 50 to 100 mg at bedtime.
b. Mirtazapine (Remeron) 7.5 to 15 mg at bedtime.

FOLLOW-UP

A. Follow up in 1 week to assess the client's status.

B. Then, follow up every 2 weeks to check the client's progress.

C. Once positive change is seen, the client can be seen monthly as needed.

D. Assess the client's potential for suicide with every office visit.

E. If coexisting medication conditions exist, refer to medical diagnosis for follow-up recommendations.

CONSULTATION/REFERRAL

A. Refer to a psychiatric clinician for medication management and psychotherapy after initial assessment if psychiatric differential diagnosis is made.

B. Refer for psychological testing.

C. If insomnia continues for more than 1 month, refer to a physician if the client requires a sedative anxiolytic hypnotic for more than 4 weeks.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Sleep difficulties are more prevalent in the second and third trimesters of pregnancy due to the growing size of the uterus and difficulty finding comfortable sleeping positions. Treat with comfort measures.

B. Adults:

1. The "right" amount of sleep results in optimal daytime alertness and a sense of mental efficiency and well-being.

2. Daytime naps often interfere with the quality of night sleep. Encourage eliminating naps during the day.

3. Nocturia and disturbed sleep are the symptoms that cause older males to seek medical help with prostatism.

C. Geriatrics:

1. With aging, it is normal for sleep time to decrease (<7 hours), with a tendency toward sleep fragmentation and an increase in the frequency of awakenings and brief arousals. Explain this to older adults to avoid "worry over sleeplessness."

2. Always assess depression in older adult clients.

3. Start with the lowest dose of pharmaceutical agents. Avoid use of sedating antihistamines and hypnotics in older adults due to potential for delirium, falls, fractures, and car accidents.

4. Chronic pain is a leading cause of sleep disruption in older adults with degenerative joint pain.

5. Review older adults' medications and dosage for insomnia due to known side effects or as a medication toxicity.

6. Gastroesophageal reflux is commonly seen in geriatric clients at nighttime and causes them to wake up at night. Correcting the reflux with sleeping position, avoiding late night meals and spicy food, prescribing medications (proton pump inhibitors [PPIs]), and making possible weight loss interventions could improve sleep disturbance in these clients.

7. Clients with end-stage renal disease may have nocturnal leg movement disorders and anemia due to renal failure. Improving the anemia will improve the insomnia and will decrease the leg movements.

D. Partners:

1. Those who find it necessary to sleep in another room because of their partner's snoring should refer the snorer to the primary care physician/advanced practice nurse to rule out an OSA syndrome.

RESOURCE

National Sleep Foundation: www.sleepfoundation.org

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SUBSTANCE USE DISORDERS

DEFINITION

A. Substance use disorders range from mild to severe forms of pathologic patterns of behavior related to substance use. The *Diagnostic and Statistical Manual of Mental Disorders,* Fifth Edition *(DSM-5;* American Psychiatric Association [APA], 2013) specifies 10 separate classes of drugs that are included in substance-related disorders: alcohol; caffeine; cannabis; hallucinogens; inhalants; opioids; sedatives, hypnotics, and anxiolytics; stimulants (amphetamine-type substances, cocaine, and other stimulants); tobacco; and other (or unknown) substances. All of these substances may be used in excess, producing an intense activation of the brain reward system.

B. The diagnosis of substance use disorders is based on impaired control over substance use, the use of larger amounts of a substance over a greater period of time, unsuccessful attempts to discontinue use of a substance, and spending a great amount of time to obtain, use the substance, and recover from its effects. Substance use may result in inability to maintain responsibilities at home, school, or work. Normal daily activities may be decreased or discontinued due to substance use. Risky behaviors may be exhibited, as continued substance use results in physical and psychological problems and the individual fails to abstain from the substance, despite the problems associated with its use.

INCIDENCE

A. From 1999 to 2018, the age-adjusted rate of opioid-involved overdose deaths nearly quadrupled in the United States. The biggest driver of overdose deaths has been synthetic opioids (other than methadone), in particular illicitly manufactured fentanyl where the number of attributable overdose deaths swelled from 0.3 to 9.9 per 100,000 standard population from 1999 to 2018. The rate of heroin-related overdose deaths also rose sharply from 1999 to 2017, from 0.7 to 4.9 per 100,000. During this time, overdose deaths involving natural and semisynthetic opioid analgesics, such as oxycodone and hydrocodone, increased from 1.0 to 4.4 per 100,000. Altogether, opioids accounted for 71% of the 70,630 drug overdose deaths in the United States in 2019.

B. Alcohol: Binge drinking is on the rise among older adults—more than 10% of adults aged 65 and older reported binge drinking in the past month and the prevalence is increasing. The increase in this group is of particular concern because many older adults use medications that can interact with alcohol, have health conditions that can be exacerbated by alcohol, and may be more susceptible to alcohol-related falls and other accidental injuries.

C. The National Institute on Drug Abuse (NIDA) reports the abuse of tobacco, alcohol, and illicit drugs costs over $600 billion annually in costs related to healthcare, lost work productivity, and crime. Drug use tends to be highest among people in their teens to 20s; however, an increase has also been noted in persons in their 50s. This is thought to be secondary to the aging of "baby boomers," whose rate of illicit drug use has been higher than that of previous generations. Alcohol abuse is the primary substance abuse problem of those older than age 50.

D. NIDA estimates that although 23.9 million Americans need treatment for substance use disorder, only 2.5 million have received treatment. See Table 25.2 for the NIDA list of commonly abused drugs and Table 25.3 for the NIDA list of abused prescription drugs.

E. Studies indicate that 8% of adults in the United States had a substance use disorder in the past 12 months. Approximately 40% of hospital admissions are related to substance abuse or to the effects of using substances.

F. Approximately 90% of the American population consumes some alcohol (at one time or another). Alcohol use is believed to be involved in 20% to 50% of all hospital admissions, but alcohol use disorders are formally diagnosed less than 5% of the time; 5% to 7% of Americans have alcoholism in a given year, and 13% will have it sometime during their life. Prevalence rates of alcoholism are 9.5% for males and 3.3% for females. Binge and heavy drinking is more common in males than females. It is highest in males aged 18 to 64 and females aged 18 to 24.

G. Approximately 21.3% of Americans use tobacco products. One study states that there is a link between early nicotine use and alcohol abuse and depression. Smoking before the age of 13 significantly increases the risk of drug dependence. The smoking trend is decreasing slowly from the year 2002 when it was 26%. Teen smoking has also declined.

H. It is estimated that 37% of the population aged 12 or older have used an illicit psychoactive drug at least once in their lifetime.

A substance abuse problem is recognized in as few as 1 in 20 substance-abusing clients seeking medical attention.

PATHOGENESIS

A. No single gene has been identified as the culprit in the predisposition to substance dependence. Certain biologic features seem to be inherited by first-degree relatives (particularly males) of alcoholics, for example, a resistance to intoxication, a subnormal cortisol rise after drinking, and a subnormal epinephrine release following stress.

B. Some theories postulate alterations in the metabolism of alcohol and drugs in people who are dependent. Studies pertaining to alcohol have included research into genetic heritability, flawed metabolism of alcohol by alcoholics, insensitivity to alcohol inherited by alcoholics (thus tending to increase tolerance or ability to know when to stop), and alterations in brain waves in alcoholics.

C. Although much of the research is specific to only one drug, much of what we know about the research can be applied to other drugs. There appears to be a higher rate of substance dependence, not limited to alcohol, in children of alcoholics.

PREDISPOSING FACTORS

A. Factors vary among individuals, and no one factor can account entirely for the risk of substance abuse. Studies indicate a high correlation between substance use and the presence of psychiatric disorders, especially anxiety disorders, depression, schizophrenia, and, in females, eating disorders:

1. Genetic.
2. Familial.
3. Environmental.
4. Occupational.
5. Socioeconomic.
6. Cultural.
7. Personality.
8. Life stress.
9. Psychiatric comorbidity.
10. Biologic.
11. Social learning and behavioral conditioning.

PRINCIPLES OF DRUG ADDICTION TREATMENT

A. More than three decades of scientific research show that treatment can help drug-addicted individuals stop drug use, avoid relapse, and successfully recover their lives. Based on this research, 13 fundamental principles that characterize effective drug abuse treatment have been developed. These principles are detailed in *NIDA's Principles of Drug Addiction Treatment: A Research-Based Guide*. The guide also describes different types of science-based treatments and provides answers to commonly asked questions.

1. Addiction is a complex but treatable disease that affects brain function and behavior. Drugs alter the brain's structure and how it functions, resulting in changes that persist long after drug use has ceased. This may help explain why abusers are at risk for relapse even after long periods of abstinence.
2. No single treatment is appropriate for everyone. Matching treatment settings, interventions, and services to an individual's particular problems and needs is critical to their ultimate success.
3. Treatment needs to be readily available. Because drug-addicted individuals may be uncertain about entering treatment, taking advantage of available services the moment people are ready for treatment is critical. Potential clients can be lost if treatment is not immediately available or readily accessible.
4. Effective treatment attends to multiple needs of the individual, not just their drug abuse. To be effective, treatment must address the individual's drug abuse and any associated medical, psychological, social, vocational, and legal problems.
5. Remaining in treatment for an adequate period of time is critical. The appropriate duration for an individual depends on the type and degree of their problems and needs. Research indicates that most addicted individuals need at least 3 months in treatment to significantly reduce or stop their drug use and that the best outcomes occur with longer durations of treatment.
6. Counseling—individual and/or group—and other behavioral therapies are the most commonly used forms of drug abuse treatment. Behavioral therapies vary in their focus and may involve addressing a client's motivations to change, building skills to resist drug use, replacing drug-using activities with constructive and rewarding activities, improving problem-solving skills, and facilitating better interpersonal relationships.
7. Medications are an important element of treatment for many clients, especially when combined with counseling and other behavioral therapies. For example, methadone ▶

TABLE 25.2 COMMONLY ABUSED DRUGS

NIDA NATIONAL INSTITUTE ON DRUG ABUSE

Visit NIDA at www.drugabuse.gov
National Institutes of Health
U.S. Department of Health and Human Services
NIH . . . Turning Discovery Into Health

Substances: Category and Name	Examples of *Commercial* and Street Names	DEA Schedule[a]/ Mode of Administration[b]	Acute Effects/Health Risks
Tobacco			Increased blood pressure and heart rate/chronic lung disease; cardiovascular disease; stroke; cancers of the mouth, pharynx, larynx, esophagus, stomach, pancreas, cervix, kidney, bladder, and acute myeloid leukemia; adverse pregnancy outcomes; addiction
Nicotine	Found in cigarettes, cigars, bidis, and smokeless tobacco (snuff, spit tobacco, chew)	Not scheduled/ smoked, snorted, chewed	
Alcohol: Alcohol remains the most commonly used drug in crimes of sexual assault.			In low doses, euphoria, mild stimulation, relaxation, lowered inhibitions; in higher doses, drowsiness, slurred speech, nausea, emotional volatility, loss of coordination, visual distortions, impaired memory, sexual dysfunction, loss of consciousness/increased risk of injuries, violence, fetal damage (in pregnant females); depression; neurologic deficits; hypertension; liver and heart disease; addiction; fatal overdose
Alcohol (ethyl alcohol)	Found in liquor, beer, and wine	Not scheduled/ swallowed	
Cannabinoids			Euphoria; relaxation; slowed reaction time; distorted sensory perception; impaired balance and coordination; increased heart rate and appetite; impaired learning, memory; anxiety; panic attacks; psychosis/cough; frequent respiratory infections; possible mental health decline
Marijuana	Blunt, dope, ganja, grass, herb, joint, bud, Mary Jane, pot, reefer, green, trees, smoke, sinsemilla, skunk, weed	I/smoked, swallowed	
Hashish	Boom, gangster, hash, hash oil, hemp	I/smoked, swallowed	Addiction
Opioids			Euphoria; drowsiness; impaired coordination; dizziness; confusion; nausea; sedation; feeling of heaviness in the body; slowed or arrested breathing/constipation; endocarditis; hepatitis; HIV; addiction; fatal overdose
Heroin	Diacetylmorphine: smack, horse, brown sugar, dope, H, junk, skag, skunk, white horse, China white; cheese (with OTC cold medicine and antihistamine)	I/injected, smoked, snorted	
Opium	Laudanum, paregoric: big O, black stuff, block, gum, hop	II, III, V/swallowed, smoked	
Stimulants			Increased heart rate, blood pressure, body temperature, metabolism; feelings of exhilaration; increased energy, mental alertness; tremors; reduced appetite; irritability; anxiety; panic; paranoia; violent behavior; psychosis/ weight loss; insomnia; cardiac or cardiovascular complications; stroke; seizures; addiction Also, for cocaine: nasal damage from snorting Also, for methamphetamine: severe dental problems
Cocaine	Cocaine hydrochloride: blow, bump, C, candy, Charlie, coke, crack, flake, rock, snow, toot	II/snorted, smoked, injected	
Amphetamine	*Biphetamine, Dexedrine:* bennies, black beauties, crosses, hearts, LA turnarounds, speed, truck drivers, uppers	II/swallowed, snorted, smoked, injected	
Methamphetamine	*Desoxyn:* meth, ice, crank, chalk, crystal, fire, glass, go fast, speed	II/swallowed, snorted, smoked, injected	

(*continued*)

TABLE 25.2 COMMONLY ABUSED DRUGS (*CONTINUED*)

NIDA NATIONAL INSTITUTE ON DRUG ABUSE

Visit NIDA at www.drugabuse.gov
National Institutes of Health
U.S. Department of Health and Human Services
NIH . . . Turning Discovery Into Health

Substances: Category and Name	Examples of *Commercial* and Street Names	DEA Schedule[a]/ Mode of Administration[b]	Acute Effects/Health Risks
Club Drugs: commonly associated with sexual assault			MDMA: mild hallucinogenic effects; increased tactile sensitivity, empathic feelings; lowered inhibition; anxiety; chills; sweating; teeth clenching; muscle cramping/ sleep disturbances; depression; impaired memory; hyperthermia; relaxation; confusion; memory loss; dizziness; impaired coordination/addiction GHB: drowsiness; nausea; headache; disorientation; loss of coordination; memory loss/unconsciousness; seizures; coma addiction Flunitrazepam: sedation; muscle
MDMA	*Ecstasy,* Adam, clarity, Eve, lover's speed, peace, uppers	I/swallowed, snorted, injected	
Flunitrazepam	*Rohypnol:* forget-me pill, Mexican Valium, R2, roach, Roche, roofies, roofinol, rope, rophies Mexican Valium, R2, roach, Roche, roofies, roofinol, rope, rophies	IV/swallowed, snorted	
GHB[c]	GHB: G, Georgia home boy, grievous bodily harm, liquid ecstasy, soap, scoop, goop, liquid X	I/swallowed	
Dissociative Drugs			Feelings of being separate from one's body and environment; impaired motor function/anxiety; tremors; numbness; memory loss; nausea Also, for ketamine: analgesia; impaired memory; delirium; respiratory depression and arrest; death Also, for PCP and analogs: analgesia; psychosis; aggression; violence; slurred speech; loss of coordination; hallucinations Also, for DXM: euphoria; slurred speech; confusion; dizziness; distorted visual perceptions
Ketamine	*Ketalar SV:* cat Valium, K, Special K, vitamin K	III/injected, snorted, smoked	
PCP and analogs	Phencyclidine: angel dust, boat, hog, love boat, peace pill	I, II/swallowed, smoked, injected	
Salvia divinorum	Salvia, Shepherdess's Herb, Maria Pastora, magic mint, Sally-D	Not scheduled/ chewed, swallowed, smoked	
DXM	*Found in some cough and cold medications:* robotripping, robo, triple C	Not scheduled/ swallowed	
Hallucinogens			Altered states of perception and feeling; hallucinations; nausea Also, for LSD and mescaline: increased body temperature, heart rate, blood pressure; loss of appetite; sweating; sleeplessness; numbness; dizziness; weakness; tremors; impulsive behavior; rapid shifts in emotion Also, for LSD: flashbacks, hallucinogen persisting perception disorder Also, for psilocybin: nervousness; paranoia; panic
LSD	LSD: acid, blotter, cubes, microdot, yellow sunshine, blue heaven	I/swallowed, absorbed through mouth tissues	
Mescaline	Buttons, cactus, mesc, peyote	I/swallowed, smoked	
Psilocybin	Magic mushrooms, purple passion, shrooms, little smoke	I/swallowed	
Other Compounds			Steroids: no intoxication effects/hypertension; blood clotting and cholesterol changes; liver cysts; hostility and aggression; acne; in adolescents: premature stoppage of growth; in males: prostate cancer, reduced sperm production, shrunken testicles, breast enlargement; in females: menstrual irregularities, development of beard and other masculine characteristics Inhalants (varies by chemical): stimulation; loss of inhibition; headache; nausea or vomiting; slurred speech; loss of motor coordination; wheezing/cramps; muscle weakness; depression; memory impairment; damage to cardiovascular and nervous systems; unconsciousness; sudden death
Anabolic steroids	*Anadrol, Oxandrin, Durabolin, Depo-Testosterone, Equipoise:* roids, juice, gym candy, pumpers	III/injected, swallowed, applied to skin	

(*continued*)

TABLE 25.2 COMMONLY ABUSED DRUGS (*CONTINUED*)

NIDA NATIONAL INSTITUTE ON DRUG ABUSE	**Visit NIDA at www.drugabuse.gov National Institutes of Health U.S. Department of Health and Human Services NIH . . . Turning Discovery Into Health**		
Substances: Category and Name	**Examples of *Commercial* and Street Names**	**DEA Schedule[a]/ Mode of Administration[b]**	**Acute Effects/Health Risks**
Inhalants	Solvents (paint thinners, gasoline, glues); gases (butane, propane, aerosol propellants, nitrous oxide); nitrites (isoamyl, isobutyl, cyclohexyl): laughing gas, poppers, snappers, whippets	Not scheduled/ inhaled through nose or mouth	
Prescription Medications			
CNS depressants	For more information on prescription medications, please visit www.nida.nih.gov/DrugPages/PrescripDrugs Chart.html.		
Stimulants			
Opioid pain relievers			

CNS, central nervous system; DEA, Drug Enforcement Agency; DXM, dextromethorphan; GHB, gamma-hydroxybutyrate; LSD, lysergic acid diethylamide; MDMA, methylenedioxymethamphetamine; NIDA, National Institute on Drug Abuse; OTC, over-the-counter; PCP, *Pneumocystis carinii* pneumonia.

Source: From National Institute on Drug Abuse (2012). Commonly abused drugs. https://www.drugabuse.gov/sites/default/files/cadchart.pdf

[a]DEA Scheduled Drugs: Schedule I and II drugs have a high potential for abuse. They require greater storage security and have a quota on manufacturing, among other restrictions. Schedule I drugs are available for research only and have no approved medical use; Schedule II drugs are available only by prescription (unrefillable) and require a form for ordering. Schedule III and IV drugs are available by prescription, may have five refills in 6 months, and may be ordered orally. Some Schedule V drugs are available OTC.

[b]Administration: Some of the health risks are directly related to the route of drug administration. For example, injection drug use can increase the risk of infection through needle contamination with staphylococci, HIV, hepatitis, and other organisms.

TABLE 25.3 COMMONLY ABUSED PRESCRIPTION DRUGS

NIDA NATIONAL INSTITUTE ON DRUG ABUSE	**Visit NIDA at www.drugabuse.gov National Institutes of Health U.S. Department of Health and Human Services**		
Substances: Category and Name	**Examples of *Commercial* and Street Names**	**DEA Schedule[a]/ Mode of Administration**	**Intoxication Effects/Health Risks**
Depressants			Sedation/drowsiness, reduced anxiety, feelings of well-being, lowered inhibitions, slurred speech, poor concentration, confusion, dizziness, impaired coordination and memory/ slowed pulse, lowered blood pressure, slowed breathing, tolerance, withdrawal, addiction; increased risk of respiratory distress and death when combined with alcohol for barbiturates—euphoria, unusual excitement, fever, irritability/ life-threatening withdrawal in chronic users
Barbiturates	*Amytal, Nembutal, Seconal, Phenobarbital*: barbs, reds, red birds, phennies, tooies, yellows, yellow jackets	II, III, IV/injected, swallowed	
Benzodiazepines	*Ativan, Halcion, Librium, Valium, Xanax, Klonopin*: candy, downers, sleeping pills, tranks	IV/swallowed	
Sleep medications	*Ambien (zolpidem), Sonata (zaleplon), Lunesta (eszopiclone)*	IV/swallowed	

(*continued*)

TABLE 25.3 COMMONLY ABUSED PRESCRIPTION DRUGS (*CONTINUED*)

NIDA NATIONAL INSTITUTE ON DRUG ABUSE

Visit NIDA at www.drugabuse.gov
National Institutes of Health
U.S. Department of Health and Human Services

Substances: Category and Name	Examples of *Commercial* and Street Names	DEA Schedule[a]/ Mode of Administration	Intoxication Effects/Health Risks
Opioids and Morphine Derivatives[b]			Pain relief, euphoria, drowsiness, sedation, weakness, dizziness, nausea, impaired coordination, confusion, dry mouth, itching, sweating, clammy skin, constipation/slowed or arrested breathing, lowered pulse and blood pressure, tolerance, addiction, unconsciousness, coma, death; risk of death increased when combined with alcohol or other CNS depressants For fentanyl—80–100 times more potent analgesic than morphine for oxycodone—muscle relaxation/twice as potent analgesic as morphine; high abuse potential For codeine—less analgesia, sedation, and respiratory depression than morphine For methadone—used to treat opioid addiction and pain; significant overdose risk when used improperly
Codeine	*Empirin with Codeine, Fiorinal with Codeine, Robitussin A-C, Tylenol with Codeine:* Captain Cody, Cody, schoolboy (with glutethimide: doors and fours, loads, pancakes and syrup)	II, III, IV/injected, swallowed	
Morphine	*Roxanol, Duramorph:* M, Miss Emma, monkey, white stuff	II, III/injected, swallowed, smoked	
Methadone	*Methadone, Dolophine:* fizzies, amidone (with MDMA: chocolate chip cookies)	II/swallowed, injected	
Fentanyl and analogues	*Actiq, Duragesic, Sublimaze:* Apache, China girl, dance fever, friend, goodfella, jackpot, murder 8, TNT, Tango and Cash	II/injected, smoked, snorted	
Other opioid pain relievers: OxyContin HCl Hydrocodone Bitartrate Hydromorphone Oxymorphone Meperidine Propoxyphene	*Tylox, OxyContin, Percodan, Percocet:* Oxy, O.C., oxycontin, oxycet, hillbilly heroin, percs *Vicodin, Lortab, Lorcet:* vike, Watson-387 *Dilaudid:* juice, smack, D, footballs, dillies *Opana, Numorphan, Numorphone:* biscuits, blue heaven, blues, Mrs. O, octagons, stop signs, O Bomb *Demerol, meperidine hydrochloride:* demmies, painkiller *Darvon, Darvocet*	II, III, IV/chewed, swallowed, snorted, injected, suppositories	
Stimulants			Feelings of exhilaration, increased energy, mental alertness/increased heart rate, blood pressure, and metabolism, reduced appetite, weight loss, nervousness, insomnia, seizures, heart attack, stroke For amphetamines: rapid breathing, tremor, loss of coordination, irritability, anxiousness, restlessness/delirium, panic, paranoia, hallucinations, impulsive behavior, aggressiveness, tolerance, addiction For methylphenidate: increase or decrease in blood pressure, digestive problems, loss of appetite, weight loss
Amphetamines	*Biphetamine, Dexedrine, Adderall:* bennies, black beauties, crosses, hearts, LA turnarounds, speed, truck drivers, uppers	II/injected, swallowed, smoked, snorted	
Methylphenidate	*Concerta, Ritalin:* JIF, MPH, R-ball, Skippy, the smart drug, vitamin R	II/injected, swallowed, snorted	
Other Compounds			Euphoria, slurred speech/increased heart rate and blood pressure, dizziness, nausea, vomiting, confusion, paranoia, distorted visual perceptions, impaired motor function
DXM	*Found in some cough and cold medications:* robotripping, robo, triple C	Not scheduled/swallowed	

CNS, central nervous system; DEA, Drug Enforcement Agency; DXM, dextromethorphan; MDMA, methylenedioxymethamphetamine; NIDA, National Institute on Drug Abuse.

Source: From National Institute on Drug Abuse. (2011). Commonly abused prescription drugs. https://www.drugabuse.gov/sites/default/files/rx_drugs_placemat_508c_10052011.pdf

[a]DEA Schedule: Schedule I and II drugs have a high potential for abuse. They require greater storage security and have a quota on manufacturing, among other restrictions. Schedule I drugs are available for research only and have no approved medical use. Schedule II drugs are available only by prescription and require a new prescription for each refill. Schedule III and IV drugs are available by prescription, may have five refills in 6 months, and may be ordered orally. Most Schedule V drugs are available over the counter.

[b]Administration: Taking drugs by injection can increase the risk of infection through needle contamination with staphylococci, HIV, hepatitis, and other organisms. Injection is a more common practice for opioids, but risks apply to any medication taken by injection.

and buprenorphine are effective in helping individuals addicted to heroin or other opioids stabilize their lives and reduce their illicit drug use. Also, for persons addicted to nicotine, a nicotine replacement product (nicotine patches or gum) or an oral medication (bupropion or varenicline) can be an effective component of treatment when part of a comprehensive behavioral treatment program.

8. An individual's treatment and services plan must be assessed continually and modified as necessary to ensure it meets their changing needs. A client may require varying combinations of services and treatment components during the course of treatment and recovery. In addition to counseling or psychotherapy, a client may require medication, medical services, family therapy, parenting instruction, vocational rehabilitation, and/or social and legal services. For many clients, a continuing care approach provides the best results, with treatment intensity varying according to a person's changing needs.

9. Many drug-addicted individuals also have other mental disorders. Because drug abuse and addiction—both of which are mental disorders—often co-occur with other mental illnesses, clients presenting with one condition should be assessed for the other(s). And when these problems co-occur, treatment should address both (or all), including the use of medications as appropriate.

10. Medically assisted detoxification is only the first stage of addiction treatment and by itself does little to change long-term drug abuse. Although medically assisted detoxification can safely manage the acute physical symptoms of withdrawal, detoxification alone is rarely sufficient to help addicted individuals achieve long-term abstinence. Thus, clients should be encouraged to continue drug treatment following detoxification.

11. Treatment does not need to be voluntary to be effective. Sanctions or enticements from family, employment settings, and/or the criminal justice system can significantly increase treatment entry, retention rates, and the ultimate success of drug treatment interventions.

12. Drug use during treatment must be monitored continuously, as lapses during treatment do occur. Knowing their drug use is being monitored can be a powerful incentive for clients and can help them withstand urges to use drugs. Monitoring also provides an early indication of a return to drug use, signaling a possible need to adjust an individual's treatment plan to better meet their needs.

13. Treatment programs should assess clients for the presence of HIV/AIDS, hepatitis B and C, tuberculosis, and other infectious diseases, as well as provide targeted risk-reduction counseling to help clients modify or change behaviors that place them at risk of contracting or spreading infectious diseases. Targeted counseling specifically focused on reducing infectious disease risk can help clients further reduce or avoid substance-related and other high-risk behaviors. Treatment providers should encourage and support HIV screening and inform clients that highly active antiretroviral therapy (HAART) has proven effective in combating HIV, including among drug-abusing populations.

COMMON COMPLAINTS

A. Clients' complaints will be focused on the symptoms of the problem rather than the substance dependence. The problem itself will be avoided through the use of denial, minimization, blaming, and projection (all signs of the disease of substance dependence):

1. Chronic anxiety and tension.
2. Insomnia.
3. Chronic depression.
4. Headaches and/or back pain.

Consider clients who present frequently with somatic complaints, such as back pain or headache, as "drug seeking," especially when the client knows what drugs work best or asks for specific narcotic analgesics.

5. Blackouts.
6. Gastrointestinal problems.
7. Tachycardia/palpitations.
8. Frequent falls or minor injuries.
9. Problems with a loved one; problems at work or with friends.

Substance abuse should be suspected in all clients who present with accidents or signs of repeated trauma, especially to the head.

OTHER SIGNS AND SYMPTOMS

A. Defensiveness about alcohol/drug use or vagueness with answers.

B. History of problems with family life, marital relationships, work, finances, and physical health.

C. Change in spiritual beliefs (stops attending religious services).

D. Unexplained job changes and multiple traffic accidents.

E. History of impulsive behavior, fighting, or unexplained falls.

F. Arrest for public drunkenness, driving under the influence, or illegal activity when alcohol/drugs were involved.

G. Tremors (shakes).

H. Delirium tremors (DTs).

I. Seizures related to drugs.

J. Hallucinations.

K. History of chronic family chaos and instability.

L. Physical indications of chronic alcohol/drug use include spider angiomas, ruddy nose and face, nasal lesions, bruxism, swollen features, bruises, needle marks/tracks, cutaneous abscesses, malnourished, anemia, jaundice, and severe dental problems such as "meth mouth."

M. Active withdrawal symptoms include nausea and vomiting, malaise, weakness, tachycardia, diaphoresis, tremors, lightheadedness or dizziness, insomnia, irritability, confusion, perceptual abnormalities or hallucinations (auditory, visual, or tactile), paresthesia, blurred vision, diarrhea, anorexia, abdominal cramps, severe depression, severe anxiety, piloerection, fasciculation (muscle twitching), rhinorrhea, fever, elevated blood pressure and pulse, tinnitus, nystagmus, delirium, or seizures.

N. Overdose symptoms related to drug(s) include seizures, cardiovascular depression/collapse, and respiratory depression/collapse. Be prepared to provide cardiovascular and respiratory support and supportive care until transport.

SUBJECTIVE DATA

A. Review the onset, duration, and course of presenting complaints.

B. The U.S. Preventive Services Task Force (USPSTF) recommends that all adults be screened in primary care for alcohol and drug use.

C. Question the clients regarding relatives with a history of alcohol, tobacco, or drug use or problems pertaining to use.

D. When questioning the clients, assume some use; for example, "At what age did you first start drinking?" Start with ▶

the least invasive questions first: cigarettes, over-the-counter (OTC) medications, and prescription medications, then alcohol, marijuana, stimulants, opiates, sedatives, hypnotics, benzodiazepines, barbiturates, hallucinogens, inhalants, steroids, and other drugs.

E. Review use of the following drugs concerning quantity and type (if cigarettes, brand smoked; if alcohol, type of alcohol: beer, wine, hard liquor), and age at initiation. Query regarding previous attempts to stop use.

F. Start with the past and proceed to the present with use; include first use of the mood-altering substance, amounts, and the last use of the particular substance and amount.

G. Follow the CAGE test. The **CAGE** (2 out of 4) is highly predictive of addiction:

1. Have you ever tried to **c**ut down on your alcohol/drug use?

2. Do you get **a**nnoyed if someone mentions your use is a problem?

3. Do you ever feel **g**uilty about your use?

4. Do you ever have an "**e**ye-opener" first thing in the morning after you've been drinking or using the night before?

H. If the client admits drinking or drug use, ascertain specific amounts and last use of each substance.

I. Establish usual weight and recent loss and in what length of time.

J. Determine whether client experiences suicidal ideation and if there is a history of past attempts (see the section "Suicide" of this chapter).

PHYSICAL EXAMINATION

A. Check temperature, pulse, respirations, blood pressure, and weight.

B. Inspect:

1. Observe general appearance, dress, grooming, breath odor, wasted appearance, attitude, sad affect, psychomotor retardation, or tremors.

2. Conduct a dermal examination for spider angiomas, bruises, track marks, color, pallor, rash, jaundice, petechiae, and gynecomastia in males (hallucinogens).

3. Examine the eyes for sclera color and features, pupil size, and reactivity.

4. Inspect the nasal mucosa for erythema, edema, spider telangiectasia, and discharge; look for septal lesions or perforation, deviation, and polyps.

5. Inspect the mouth/pharynx: oral lesions, poor dental hygiene, erythema, and teeth for uneven surfaces, tooth decay, and gum erosion.

C. Palpate:

1. Palpate the neck and thyroid.

2. Palpate the axilla and groin for lymphadenopathy.

3. Palpate the abdomen; note hepatomegaly/tenderness.

D. Percuss:

1. Percuss the chest; note pulmonary consolidation.

2. Percuss the abdomen for hepatosplenomegaly.

E. Auscultate:

1. Auscultate the heart for murmur, new S4 gallop, single S2, and arrhythmias.

2. Auscultate the lungs for rales, effusion, and consolidation.

F. Neurologic examination/mental status.

DIAGNOSTIC TESTS

A. Blood alcohol level.

B. Cotinine level (nicotine; where available).

C. Urine drug screen.

D. Complete blood count (CBC) with differential.

E. Platelet count.

F. HIV or hepatitis.

Intravenous drug use contributes strongly to the spread of AIDS, hepatitis B and C, and other infectious diseases. Consider evaluation for sexually transmitted infections.

G. Antinuclear antibody, erythrocyte sedimentation rate (ESR), and rheumatoid factor.

H. Electrolytes.

I. Liver panel:

1. Elevated liver enzymes can also be attributed to overuse of acetaminophen (Tylenol), found in combination with opiates.

J. Blood cultures (fever).

K. Bone density studies:

1. Clients who have been drinking for years should have bone density studies done because alcohol increases the risk for osteoporosis.

DIFFERENTIAL DIAGNOSES

A. Substance use disorder.

B. Chronic pain syndrome.

C. Anxiety.

D. Depression.

PLAN

A. General interventions:

1. Discuss your concerns about alcohol, nicotine, or drug use and discuss addiction treatment with the client.

2. At each office visit, provide support to help prevent relapse. If relapse occurs, encourage the client to try again immediately.

3. Consider signing a contract with the client to stop smoking, drinking, or using drugs.

4. Assess potential for suicide with every office visit.

5. If possible, obtain confirmation of the client's abstinence from a family member.

6. Stress the importance of 12-step meetings such as Alcoholics Anonymous (AA), Cocaine Anonymous (CA), and Narcotics Anonymous (NA).

7. Have the client sign a written release of information so that you can speak with a rehabilitation counselor. If the client is willing, refer to an alcohol and drug treatment facility or smoking cessation program, after initial assessment and differential diagnosis are made.

8. Treat physical/laboratory findings as indicated.

9. Identify potential withdrawal symptoms from the cessation of stimulants, such as caffeine intake reduction, alcohol, and drug use.

10. If malnourished, discuss dietary needs and treatment.

B. Client teaching: *See Client Teaching Guide for this chapter, "Alcohol and Drug Dependence."*

1. Educate the client about the impact of alcohol, tobacco, and drugs on physical/emotional health.

a. Provide information for the client to read at home.

C. Pharmaceutical therapy:

1. Nicotine replacement: Consider nicotine replacement for those who smoke more than one pack of cigarettes per day or who smoke their first cigarette within 30 minutes of waking. Stress that there is "no smoking" while using nicotine replacement.

a. Nicotine patch:
   i. 21 mg/24 hours for 4 weeks. *Then*
   ii. 14 mg/24 hours for 2 weeks. *Then*
   iii. 7 mg/24 hours for 2 weeks.
b. Nicotine gum:
   i. 2 to 4 mg of nicotine gum per hour with maximum of 24 pieces per day for up to 12 weeks.
c. Nicotine lozenges:
   i. 2 to 4 mg every 1 to 2 hours for 6 weeks.
   ii. 2 mg every 2 to 4 hours for 3 weeks.
   iii. 2 mg every 4 to 8 hours for 3 weeks.

2. Non-nicotine therapy:

a. Adults: bupropion (Zyban, Wellbutrin) 150 mg every day for 3 days, then increase to 150 mg BID. Treat for 7 to 12 weeks. The client may continue to smoke during the first 2 weeks of starting medication. This medication should not be given to clients with seizure disorders.

b. Varenicline; brand withdrawn in the United States on September 16, 2021, due to contamination with potential carcinogen N-nitroso-varenicline. Generic forms may contain N-nitroso-varenicline above Food and Drug Administration (FDA) acceptable daily intake limit: Start at 0.5 mg/d for the first 3 days, then for the next 4 days, 0.5 mg BID. After the first 7 days, the dose is 1 mg BID.
   i. Encourage the client to choose a stop date for smoking and start the Chantix 1 to 2 weeks prior to this stop date.
   ii. Clients should be encouraged to quit even if they have relapses.
   iii. Instruct the client that the most common side effects of varenicline are insomnia, vivid or strange dreams, and nausea. Advise that side effects are usually transient.
   iv. Give precautions to the client regarding potential side effects of mood swings, aggression, homicidal thoughts, psychosis, anxiety, and panic disorder, which may occur on rare occasions.
   v. See package inserts or *Physicians' Desk Reference* for detailed instructions.

3. Detoxification and methadone maintenance should be performed by specially licensed and trained professionals.

4. Disulfiram (Antabuse) therapy is not recommended. Clients who consume alcohol after taking Antabuse can become extremely ill.

5. Refer the client to physician or specialist if they are experiencing withdrawals; consider admission to rehabilitation center for detoxification and treatment.

FOLLOW-UP

A. Make a follow-up appointment weekly. Make contact with the referral source (smoking cessation program, alcohol/drug rehabilitation program) before the next follow-up visit to check on the client's progress. At weekly visit, question the client regarding compliance.

B. Order blood alcohol, urine drug screen, or nicotine level (as appropriate) with every office visit while in outpatient treatment and throughout the year following treatment.

C. Once positive change is seen, the client can be seen monthly. Discuss changes the client has made, past relapses, circumstances under which they occurred, and any special concerns.

D. Refer to the medical diagnosis for other applicable follow-up recommendations.

CONSULTATION/REFERRAL

A. Refer clients with drug and/or alcohol dependence to a community mental health center that has an outpatient alcohol/drug rehabilitation program or to a specialist in the community who deals frequently with substance abuse/dependence.

B. Planning a family meeting to confront the client is best done with the help of an experienced mental health professional.

C. Have referral numbers at close hand, so that the client's moment of motivation is not lost.

D. Refer family members of alcoholics/drug addicts to Al-Anon, Nar-Anon, Co-dependents Anonymous, or Adult Children of Alcoholics (ACOA) meetings.

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. Substance-dependent pregnant individuals frequently avoid early prenatal care for fear of identification and reprisal.

2. Cocaine use is associated with abruptio placenta and preterm labor. Consider drug screen for emergent admissions for clients in preterm labor and abruption.

3. Notify the hospital nursery personnel/neonatologist before delivery to closely monitor the newborn for withdrawal and seizure precautions.

4. Nicotine/smoking use is associated with intrauterine growth restriction, preterm delivery, and bleeding in pregnancy.

5. Nicotine-dependent pregnant females should be encouraged to stop smoking without pharmacologic treatment. The nicotine patch should be used during pregnancy only if the increased likelihood of smoking cessation, with its potential benefits, outweighs the risk of nicotine replacement, and potential concomitant smoking. Similar factors should be considered in lactating females.

6. Pregnant clients who use alcohol, tobacco, or drugs should always be classified as substance-dependent rather than substance-abusive.

B. Adults:

1. With females, tolerance can be established by asking the question: "How many drinks does it take to make you high?" More than two drinks indicates some tolerance.

2. In considering a diagnosis of alcohol dependence, consider the following diagnostic findings: hypertension; nonspecific EKG changes; cardiomyopathy; palpitations; increased mean cell volume; decreased red blood cell count; low platelet count; increased alanine aminotransferase (ALT), aspartate aminotransferase (AST), lactic dehydrogenase, gamma-glutamyl transpeptidase, alkaline phosphatase; type IV hyperlipoproteinemia; gout; and adult-onset diabetes mellitus.

C. Geriatrics:

1. Alcohol use disorders are often missed in this population due to reduced social and occupational functioning. Evaluate for signs of poor self-care, hygiene, and malnutrition.

2. At-risk drinking for persons older than 65 is defined as more than three drinks on one occasion or greater than seven drinks per week.

3. Pain medications and benzodiazepines, along with multiple medications for health problems, may contribute to a substance abuse problem.

D. Partners/family members:

1. For fear of retribution, the family may remain silent about the problem, even if accompanying the client. ▶

2. Some studies by corporate business show that, per capita, business spends more money on the care of family members of substance-dependent clients than on the employee.

3. Refer family members of alcoholics/drug addicts to Al-Anon, Nar-Anon, Co-dependents Anonymous, or ACOA meetings.

RESOURCES

National Institute on Drug Abuse (NIDA): www.drugabuse.gov

Substance Abuse and Mental Health Services Administration: www.samhsa.gov/treatment; offers free and downloadable publications for client education, treatment, prevention, and recovery; also includes professional and research topics and substance abuse treatment facility locator by state

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SUICIDE

DEFINITION

A. Suicide is defined as the intentional destruction of one's own life. It is the most critical consequence of mental illness and occurs in all diagnostic psychiatric categories. Knowing the risk factors for suicide and eliciting key clinical features that differentiate the truly suicidal client from the attention-seeker are of utmost importance. **Symptoms are often missed because they can be very subtle. Because there are important legal, social, and religious implications to suicide, the general healthcare practitioner should not attempt to treat these high-risk clients. This section focuses on identifying the suicidal client for immediate referral to a psychiatrist or psychiatric inpatient facility.**

INCIDENCE

A. The Rule of Sevens is helpful in the assessment of these clients:

1. One out of seven with recurrent depressive illness commit suicide.

2. 70% of suicides have depressive illness.

3. 70% of suicides see their primary care physician within 6 weeks of suicide.

4. Suicide is the **10th leading cause of death** in the United States; for young people between the ages of 10 and 34, it is the second leading cause of death and it is the fourth leading cause for ages 35 to 54.

B. The United States averages 13.9 suicides per 100,000 population annually. In 2019, over 47,000 people died from taking their own lives, not including those individuals who die as a result of fatal accidents due to impaired concentration and attention, and death due to illnesses that may be sequelae (e.g., alcohol abuse).

C. According to the World Health Organization, by the year 2020, depression will be the number two cause, worldwide, of individuals losing healthy years of their lives to depression. Estimates associate 16,000 suicides in the United States annually with depressive disorder. Fifteen percent of those hospitalized for major depressive disorder attempt suicide. Fifteen percent of clients with severe primary major depressive disorder of at least 1 month's duration eventually commit suicide. Firearm is the most common method of suicide (55.6%) for males, along with suffocation (29%). Among females, the most common method of suicide were firearm (31.4%), poisoning (30.0%), and suffocation (29.0%).

D. The rate of suicide in young adults has more than doubled since 1950. Little is known about midlife suicides compared with adolescent and older adult suicides. Midlife suicide rates tend to be highest among White males, although female suicide rates peak in midlife. Males exceed females in suicide completions but not in attempts. Whites are twice as likely as non-Whites to commit suicide, although in the 25- to 34-year age group their rates are equal. Rates for widowed, divorced, or separated individuals are higher than for those who are married. Rates are highest in Protestants, intermediate in Jews, and lowest in Catholics.

E. COVID-19: The COVID-19 pandemic increased many of the risk factors associated with suicidal behavior, such as financial or job stress, substance misuse, and adverse mental health conditions.

PATHOGENESIS

A. Recent studies confirm that some changes in the noradrenergic system along with reduced serotonin levels are associated with suicide. In recent studies independent of psychiatric diagnoses, one researcher identified a suicidality syndrome consisting of hopelessness, ruminative thinking, social withdrawal, and lack of activity as core symptoms.

B. Familial, genetic, early life loss experiences, and comorbid alcoholism may be causal factors. In adolescence, depression is the largest single risk factor for suicidal behavior, although family relationship difficulties make a significant independent contribution to this. Environmental stressors in the presence of psychiatric disorders may also be responsible for initiating the impulsive behavior leading to suicide. The risk of suicidal behaviors is higher in those with mental disorders than in those with mood disorders.

PREDISPOSING FACTORS

A. "SAD PERSONAS".

1. S = sex.
2. A = age.
3. D = depression.
4. P = previous attempts.
5. E = ethanol abuse.
6. R = rational thinking loss.
7. S = social support loss.
8. O = organized plan.
9. N = no spouse.
10. A = availability of lethal means.
11. S = sickness.

B. Also consider sex, race and ethnicity, medications, and other medical conditions.

COMMON COMPLAINTS

A. Overt or indirect suicide talk or threats: "You won't be bothered by me much longer."

Any mention of dying or ending one's life must be taken seriously.

B. Depressed or anxious mood due to depression. ▶

Every depressed client must be assessed for suicide risk.

C. Significant recent loss such as spouse, job, or self-esteem.
D. Unexpected change in behavior, such as making a will, intense talks with friends, and giving away possessions.
E. Unexpected change in attitude, such as suddenly cheerful, angry, or withdrawn.
F. Atypical symptoms of depression in older adults such as impaired ability to communicate, intractable tinnitus, and feelings of helplessness.

OTHER SIGNS AND SYMPTOMS

A. Indications for hospitalization of suicidal clients: psychosis.
B. Intoxication with drugs or alcohol that cannot be evaluated and treated over a period of time in the ED.
C. No change in affect or symptoms despite the intervention of the physician, family, and friends.
D. Command hallucinations.
E. Lack of access to, or low availability of, outpatient resources.
F. Family exhaustion.
G. Escalating number of suicide attempts.
H. Uncertainty about the risk of suicide.
I. Severe psychic anxiety, anxious ruminations, and global insomnia are acute risk factors.

SUBJECTIVE DATA

A. Ascertain the client's intention. Ask why they want to die:
1. Asking the client about suicide does not give the client any ideas about suicide.
B. Determine whether the client has thought of a suicide plan. The more specific the plan, the more likely the act. A well-worked out, realistic, and potentially lethal plan suggests great risk.
C. Rule out the presence of psychiatric or organic factors such as psychotic depression, thought disorder, or sedative self-medication.
D. Determine whether the precipitating crisis is resolving satisfactorily to the client.
E. Take an "inventory of loss." Determine the losses the client has incurred in the last several months or years.
F. Review the client's plans for the future.
G. Determine whether the client thinks they are going to commit suicide.
H. Evaluate whether the client has a caring family or other support systems.

PHYSICAL EXAMINATION

A. Check pulse, respirations, blood pressure, and weight.
B. Inspect:
1. Observe overall appearance; note grooming, tone of voice, and conduct of client during communication.
2. Complete dermal examination. Check for physical evidence of suicidal behavior, such as wrist lacerations or rope burns around the neck. Look for signs of substance use.
C. Palpate:
1. Neck and thyroid; note the goiter.
2. Axilla and groin for lymphadenopathy (infectious etiology).
D. Auscultate:
1. Heart, lungs, and abdomen (as applies to physical complaints).
E. Neurologic examination:
1. Complete mental status review: Specific concern exists when client displays a flat affect when discussing thoughts or plans for suicide.
2. Assess for thought disorder: Is the client having command hallucinations telling them to harm or kill themselves? Is the client having delusions or a strong sense of "burdensomeness," thinking that others will be better off without them?
3. Is the client exhibiting obsession with taking their own life?

DIAGNOSTIC TESTS

A. Complete blood count (CBC) with differential.
B. Electrolytes, serum calcium, and phosphorus.
C. Thyroid profile.
D. Liver profile.
E. Blood urea nitrogen (BUN), creatinine.
F. Blood alcohol.
G. Urine drug screen.
H. EKG.
I. Rapid plasma reagin (RPR).
J. CT and MRI scans.
K. Perform the Mini-Mental State Examination to rule out dementia/delirium.
L. Perform suicide assessment using an assessment tool such as the Suicide Behavior Questionnaire-Revised (SBQ-R), available at www.integration.samhsa.gov/images/ res/SBQ.pdf.

DIFFERENTIAL DIAGNOSES

A. Mood disorder due to another medical condition.
B. Adjustment disorder with depressed mood.
C. Personality disorders.
D. Psychotic disorder.
E. Alcoholism and drug abuse/dependence.
F. Dementia/delirium.
G. Side effect of medication (antidepressants, antipsychotics).

PLAN

A. General interventions:
1. If the client is suicidal, refer immediately. Make sure there is someone with the client at all times.
2. If the client came alone, call a family member or friend to accompany the client to the hospital ED or treatment center.
3. If you are fearful the client will try to escape or leave unaccompanied, escort the client to the hospital ED where commitment papers for involuntary hospital admission can be completed.

Be sure to advise the hospital staff of your concerns regarding the client's suicidal status.

B. Client teaching:
1. Educate client and family regarding treatment with medication for depression. Discuss benefits/risks of medication and side effects.
2. Encourage the client to enroll in counseling with a psychologist/therapist to discuss current problems/needs.
3. Determine if social services need to be contacted for client for support services.
4. Provide local resources for counseling and social services as appropriate.
5. If client is not hospitalized, make sure that the family or friends are aware of the client's status and that they ▶

have someone to talk to and monitor their condition until the next office appointment.

C. Pharmaceutical therapy:

1. Clients with suicide potential should never be given more than 1 g or 1 week's supply of tricyclic antidepressants (TCAs).

2. Documentation is critical. Make sure all statements are recorded and the decision-making process is followed.

3. See this chapter for sections on depression and/or bipolar disorder for complete pharmacologic interventions for specific mood disorders.

4. Antidepressants such as selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), and antianxiety medications can help to reduce symptoms of depression and severe anxiety, which may help the client to feel less suicidal.

5. Clozapine is a Food Drug Administration (FDA)-approved medication for reduction of suicidal behavior in clients with schizophrenia.

6. Quetiapine and lithium have been shown to prevent suicide in clients with bipolar depression.

FOLLOW-UP

A. After emergency admission for suicidal ideation or threats, clients should be closely observed, especially in the first year after the serious suicide attempt.

B. With each visit, question the client regarding suicidal ideation or a plan (see section "Subjective Data" for important questions to ask).

CONSULTATION/REFERRAL

A. Consult with the client's psychiatric practitioner. Obtain a release of information from the client.

B. Be sure the client continues to follow up with psychiatric counseling and medication management (see "Depression" section of this chapter).

INDIVIDUAL CONSIDERATIONS

A. Pregnancy:

1. A female with a history of depression or previous postpartum depression is at high risk for postpartum depression (recurrent; see "Postpartum Depression" section in Chapter 16, "Obstetrics Guidelines").

B. Geriatrics:

1. White males and age older than 80 have the highest rate of suicide of any age group in the United States.

2. Older persons use the usual means for suicide as well as a slower plan, including not eating, stopping prescription drugs or overmedicating, increasing alcohol intake, and refusing treatment.

3. Older adult population is more susceptible to the adverse effects of medications.

4. Antidepressants should be started at the lowest doses and slowly increased for older adults. The SSRIs are considered the first line of antidepressant therapy in older adults population (see the "Depression" section of this chapter).

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VIOLENCE AGAINST THE OLDER ADULT

DEFINITION

A. Abuse of older individuals, defined as older than age 65, is associated with loss of functional capacity, depression, cognitive impairment, increased morbidity, and mortality. Perpetrators include partners, family members (of all ages), as well as strangers. There are several types of maltreatment in this population:

1. Physical abuse: willful unnecessary restraint, the infliction of physical pain or injury.

2. Sexual abuse: nonconsensual sexual contact.

3. Psychological abuse: infliction of emotional harm, bullying, ridicule, verbal abuse, terrorizing, and threatening to place in a long-term facility until the elder submits.

4. Neglect: failing to provide for needs and protection of a vulnerable adult.

5. Self-neglect (subset of neglect): malnutrition, dehydration, lack of personal hygiene, listlessness, hoarding.

6. Abandonment: desertion.

7. Financial exploitation: misappropriation of resources, utilities turned off for nonpayment, essential purchases including food or medicine not made, discrepancies in personal bookkeeping including unexplained credit card activities, recent changes in property titles, deeds, and refinanced mortgages.

8. Healthcare fraud and abuse: not providing care, but charging for services; overmedicating or undermedicating.

INCIDENCE

A. In 2011, the population age 65 and older was estimated to be 41.4 million. It is estimated that 1 in every 10 people in the United States is an older person (60).

B. The exact incidence of elder abuse, neglect, exploitation, and self-neglect is unknown; however, it is believed to be common. The incidence is underreported due to the reluctance to report abuse, fear of implicating family members, and fear of being removed from the home.

C. Abuse is not uncommon in the institutional setting.

D. The highest rate of abuse is among older adult females older than age 80, with the abuser being the spouse or adult child. In the case of cognitive impairment, the victim may not remember or recognize abuse.

PATHOGENESIS

A. Maltreatment of vulnerable adults occurs by people who have an ongoing relationship with the older person when there is an expectation of responsibility: sons/daughters, spouses/intimate partners, other family members such as grandchildren, and others, including paid and unpaid caregivers. There are several identifying psychopathologies in the abuser:

1. Physical frailty and mental impairment of the victim plays an indirect role. The victim may have a decreased ability to defend or escape.

2. Caregiver stressors from caring for older adult client, including the client's physical and verbal demands. Psychosocial factors of the caregiver, mental illness, and alcohol or drug abuse contribute.

3. The child who was once abused may continue the cycle of violence transferred to the parent. ▶

PREDISPOSING FACTORS

A. Age: 60 years and older.
B. Institutionalized.
C. Cognitive impairment/diminished capacity.
D. Decreased capacity for performing activities of daily living (ADLs).
  1. Feeding themselves.
  2. Bathing and dressing themselves.
  3. Going to the toilet and performing hygiene themselves.
E. Decreased capacity performing instrumental activities of daily living (IADLs):
  1. Ability to prepare meals.
  2. Ability to do household chores.
  3. Ability to use the telephone.
  4. Ability to manage personal finances.
F. Females suffer a higher incidence of physical/sexual abuse.
G. Male sex is associated with self-neglect associated with impaired ADLs and IADLs.
H. Family stressors involving the caretaker.

COMMON COMPLAINTS

A. Depression.
B. Falls.
C. History of hip fracture.
D. Pressure ulcers.
E. Bruises, lacerations, and burns.

OTHER SIGNS AND SYMPTOMS

A. Abrasions, lacerations, or bruises that are seen in unusual locations or poorly explained may be a sign of physical abuse.
B. Indications of healing spiral fractures on x-ray.
C. Poor nutrition: lack of resources/transportation to obtain food; caregiver not providing adequate nutrition/withholding food and/or dehydration.
D. Multiple hospitalizations.
E. Recurrent urinary tract infections.
F. Noncompliance: may not be able to pay for medications; medications may be withheld or even given in excess by the caregiver.
G. Complaints of sexual abuse:
  1. Pain or soreness in the genital area.
  2. Bruises or lacerations on the perineum/rectum.
  3. Vaginal or rectal bleeding.
  4. Extremely upset when bathed, changed, or examined.
H. Traumatic tooth and/or hair loss.
I. Sedation from overmedicating.
J. Changes in personality.
K. Frequent unexplained injuries.

SUBJECTIVE DATA

A. The caregiver often refuses to leave the client alone and may answer questions for the client.
B. The caregiver has a different explanation of the injury.
C. Ask the client directly about abuse, neglect, or exploitation:
  1. Has anyone at home threatened or ever hurt you?
  2. Are you afraid of anyone at home?
  3. Are you left alone for long periods of time?
  4. Who cooks your meals? How often and what amounts of food do you eat?
  5. Who handles your financial business? Have you signed any documents that you didn't understand?
D. Assess the client's living arrangements. Has the client ever told family or friends, called hotlines, or attempted to leave the caregiver?

PHYSICAL EXAMINATION

A. Assessment:
  1. Observation: If abuse is suspected, enforce the need to do the physical examination in private. Do a full body examination:
    a. Forensic exams need thorough documentation of injuries:
      i. Use color photographs before any treatment is started.
      ii. Make at least one full body photograph and a facial photograph.
      iii. Make closeup photographs of all injuries.
      iv. Use a ruler to identify/document the size of the injuries.
      v. Documentation on the back of the photographs should include the client's name, date, photographer's name, as well as any witness to the examination. The photographer should also sign each photograph.
      vi. Use direct quotes of the victim's history.
  2. Check blood pressure, pulse, respirations, and weight.
  3. General observation: Observe for depression, withdrawn, flat affect, fearfulness, poor eye contact, inappropriate dress, and signs of malnutrition.
  4. Observe for poor hygiene, presence of urine and feces, matted or lice-infected hair, odors, dirty nails and skin, and soiled clothing.
  5. Assess cognitive abilities, depression, and functional ability of ADLs and IADLs.
B. Inspect:
  1. Dermal examination for signs of burns, tears, lacerations, impression marks, and bruises in different stages of healing. Frequent areas of the body involved are the neck, arms, and/or legs. Evaluate for the presence of decubitus/pressure ulcers. Signs of dehydration include dry fragile skin, dry sore mouth, and mental confusion.
  2. Oral examination for poor oral hygiene, absence of dentures, and dry mucus membranes.
  3. Evaluate breasts and genitals for lacerations, and hematomas of the vagina or labia.
C. Auscultate:
  1. Auscultate all lung fields.
  2. Auscultate heart sounds.
  3. Auscultate bowel sounds in all four quadrants of the abdomen.
D. Palpate:
  1. Evaluate for dislocation, fractures, sprains, and contusions to the wrists, forearms, and shoulders.
E. Percuss:
  1. Abdomen and chest (if indicated).
F. Genital/rectal examination:
  1. Evaluate genitals/anal area for redness, swelling, bruising, hematomas, abrasions, or lacerations.
  2. Evaluate for evidence of sperm.
  3. Evaluate for presence of foreign bodies.

DIAGNOSTIC TESTS

A. Diagnostic tests and x-rays are ordered dependent on the type of presenting complaints.
B. Obtain a CT for evaluation of injuries to the head and assault to the face, neck, or head. A CT or Doppler may be ordered for abdominal injuries.
C. Order laboratory testing to evaluate dehydration, malnutrition, electrolyte imbalance, and medication/substance abuse:

1. Complete blood count (CBC).
2. Chemistry-7.
3. Urinalysis.
4. Calcium, magnesium, and phosphorus.
5. Drug/alcohol screen.
6. Serum levels for relevant medications.

D. Obtain DNA samples if sexual abuse is present.

DIFFERENTIAL DIAGNOSES

A. Elder abuse.
B. Depression.
C. Abdominal trauma.
D. Sexual assault.
E. Gait disturbance/fall.
F. Pathologic fracture.
G. Epidural/subdural hematoma.

PLAN

A. General interventions:
1. Provide a safe environment.
2. Clearly document the history, physical findings, and interventions. Document specific details about the abuse.
3. Determine the perpetrator(s).
4. Evaluate the need for ED/hospital admission.
5. Determine and plan a safety plan. Include a safe place for the client.
6. Discuss strategies to reduce harm and protect the client from the potential abuse.
7. Provide emergency phone numbers of family, friends, community resources, police, and medication providers/ED.
8. Establish routine primary care and follow-up appointments to determine safety.

B. Client teaching:
1. Reinforce that abuse/neglect is not the victim's fault. Older adult abuse is very common, and the aged do not deserve to be abused.
2. Help is available.

C. Pharmaceutical therapy:
1. Prescriptions are related to physical injuries.
2. Recommend treatment for sexually transmitted infections in the oral anal genital areas.

FOLLOW-UP

The Department of Health and Human Services National Center on Elder Abuse Administration on Aging maintains a State-by-State Resource site that includes directories, helplines, hotlines, referral sources, state government agencies and laws, and regulations related to elder abuse; it is located at ncea.acl.gov/resources/state.html.

A. Develop a follow-up plan. All states have legislation protecting against abuse, neglect, and exploitation of the older population.
B. Know if your state has mandatory requirements to report any suspicion of elder mistreatment.
C. Abuse of a disabled person must be reported to the Disabled Person Protection Commission.
D. Know if your state has additional regulations related to self-neglect. Contact adult protective services or law enforcement agencies.
E. The American Medical Association and the American Academy of Neurology advise screening persons 65 years and older for abuse.

CONSULTATION/REFERRAL

A. Arrange a social work consultation to coordinate an in-home geriatric assessment visit.
B. Facilitate referrals to a shelter, counseling, and legal services.
C. Contact a sexual assault nurse examiner (SANE) qualified healthcare provider, if indicated.
D. Refer to the community area agency for assistance.
E. Refer for a psychiatric consultation, if indicated.
F. Refer for a neurologic or neurosurgical consultation for intracranial injuries or focal neurologic findings.
G. Refer for an orthopedic consultation for fractures.

INDIVIDUAL CONSIDERATIONS

A. Geriatrics:
1. This population is at high risk for mental and physical abuse. Careful evaluation and treatment for all clients is imperative.
2. Clients diagnosed with dementia and other cognitive disabilities require careful assessment for mental and physical abuse.

RESOURCES

American Association of Retired Persons (AARP): www.aarp.org
Clearinghouse on Abuse and Neglect of the Elderly (CANE): www.cane.udel.edu
Help Hotline for suspected elder abuse, neglect, or exploitation: 1-800-677-1116
National Adult Protective Services Association (NAPSA): www.napsa-now.org
National Center on Elder Abuse Administration on Aging (NCEA): www.ncea.aoa.gov

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CLIENT TEACHING GUIDE

ADHD: COPING STRATEGIES FOR TEENS AND ADULTS

PROBLEM Attention deficit hyperactivity disorder (ADHD) is one of the most common neurodevelopmental disorders of childhood. It is usually first diagnosed in childhood and often lasts into adulthood. People with ADHD often have difficulty paying attention, they have a short attention span, and frequently gets in trouble because of behavior issues. The client may show signs of hyperactivity, difficulty with concentrating, reacting without thinking first, and may also have anger issues.

CAUSE The cause of ADHD is unknown. However, it may be hereditary.

TREATMENT PLAN/PREVENTION CARE

A. Identify coping skills. Identify what things help you to control your behavior and prevent outbursts.
B. Counseling and therapy are commonly encouraged to help you learn new coping skills to help with your problem. Make sure you keep all appointments with your counselor/therapist.
C. Make sure to clarify all assignments and tasks.
D. Keep simple lists of your chores for the day.
E. Attach reminder notes to logical items; for example, put a self-adhesive note on your bathroom mirror to remind yourself to do something.
F. Organize your things in a logical way. For example, put your medication bottle with your toothbrush so you will be sure to take it in the morning.
G. Establish a daily routine and stick to it.
H. Keep your environment as quiet and peaceful as possible. Seek out work and study sites that are quiet and peaceful.
I. Be good to yourself: eat well, get enough rest, and exercise.
J. Take your medications as prescribed. If you feel that your medication is not improving your behavior, notify your parent and/or care provider so this can be evaluated.
K. Limit the amount of daily screen time including TVs, computers, phones, and other electronics.

RESOURCES Children and Adults With Attention Deficit Hyperactivity Disorder (CHADD): www.chadd.org

The National Attention Deficit Disorder Association: www.add.org

National Resource Center for ADHD: www.help4adhd.org What is ADHD: www.cdc.gov

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CLIENT TEACHING GUIDE

ALCOHOL AND DRUG DEPENDENCE

PROBLEM Dependence on alcohol or drugs is a disease with signs and symptoms and a progressive course that requires treatment, just like diabetes or cancer.

CAUSES Factors that contribute to dependence on alcohol and drugs may include inherited traits, the environment, occupation, socioeconomic status, family and upbringing, personality, life stress, and emotional stress. These factors vary among individuals, but no one factor can account entirely for the risk.

PREVENTION The only way to prevent alcohol and/or drug abuse and dependence is not to start. Warning signs for needing help are not always dramatic. The following questions can help identify dependence.

A. Are you or someone you know experiencing any of the following:

1. Steadily drinking or using more at a time or more often?
2. Setting limits on how much, how often, when, or where you will drink or use other drugs, and repeatedly violating your limits?
3. Keeping a large supply on hand or becoming concerned when you run low?
4. Drinking or using other drugs before going to activities where they would not be available (e.g., class or work)?
5. Drinking or using other drugs alone? Drinking or using other drugs every day?
6. Spending more money than you can afford on alcohol or other drugs?
7. Doing or saying things when you are under the influence that you regret later or do not remember?
8. Lying to friends and family about your drinking or other drug use?
9. Becoming accident prone when you are under the influence (spilling, dropping, or breaking things)?
10. Regularly hung over the morning after drinking?
11. Worrying about your drinking or other drug use?
12. Having work or school problems, such as tardiness or absenteeism?
13. Reducing contact with friends, or experiencing increasing problems with important relationships?

B. If you answered "yes" to any of these questions, it suggests your drinking or drug use may be a problem.

TREATMENT PLAN

A. There are no "quick cures" for alcohol and drug dependence, but early intervention is of utmost importance because it helps avoid the harmful effects of long-term alcohol or other drug use.

B. Your healthcare provider will be suggesting a plan of action for you to consider. You are strongly encouraged to follow the recommendations.

C. Do not hang around your drinking/drug-using friends. Instead, go to new areas, play new sports, and develop new hobbies. Join groups to make new friends and find other interests that you enjoy sharing with other people.

D. Talk to your provider about seeking professional rehab treatment. Inpatient and outpatient facilities are available to assist you in your goal of becoming alcohol/drug free.

E. Ask about a support group in your area. Resources are available from your provider and you may also search on the internet for local support groups to meet your needs.

Activity: Daily exercise is helpful in alleviating the craving for alcohol and drugs. Walking daily and increasing your tolerance for distance are recommended.

Diet:

A. Avoid caffeine and nicotine, if possible.

B. Try to eat three meals a day and three snacks.

C. You may be given vitamin supplements to help restore the vitamins and minerals that have been depleted as a result of your alcohol or drug dependence.

Medications: Medications may or may not be prescribed, depending on your need. If you are prescribed any medications, take them exactly as directed by your healthcare provider. If you have any side effects or problems, notify your provider. Medications are intended to help curb the cravings, and if you are having withdrawal symptoms you will need to notify your provider or go to the ED.

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CLIENT TEACHING GUIDE

ALCOHOL AND DRUG DEPENDENCE (*CONTINUED*)

You Have Been Prescribed: _____

Vitamins/Minerals: _____

You Need to Take: _____

You Need to Notify the Office if You Have:

A. Severe craving and the urge to use alcohol or drugs.
B. Any thoughts of hurting yourself or others.
C. Impulsive feelings, like you might do something you will later regret.

Phone: _____

RESOURCES Al-Anon Family: 888-4AL-ANON (888-425-2666) and www.al-anon.alateen.org

Alcohol Help Line 24 hours a day, 7 days a week: 800-ALCOHOL and www.adcare.com

American Council for Drug Education: www.health.gov/nhic

Cocaine Anonymous: www.ca.org. State by state has local contact number to call on the website.

Families Anonymous: 800-736-9805 and familiesanonymous.org

Mothers Against Drunk Driving (MADD) 24 hours a day, 7 days a week: 877-ASK-MADD and www.madd.org

New Life (Women for Sobriety): 215-536-8026 and www.womenforsobriety.org

Phoenix House: 1-888-671-9392

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CLIENT TEACHING GUIDE

GRIEF

PROBLEM Normal grief resolution may take from 6 months up to as long as 2 years.

CAUSES Grief can follow the death of a loved one, but grief also follows other losses such as a loss of independence, loss of affection, or loss of body parts after an accident.

TREATMENT PLAN It is important that you talk about your loss and how it affects you. The more you are able to talk about your feelings related to the loss you are experiencing, the more you will be able to work through your feelings.

You may be encouraged to seek supportive therapy in the form of divorce groups, loss groups, or groups dealing specifically with death and dying.

Activity: Moderate exercise, such as daily walking, is encouraged. Vigorous, prolonged exercise, however, may precipitate anxiety attacks.

Diet: Avoid caffeinated beverages, including coffee, tea, and carbonated colas that do not specify "decaffeinated." Try to eat healthy food and avoid overeating or skipping meals.

Medications: Medications for loss and grief are usually avoided at first because the use of drugs can prolong the time it takes to work through your grief. However, your healthcare provider will discuss medications in more depth. If you are having signs of depression or think you may need treatment with medication, contact your healthcare provider.

You Have Been Prescribed: _____

You Need to Take: _____

Do not stop antidepressants quickly. Talk to your healthcare provider if you want to discontinue them.

You Need to Notify the Office If:

A. You feel like you cannot stop crying.
B. You are not eating and are losing weight.
C. You feel like you have no one to turn to and talk about your feelings.
D. You have difficulty sleeping after several weeks.
E. You have thoughts of hurting yourself or others.
F. You do not think you are getting better on the prescribed medication.

Phone: _____

Local Support Group: _____

SUGGESTED READING Smith, H. I. (2011). *A Decembered grief: Living with loss while others are celebrating*. Beacon Hill Press. (Original work published 1999.)

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CLIENT TEACHING GUIDE

NICOTINE DEPENDENCE

PROBLEM Cigarette smoking is one of the most **preventable causes of death and disability in the United States.** Other forms of nicotine include chewing tobacco and pipe tobacco, which can be just as harmful. Risks of lip, tongue, mouth, and throat cancer are associated with the use of nicotine. Smoking may cause bleeding in pregnancy and may be responsible for the baby not growing well. It is well documented that infants and children who are exposed to long-term, secondhand smoke inhalation are at increased risk of **sudden infant death syndrome** and frequent **ear infections and chronic illnesses.**

Nicotine is addictive; therefore, stopping smoking is difficult.

The effectiveness and safety of e-cigarettes as a smoking cessation aid is uncertain at present.

CAUSE Seeing your parents smoke and using tobacco may be one of the reasons that caused you to start using tobacco. Peer pressure is a big reason why you may have started smoking as a teenager. You may also feel nervous and use smoking for its instant calming effect.

REASONS TO QUIT

A. Quitting tobacco will add years to your life. **It is never too late to quit smoking.**
B. You will have healthier lungs, which will decrease your risk of developing cancer and having a heart attack or stroke.
C. You will also have more energy and feel better physically and mentally.
D. Smoking cessation will also decrease the secondhand-smoke exposure around your family and friends, which will make them healthier, too.
E. Secondhand smoke causes asthma attacks and other health problems.
F. Some insurance plans are more expensive if you smoke.
G. You will save a lot of money by not buying cigarettes.
H. Do not quit for yourself: Quit for someone you love.
I. You will have fewer wrinkles.
J. Other treatment is available if you use smoking to help control nervousness. Please talk to your healthcare provider if you feel this is the reason you smoke.

TREATMENT PLAN

A. Set a quit date within 2 to 4 weeks and get information/treatment from your provider.
B. Throw away all cigarettes, matches, lighters, and ashtrays in your home, car, and workplace.
C. Make smoking very inconvenient.
D. Ask your family/friends for support and encouragement to help you stop.
E. Stay in nonsmoking environments and avoid friends/family members who smoke.
F. If you get the urge to smoke, take deep cleansing breaths and try to occupy your time with something else, like chewing gum.
G. Leave the table and change the smoking routine you used to have, such as smoking with your coffee after meals.
H. Reward yourself often for staying smoke-free.
I. It is not unusual that you will go back to smoking; it is difficult to quit. **Smoking even one fewer cigarette counts.**
J. It may take several times to finally quit.
K. Use an alternative method to help during times of extreme stress (singing, music, prayer, reciting a favorite verse, taking deep abdominal breaths [five times in over 1 minute]).

Activity:

A. Exercise daily to help alleviate the craving for nicotine.
B. Avoid caffeine if possible.
C. Chew gum or hard candy when you crave a cigarette.
D. Eat celery sticks or carrots in place of a cigarette.
E. Drink a lot of water and other fluids to keep hydrated.

(*continued*)

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CLIENT TEACHING GUIDE

NICOTINE DEPENDENCE (*CONTINUED*)

Medications:

A. Discuss options available with your healthcare provider to help you quit.
B. Medications are available to help you quit.
C. The nicotine patch, inhaler, and gum are available and may be right for you. Discuss these options with your healthcare provider since they are good steps to quitting.
D. Many of the stop-smoking medicines may be covered by your insurance.

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office if You Have:

A. Severe craving and the urge to smoke or chew tobacco even on medicines.
B. Feelings of impulsiveness, like you might do something you will later regret.
C. Started to smoke again after you have stepped down while using the nicotine patch.
D. Other: _____

Phone: _____

RESOURCES The American Cancer Society has some good tips on quitting smoking at www.cancer.org.

The American Lung Association also has good guides to help stop smoking at www.lung.org.

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CLIENT TEACHING GUIDE

SLEEP DISORDERS/INSOMNIA

PROBLEM Continued loss of sleep over a long period of time (several days to weeks) can produce a decrease in daytime awareness and functioning, as well as work and driving impairment.

CAUSE Most sleep difficulties are related to situational problems and are best treated without medications. Medical illness may also predispose you to a sleeping disorder.

PREVENTION

A. Practice good "sleep hygiene." Here are a few tips:
  1. Try to get to bed at the same time every night (even on weekends).
  2. Plan for 8 hours of sleep.
  3. Maintain the same waking time every morning (even on weekends).
  4. Reserve the bedroom for sleeping. Do not do your work/homework in the bed. Do not watch TV in the bedroom, unless you sit in a chair. Do not read in bed.
  5. Put your children in their own bed/bedroom.
  6. Try a small bedtime snack.
  7. Do not drink any liquids for at least 1 hour before going to bed. This helps prevent getting up to go to the bathroom during the night.
  8. Review all of your prescription medications, over-the-counter drugs, and herbal products with your healthcare provider to evaluate if the drugs are making you stay awake.
  9. Avoid alcohol and tobacco.

B. If you do not get a restful night's sleep, awaken yourself snoring, or your partner complains of your snoring, gasping, or stopping breathing, you may need to have a sleep study.

TREATMENT PLAN

A. Develop a "sleeping ritual." Wear your favorite pajamas and use your favorite pillows.
B. Keep the room dark and quiet. Try not to use a nightlight.
C. Run a small fan for background noise.
D. Arise promptly in the morning.
E. Avoid sleeping medication if possible.
F. Try some relaxation exercises, like yoga, self-hypnosis, or meditation. There are many books on these subjects that will assist you in developing good sleep hygiene. (Try your local library or bookstore.)
G. Keep a sleep diary to help identify the causes of your inability to sleep.
H. Discuss snoring and your partner's concerns about your breathing and the need for a sleep study.

Activity: Regular exercise during the day helps. (Do not exercise vigorously 1 hour before your bedtime—this could keep you awake.)

Relaxation techniques such as progressive relaxation, biofeedback, self-hypnosis, and meditation are helpful when done before bedtime. Avoid vigorous mental activities late in the evening.

Diet:
A. Avoid caffeinated drinks, including coffee, tea, and soda.
B. Avoid large meals in the evening.
C. Eat a nutritious balanced diet.
D. Limit your liquids to early evening. Do not drink water after 8 p.m.

Medications: Discuss the need for "short-term" medications to help you sleep.

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CLIENT TEACHING GUIDE

SLEEP DISORDERS/INSOMNIA (*CONTINUED*)

You Have Been Prescribed: _____

You Need to Take: _____

You Need to Notify the Office if You Have:

A. No relief of your symptoms and you are following the previous recommendations.
B. No relief of your symptoms and you are taking your medications as directed.
C. Feelings of depression or thoughts of hurting yourself or others.
D. Noticeable physical symptoms that were not mentioned in your office visit or any new symptoms that you are concerned about.

Phone: _____

RESOURCES National Institutes of Health: https://medlineplus.gov/insomnia.html

National Sleep Foundation: www.sleepfoundation.org

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SECTION II

PROCEDURES

- Bimanual Examination: Cervical Evaluation During Pregnancy
- Canalith Repositioning (Epley) Procedure for Vertigo
- Clock-Draw Test
- Cystometry
- Establishing the Estimated Date of Delivery
- Evaluation of Sprains
- Hernia Reduction (Inguinal/Groin)
- Inserting an Oral Airway
- Intrauterine Device Insertion
- Neurologic Examination
- Pessary Insertion and Management
- Prostatic Massage Technique: Two-Glass Test
- Rectal Prolapse Reduction
- Removal of a Foreign Body From the Nose
- Removal of a Tick
- Trichloroacetic Acid/Podophyllin Therapy
- Wet Mount/Cervical Cultures

The contributions of Rhonda Arthur, Kathleen Bradbury-Golas, Cheryl A. Glass, and Amy Hull to the procedures in prior editions are acknowledged here.

BIMANUAL EXAMINATION: CERVICAL EVALUATION DURING PREGNANCY

DESCRIPTION

A. The bimanual examination is a digital evaluation of the cervix with the index and middle fingers of the examiner's hand.

INDICATIONS

A. To assess cervical dilation, effacement, presentation of the fetus, and position and station of fetal presenting part.

B. The cervix may be assigned a Bishop score to evaluate cervical change and to determine difficulty/ease with induction of labor. There is an inherent subjective difference between examiners; however, the Bishop score can be used to evaluate preterm labor cervical changes over time (Table II.1).

PRECAUTIONS

A. Bimanual exam should never be performed when there is suspicion of or history of documented placenta previa. Vaginal bleeding in the second or third trimester of pregnancy should be treated as placenta previa until this is ruled out.

B. Suspicion of ruptured membranes with preterm contractions:

1. First perform sterile speculum exam to assess for rupture.
2. Then, if membranes are intact, proceed with bimanual exam.

C. If membranes are ruptured in the absence of active labor, avoid bimanual examination until frequent, painful uterine contractions are present.

EQUIPMENT REQUIRED

A. Gloves (nonsterile if membranes are intact; sterile if suspicion of or documented ruptured membranes).

B. Water-soluble lubricant (sterile lubricant required if ruptured membranes).

C. Examination table or bed.

PROCEDURE

A. Stand at the foot of the examination table or sit near the foot of the bed and face the client.

B. Place the client in the lithotomy position, or have the client draw their knees up and allow the knees to drop to opposite sides.

C. Suggest relaxation techniques to reduce discomfort. If the client is in active labor, the cervix will be tender.

D. Place lubricant on the index and middle fingers of the examining hand.

E. Inform the client of each step prior to performance: Touch the inner thigh first, then touch the posterior portion of the introitus and introduce gloved fingers into the vagina. Encourage the client to relax pelvic muscles (a deep breath will often encourage relaxation).

F. Palpate the cervix and lower uterine segment evaluating (Figure II.1):

1. Dilatation.
2. Effacement.
3. Station.
4. Consistency (softness).
5. Anterior versus midposition versus posterior placement of the cervix.
6. In cases of preterm uterine contractions, it is helpful to assess the development of the lower uterine segment. A well-developed lower uterine segment (even in the absence of cervical dilation or effacement) is often a sign of preterm labor.
7. Note fetal presentation and station of the presenting part. Use ischial spines as landmarks to determine fetal descent through the pelvis.
8. If the fetus is sufficiently descended into the pelvis, determine the position of the presenting part.
9. If present, note the bag of amniotic membranes. A bulging bag, known as a "forebag," may be present, even in the presence of documented ruptured membranes.

G. Following the exam, note if blood or vaginal discharge is on the gloves.

TABLE II.1 BISHOP SCORING

Score	0	1	2	3
Dilation	0	1–2	3–4	5–6
Effacement (%)	0%–30%	40%–50%	60%–70%	80% or more
Station	–3	–2	–1 to 0	+1 to +2
Consistency	Firm	Medium	Soft	—
Position	Posterior	Mid	Anterior	—

Total score: 0 to 5, difficult induction; >6, easy induction.

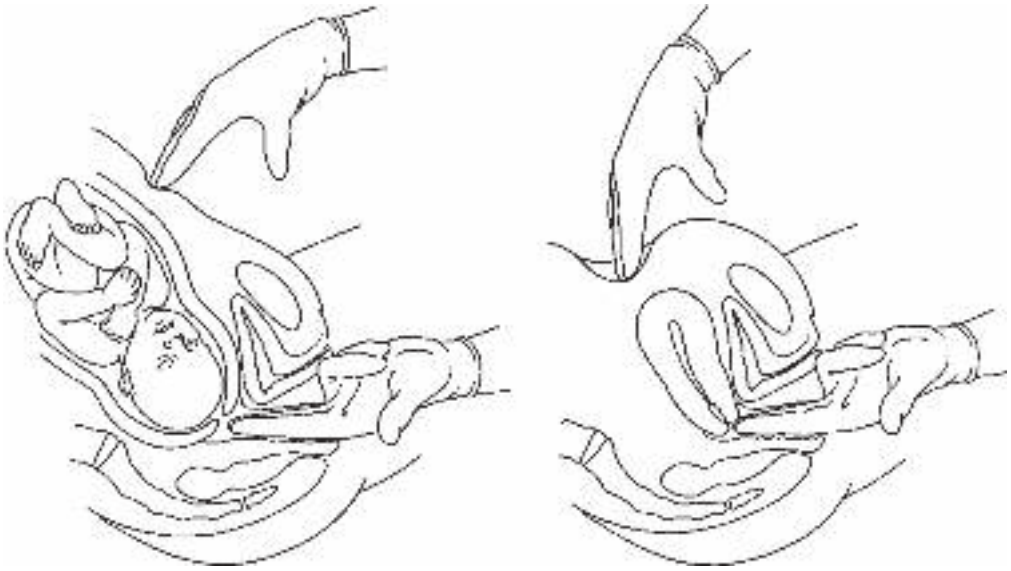


FIGURE II.1 Bimanual examination.

CANALITH REPOSITIONING (EPLEY) PROCEDURE FOR VERTIGO

DESCRIPTION

A. Benign paroxysmal positional vertigo (BPPV) occurs with changing position, and rapid head movement causes imbalance, disorientation, and nausea. Vertigo develops when there is a disturbance of the crystals that are normally evenly distributed in the fluid-filled semicircular canal and they become clumped together. Often the imbalance can be corrected by performing canalith repositioning (Epley maneuver) to redistribute the crystals in the vestibule.

INDICATIONS

A. BPPV.

CONTRAINDICATIONS

A. Testing should never be done in the case of head injury or suspected or confirmed cervical spine injury/dysfunction, severe kyphoscoliosis, a history of unstable carotid disease, or recent retinal detachment.

EQUIPMENT REQUIRED

A. Examination table.

B. Stool at the head of the table for the practitioner.

PROCEDURE

A. Explain the procedure (Figure II.2).

B. Have the client sit on the examination table (position 1):

1. Reposition the client to sitting in the center of the table.

2. The edge at the head of the table should be at the client's shoulder height (head will hang off the table).

3. Your position will be at the head of the examination table, and you use the stool for comfort while holding the client's head with your hands.

C. Rapidly lay the client down (position 2):

1. The client's head should be hanging over the table in a slightly downward tilt, supported by both of your hands.

2. The client should be instructed to keep their eyes open and try to focus on a stationary object on the wall throughout the positions on their back and sides.

3. The client's head is turned to the same side as the affected ear with the provider supporting the head:

a. If the client has vertigo affecting both inner ears, turn the client's head to the dominantly affected side.

b. As an alternative to just turning the head, the client may quickly turn and lie down on the affected side while you support the head with both of your hands.

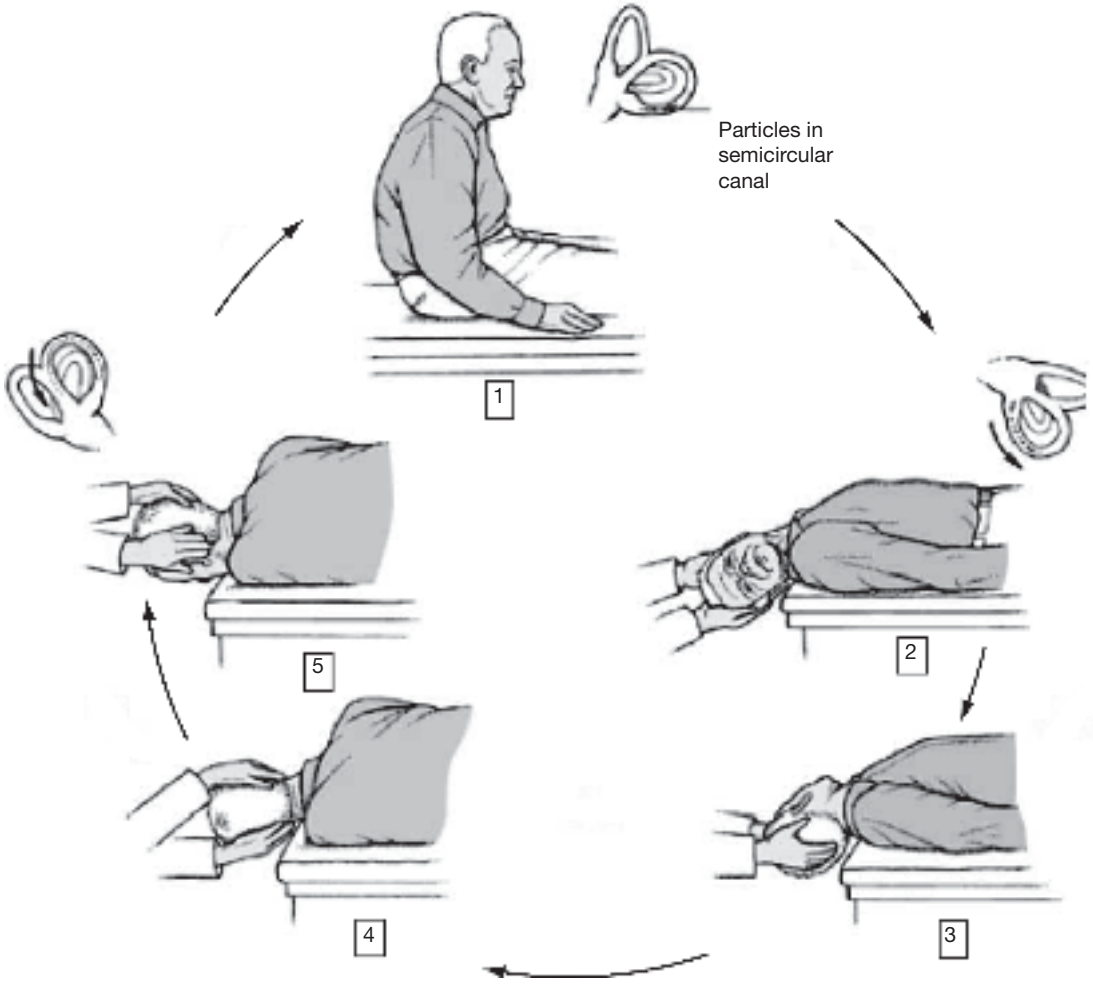


FIGURE II.2 Epley procedure for vertigo.

Source: From Lustig, L. R. (2018). Benign paroxysmal positional vertigo. In R. Porter (Ed.), *Merck manual professional version*. Merck Sharp & Dohme. https://www.merckmanuals.com/professional/ear,-nose,-and-throat-disorders/inner-ear-disorders/benign-paroxysmal-positional-vertigo. Copyright (2018) by Merck Sharp & Dohme Corp., a subsidiary of Merck & Co., Inc.

4. After a few minutes the client will be ready to be repositioned evidenced by their lack of nystagmus (rhythmic side-to-side eye movement) and confirmation that the dizziness has stabilized.

D. Turn the client's head in the opposite direction while you continue to support the head (position 3). Again, the head should be in a slightly downward tilt:

1. If the client was using the side-lying position, have them turn to their back.
2. Have the client continue to keep the eyes open and focus on a stationary object on the wall.
3. Again, the time lapse to repositioning is evidenced by the lack of nystagmus and subjective confirmation that the dizziness has stabilized/abated.

E. Ask the client to roll from their back to the opposite side (or from the back to the opposition side; position 4). The client's head should be parallel to the floor.

F. With the client side-lying (position 5), have them quickly turn only their face to the floor, while you are supporting the head.

G. After the client verbalizes that the dizziness has abated from side-lying (position 5):

1. The client is to swing the legs to the edge of the table and quickly sit up.
2. They will continue to feel vertigo at this point.

H. Assist the client off the examination table.

I. The client should be instructed to be in a sitting position or with the head no farther back than a 45° angle for the next 24 hours.

J. The Epley does not immediately stop the vertigo. The client may begin to feel better within a few days.

K. If the Epley procedure needs to be repeated, allow 1 week between attempts.

CLOCK-DRAW TEST

DESCRIPTION

A. The clock-draw test (CDT) is a quick screening tool assessing visuospatial and praxis abilities (may reflect both attention and executive dysfunction). The tool can be used by itself or as a complement to other screening tests. The test is a component of the Mini-Cog and also the "7-Minute Neurocognitive Screening Battery." It can be administered repeatedly over time to evaluate deterioration of function. The CDT evaluates:

1. Orientation.
2. Conceptualization of time.
3. Visual spatial organization.
4. Visual memory.
5. Auditory comprehension.
6. Numeric knowledge.
7. Concentration.
8. Frustration tolerance.

INDICATIONS

A. Evaluation of dementia.
B. Evaluation of delirium.
C. Evaluation of neurologic insult:
1. Head trauma.
2. Stroke.
3. Alcohol and drug.
D. Evaluation of psychiatric illness:
1. Schizophrenia.
2. Psychotic state.

ADMINISTERING THE CLOCK-DRAW TEST

A. A number of variations can be used in administering the CDT.
B. This is not a timed test. There is no limitation to administration time; the test generally takes 1 to 2 minutes. However, in the Mini-Cog test, if the clock is not completed within 3 minutes, the client should proceed with word recall.

GENERAL INFORMATION

A. Provide the client with an 8.5- by 11-in. sheet of paper with a predrawn clock and a pencil.
B. Instructions can be repeated if necessary.
C. Clients may use their nondominant hand.
D. Pages may be used for serial testing.
E. The ages for CDT testing are adults 18 to 64 and the elderly (age 65+ years).

SETUP

A. Equipment required includes a sheet of paper with a predrawn clock, a pen, and a chair/table for ease of drawing.
B. A clock should not be visible to the client during the CDT.

CLIENT INSTRUCTIONS

A. If using a blank page, instruct the client to do the following:
1. Draw a clock face.
2. Place all of the numbers on the clock.
3. Draw hands pointing to 10 minutes after 11.

B. If using a page with a predrawn clock, instruct the client to do the following:
1. Copy the clock on the top of the page onto the lower part of the same sheet of paper.

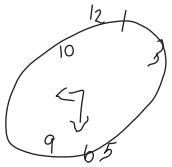
SCORING

Scoring consists of the evaluation of errors and/or distortions. There are several variations on scoring the CDT. The quickest scoring involves dividing the clock into four quadrants and counting the numbers in the correct quadrant. More complex assessment evaluates up to 20 traits or categorizes errors.

A. The placement of the arms of the clock is the most abstract feature and is useful in evaluating the early dementing process.
B. A normal clock suggests that multiple functions are intact and contributes to the assessment of the client's ability to continue independently. The following is a "normal" clock draw.



C. A grossly abnormal clock is an indicator of potential problems that require attention (see the following figures).





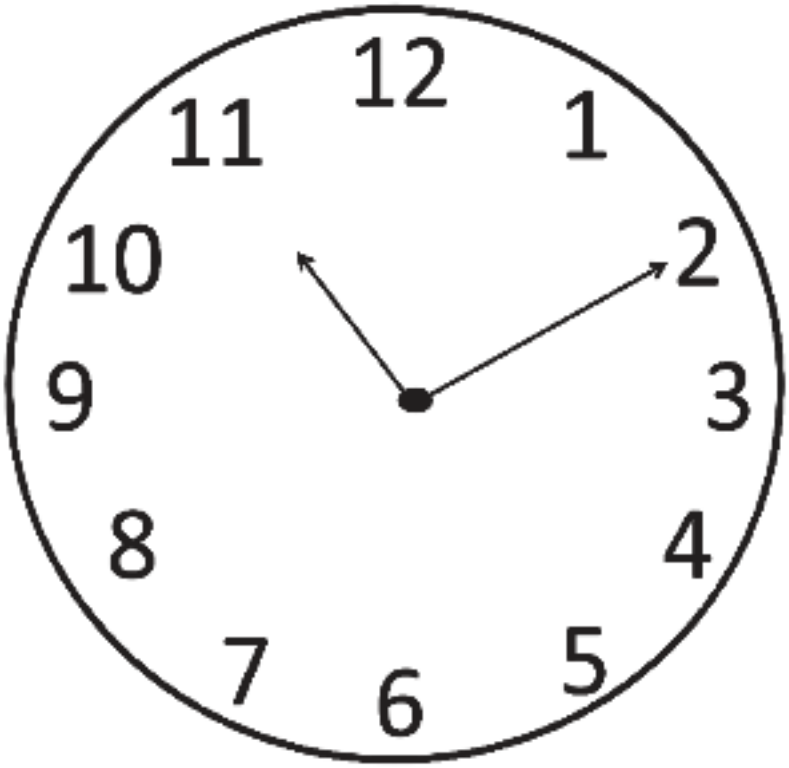
D. There are several scoring systems for evaluating the CDT results. The most basic involves awarding 1 point for each of the following:
1. 1 point for the clock circle.
2. 1 point for the numbers placed in the correct position.
3. 1 point for the clock-face numbers placed in their proper order around and inside the clock face.
4. 1 point for the presence of the two clock hands. 1 point for the correct time as instructed.

A normal score is 4 or 5 points.

Refusal to draw a clock is scored as abnormal.

Name: _____ Date: _____

INSTRUCTIONS: Copy this clock.



CYSTOMETRY

DESCRIPTION

A. Cystometry testing is an office procedure to determine bladder capacity, how full the bladder gets when the urge to urinate begins, postvoid residual (PVR), any fluid leak with stress maneuvers, or inhibited bladder capacity. Cystometry measures the intravesical bladder pressure during bladder filling. Often, cystometry is performed in conjunction with uroflowmetry.

INDICATIONS

A. Clients presenting with complaints of lower urinary tract symptoms including urinary incontinence, stress (leaking), overactive bladder (OAB), and urinary retention (inability to empty bladder) are candidates for cystometry testing.

PRIOR TO THE PROCEDURE

A. A thorough urologic/gynecologic history should be taken and a physical examination should be performed, including a digital rectal examination.

PRECAUTIONS

A. Rule out the presence of a urinary tract infection (UTI) as a cause of the incontinence prior to cystometry testing.

B. Use universal precautions. This is a sterile procedure and is contraindicated when infection or inflammation is present.

C. Risks of the procedure:

1. Discomfort.

2. Bleeding.

3. Infection.

4. Autonomic dysreflexia (AD) from bladder instrumentation and bladder distension:

a. Flushing.

b. Hypertension.

c. Reflex bradycardia.

d. Severe headache.

EQUIPMENT REQUIRED

A. Sterile straight catheter (12–14 French catheter).

B. Catheter insertion kit.

C. A 60-mL sterile syringe.

D. 1,000-mL sterile water (CO_2, saline, or contrast medium may also be used).

E. Sterile measurement container.

F. Urine collection device.

G. A vaginal or rectal pressure catheter or insertion of a second catheter with a manometer to measure pressures.

H. Light source for catheterization.

I. Gown and drape for the client.

PROCEDURE

A. Explain the procedure.

B. Obtain consent (if required).

C. Ask about any latex or Betadine allergies.

D. Throughout the procedure, ask the client to speak to their feeling the sensation of the bladder filling, any discomfort, when the urge to void is sensed, and bladder fullness. Sensations noted by the client are subject to the rate of bladder filling and temperature of the fluid.

E. During the initial physical exam, have the client bear down and/or cough to evaluate urinary incontinence.

F. Have the client void, measure the urine, and collect the specimen for urinalysis:

1. The measurement of the urine flow rate also screens abnormalities of micturition:

a. A simple way to measure the flow rate is to time the duration of urination and measure the volume voided. A female usually voids in a continuous flow rate until the bladder is empty. Intermittent flow patterns indicate dysfunction with voiding.

2. The evaluation of urine speed and flow rate may be measured by uroflowmetry equipment utilizing a special toilet with a computer to record the flow rates.

G. Catheterize, using sterile technique:

1. Measure urine drained from a catheter inserted post voiding; this amount is the PVR:

a. Normal: less than 100 mL PVR.

b. Abnormal: more than 100 mL PVR.

c. Differential diagnostic evaluation: overflow incontinence secondary to retention.

H. Attach the 60-mL syringe to the catheter:

1. Observe if there was any difficulty passing the catheter.

a. Normal findings: catheter passes with ease.

b. Abnormal findings: catheter difficult or impossible to pass.

c. Differential diagnostic evaluation: overflow incontinence possibly due to obstruction.

I. Place the rectal or vaginal catheter to measure intra-abdominal pressure and involuntary/voluntary detrusor contractions.

J. Using gravity, fill the bladder slowly through catheter/syringe with warmed sterile water (saline or contrast medium):

1. Avoid instilling the sterile water too quickly; instill approximately 30 to 50 mL amounts at a time.

2. Measure the amount of water instilled when the client has the urge to void, different degrees of filling, and pain during filling.

a. Normal: bladder capacity usually at 300 mL when urge is sensed.

b. Abnormal: urge at less than 200 mL.

c. Differential diagnostic evaluation: urge incontinence due to small capacity.

3. Continue to fill, noting if there is any fluctuation of the sterile water in the syringe during filling:

a. Normal: contractions or fluctuation occurring around 200 to 250 mL from the bladder into the syringe (detrusor muscle contraction).

b. Abnormal: fluid moving into the syringe.

c. Differential diagnostic evaluation: urge incontinence related to bladder spasm.

4. The rectal/vaginal probe measuring detrusor activity/overactivity notes phasic pressure increases associated with symptoms of urgency or urge incontinence, indicating detrusor overactivity.

5. During the procedure, ask the client to cough or bear down to help elicit involuntary bladder contractions. The Valsalva maneuver or cough assesses the sphincter competence.

6. The instillation of sterile water should be stopped when the client reports a full bladder. Measure the bladder capacity;

that is, the client reports perception that voiding can no longer be delayed.

7. If the client has not reported a full bladder prior to the instillation of 600 mL, the test should be discontinued.

8. The urodynamic test is done until the point of leakage:

a. Have the client cough or strain; observe for leaking, clamp catheter.

b. Normal: normal capacity 400 mL, no leaking with cough.

c. Abnormal: urge prior to 400 mL or leaking with pressure.

d. Differential diagnostic evaluation: urge incontinence due to small capacity and/or stress incontinence due to leakage with pressure.

e. Coughing, shifting, and straining during the procedure are also done to evaluate bladder pressure changes and evaluate the sphincters. The manometer also measures the pressure inside the bladder when leakage occurs.

9. Remove the catheter after the bladder is filled to capacity (strong urge to void); have the client cough:

a. Normal: no leakage.

b. Abnormal: leakage of fluid.

c. Differential diagnostic evaluation: stress incontinence associated with weak pelvic muscles.

10. Have the client void; measure output, compare with the amount instilled:

a. Normal: return with a maximum of 100 mL retained (PVR).

b. Abnormal: greater than 100 mL retained after voiding.

c. Postprocedure: While the client is emptying the bladder, the manometer measures the bladder pressure and flow rate. Then remove the vaginal/rectal probe.

d. Differential diagnostic evaluation: overflow incontinence either from obstruction or an atonic bladder.

K. Discuss treatment options, including pharmaceutical therapy and/or consultation with an urologist.

L. Postprocedure instruction and when to report problems are discussed:

1. Expect some discomfort; it should lessen over time.

2. Blood during urination may be expected after the procedure. The amount of blood lessens over time and with urination.

3. Drinking additional fluids (noncaffeinated and noncarbonated beverages) will increase urination and ease discomfort and passage of blood.

4. An antibiotic may be prescribed (per protocol).

5. Anti-inflammatories such as ibuprofen may be discussed.

6. Have the client report:

a. Chills and fever.

b. Any abdominal pain.

c. Continued bloody urine 1 day postprocedure.

ESTABLISHING THE ESTIMATED DATE OF DELIVERY

The first 12 weeks of pregnancy are vital, as the fetal organ systems are developing.

EARLY DIAGNOSIS OF PREGNANCY

A. Allows counseling about potential risks to the developing fetus.

B. Enables the client contemplating elective abortion to consider their options.

C. Affords diagnosis/intervention in an ectopic pregnancy.

D. Fosters early entry into prenatal care, which in turn may provide early identification of pregnancy complications.

OBTAIN GYNECOLOGICAL HISTORY

A. Obtain sexual history.

B. Obtain fertility history:

1. Length of cycles.

2. Regularity of cycles. Regularity of menstruation is influenced by:

a. Recent previous pregnancy and cycles, which have not yet reestablished themselves.
b. Breastfeeding.
c. Oral contraceptive pills.
d. Intramuscular contraceptive.
e. Subdermal implant contraceptive.
f. Intrauterine device (IUD) contraceptive.
g. Polycystic ovarian syndrome (PCOS).
h. Comorbid medical conditions.

C. Is this a planned pregnancy? If so, how long did it take to conceive?

D. Has the client been pregnant before? If so, what was the outcome of each pregnancy (miscarriage, abortion, ectopic, stillbirth, live birth), delivery method, and fetal and/or pregnancy complications for each pregnancy?

INITIAL ASSESSMENT/DIAGNOSIS OF PREGNANCY

A. Is the exact date of the last menstrual period (LMP) known, using the first day of the last period?

B. Was the LMP a normal period? If not, when was the most recent normal period?

C. Is there a known isolated sexual incident that could pinpoint a possible conception date?

D. Does the client know when conception may have occurred?

PREGNANCY TESTING

A. Testing includes analysis of maternal blood or urine for the presence of human chorionic gonadotropin (HCG), a hormone produced by the trophoblastic cells of the developing placenta.

B. Positive testing is not diagnostic of a normal intrauterine pregnancy.

1. Maternal blood: HCG:

a. Most accurate.
b. Earliest to become positive.
c. Negative test should be repeated in 2 weeks with suspected pregnancy.

2. Urine:

a. More commonly used.
b. Accurate 4 to 7 days following conception.
c. Available over the counter.
d. First-voided morning specimen is best (most concentrated).
e. Negative test should be repeated in 1 week to ensure that the first test was not done too early to detect urine HCG. HCG production decreases after 60 to 70 days, and thereafter declines below the sensitivity of some tests.

PRESUMPTIVE SIGNS OF PREGNANCY

A. Amenorrhea.

B. Has experienced pregnancy before and feels pregnant now.

C. Nausea/vomiting: Nausea is not limited to "morning sickness."

D. Fatigue.

E. Breast tenderness.

F. Urinary frequency.

G. Enlarging abdomen.

H. Unexplained weight gain.

I. Constipation.

PROBABLE SIGNS OF PREGNANCY

A. Positive Chadwick sign: purplish coloration of vagina and cervix; may be seen on speculum examination.

B. Positive Hegar sign: softening of the lower uterine segment; may be palpated on bimanual examination.

C. Uterine enlargement:

1. 8 weeks' gestation = two times nonpregnant size.

2. 10 weeks' gestation = three times nonpregnant size.

3. 12 weeks' gestation = four times nonpregnant size, uterine fundus (size of orange) palpable at symphysis pubis.

4. 16 weeks' gestation = uterine fundus midway between symphysis pubis and umbilicus (size of grapefruit).

5. 20 weeks' gestation = uterine fundus at umbilicus (size of large honeydew melon).

D. Increased skin pigmentation (face [chloasma], nipples, abdomen [linea nigra]).

E. Striae gravidarum: "stretch marks."

POSITIVE DIAGNOSTIC SIGNS

A. Fetal heart tones (FHTs):

1. 10 to 12 weeks with ultrasonic Doppler (fetal Doppler).

2. If unable to hear FHTs with Doppler at 12 weeks (by size and dates), have the client return at 14 weeks.

3. If unable to hear FHTs at 14 weeks' gestation, repeat visit; the client needs ultrasound (US) examination to confirm fetal viability. Consult with a physician if abnormal US results.

B. Fetal movements palpated by the mother or healthcare provider.

C. Ultrasound examination:

1. Gestational sac visualized by 4 to 4.5 weeks.

2. Yolk sac by 5 to 5.5 weeks.

3. Fetal cardiac activity visualized by 6 to 6.5 weeks.

4. Embryo should be visualized when the gestational sac measures greater than 25 mm.

5. Fetal cardiac activity should be visualized when the embryo measures greater than 7 mm.

ESTIMATED DATE OF DELIVERY/ESTIMATED DATE OF CONFINEMENT

A. 280 days (40 weeks) after LMP.

B. 266 days (38 weeks) from last ovulation (in a 28-day cycle).

C. Naegele's rule for establishing estimated date of confinement (EDC): first day of the LMP plus 7 days minus 3 months = EDC.

D. Gestational calculator (in the form of a wheel) may be used to determine EDC. *Gestational wheels are based on a 28-day cycle.*

E. An online calculator is located at www.perinatology.com/calculators/Due-Date.htm.

EVALUATION OF SPRAINS

Evaluate each leg, comparing the injured with the uninjured and examining for signs of edema, deformity, tenderness, range of motion, strength, and discoloration. A neurovascular assessment should be performed for any limb injury. Palpate the dorsalis pedis and the posterior tibial arterial pulses and test for sensation.

KNEE

A. Lachman test: The Lachman test is used to diagnose injury to the anterior cruciate ligaments (ACL) and posterior cruciate ligaments (PCL). This procedure is considered the gold standard for the evaluation of anterior cruciate and posterior cruciate knee ligament damage:

1. Explain the procedure.

2. Ask the client to lie down on their back (supine).

3. The injured knee is flexed 15° to 30°and slightly externally rotated (to relax the iliotiibial band).

4. Lachman anterior: Stabilize the client's femur with one hand and then pull the proximal tibia anteriorly with the other hand.

5. Lachman posterior: Perform the same procedure except repeatedly push the tibia posteriorly.

6. Positive Lachman test interpretation:

a. Pain with normal anterior translation: ACL sprain.

b. Pain with increased anterior translation or a "soft end point": ACL rupture.

c. Pain with normal posterior: PCL sprain.

d. Pain with increased posterior translation or a "soft end point": PCL rupture.

B. Active posterior drawer test: The active posterior drawer test is a method of diagnosing PCL injury:

1. Explain the procedure.

2. Ask the client to be seated with knees flexed 90°.

3. Instruct the client to isometrically contract the hamstring (as if they to pull the foot toward their buttocks, without moving the foot) while the examiner stabilizes the foot and palpate the knee joint line.

4. The test is positive if there is posterior subluxation of the tibial plateau; posterior knee instability.

C. McMurray test: The McMurray test is a clinical rotation test for evaluating the knee joint for tears in the meniscus of the knee:

1. Explain the procedure.

2. Ask the client to lie down on their back (supine) or side-lying.

3. The affected leg should have both the hip and the knee flexed 90°.

4. Hold the foot in one hand and place the other hand on the joint line, with fingers on one side and thumb on the other side of the joint line.

5. Evaluate the client's pain level when palpating the joint when the knee is flexed.

6. Apply valgus force to the knee throughout the duration of the maneuver. Move the lower leg laterally, and simultaneously extend the leg slowly to full extension.

7. Then flex the knee to 135° and switch to varus stress.

8. Finally, repeat step 6 to apply varus force while rotating the leg medially and extending the knee.

9. If the test is positive, a crepitus sensation with movement (meniscus click or pop) can be felt, as well as knee joint line pain or "locking of the knee," which is characteristic of an ACL tear or relocation.

ANKLE

A. Anterior drawer test**:** The anterior drawer test is an examination for ACL integrity:

1. Explain the procedure.

2. Perform this test on the uninjured ankle first, then repeat on the injured ankle.

3. The client should be seated with knees flexed 90°. Ask the client to position the affected foot in the neutral position (foot slightly at rest).

4. Grasp the lower portion of the uninjured leg for bracing using one hand at the shin and grasp the heel with the other hand. Attempt to draw the heel anteriorly.

5. Repeat the test on the injured ankle.

6. Anterior drawer test is positive for an ankle sprain if the talus at the injured point slides 4 mm or more than the same bone in the other joint.

7. A dimple appears in the area in the anterior talofibular ligament (ATFL) on the anterior translation. This dimple sign indicates compromise of the ligament. The ATFL will also likely be exquisitely tender to palpation if injured.

B. Prone anterior drawer test **(also tests for ligamentous instability):**

1. Explain the procedure.

2. Perform this test on the uninjured ankle first, then repeat on the injured ankle.

3. To perform the prone anterior drawer test, the client must lie prone with their feet extending over the end of the examination table. The heel is pushed anteriorly with one hand.

4. The test is positive if there is excessive anterior movement and a dimpling of the skin on both sides of the Achilles tendon.

C. Talar tilt test: The talar tilt test (inversion stress maneuver) evaluates injury to the lateral ligaments of the ankle:

1. Explain the procedure.

2. Perform this test on the uninjured ankle first, then repeat on the injured ankle.

3. The client should be supine, seated, or on their side, with the foot relaxed.

4. To perform the talar test, grasp the lower heel with one hand while adducting or inverting the foot with your other hand.

5. The degree of tilt ranges from 0° to 23°. The test is positive if the tilt is 5° to 10° greater around the injured joint.

HERNIA REDUCTION (INGUINAL/GROIN)

DESCRIPTION

A. A hernia reduction may be done by a manual technique used for external replacement of the bowel (occasionally omentum) from the hernia back into the abdomen/groin.

INDICATIONS

A. Herniation of bowel contents occurs secondary to pregnancy, straining, hard physical labor, or congenital defect.

PRECAUTIONS

A. *Do not try to reduce a strangulated hernia.* This can cause gangrenous bowel to enter the peritoneal cavity. Strangulation symptoms include tenderness, discoloration, edema, fever, and signs of bowel obstruction.

B. Sedation and analgesia may be necessary to reduce guarding and abdominal muscular constriction.

EQUIPMENT REQUIRED

A. Gown/drape for the client.

B. Nonsterile gloves.

C. Ice or cold compress.

PROCEDURE

A. After doing a complete assessment, explain the reduction procedure to the client, discussing each step just before performing it.

B. Obtain consent if required by protocol.

C. Ask the client to void.

D. Apply ice or cold compress to the hernia for several minutes to reduce swelling and allow an easier reduction.

E. Apply nonsterile gloves.

F. Have the client in a 20° Trendelenburg position to allow gravity to help retract the herniated tissue into the pelvis.

G. After the client has had the ice pack, positioned in the Trendelenburg position, and had light sedation and analgesia, waiting up to 30 minutes may self-reduce due to the reduction of edema, force of gravity, and relaxation of muscles.

H. Ask the client to take deep, slow, relaxing breaths. Hernia reduction should cause minimal discomfort:

1. Gently invaginate the scrotal skin and replace the herniated contents back through the external/internal inguinal or femoral rings by slowing applying pressure distal to the hernia while guiding the proximal portion. Use two hands to assist in the guidance through the defect and simultaneously add gentle pressure.

I. Hernias should be easily reducible. Do not force any contents back into the abdominal wall defect. Too much distal pressure causes the hernia to balloon around the fascial opening.

J. After the reduction, ask the client to cough and strain to evaluate successful reduction prior to discharge.

EVALUATION/RESULTS OF PROCEDURE

A. Consult a physician if unable to reduce the hernia. The hernia may be strangulated.

B. Have the client return in 1 week for reexamination or sooner if needed.

C. Tell the client not to strain with bowel movements, lift heavy objects, or exercise strenuously.

D. Review symptoms of obstruction or strangulation of entrapped bowel: pain, nausea, and vomiting. At discharge, educate the client to seek immediate medical attention for symptoms of strangulated bowel.

E. Recurrent hernias and congenital defects eventually require surgical repair.

F. Hernias secondary to pregnancy may not require surgery.

INSERTING AN ORAL AIRWAY

DESCRIPTION

A. An oropharyngeal airway is a semicircular device used to hold the tongue away from the posterior wall of the pharynx. To insert this device, the mouth and pharynx should first be cleared of secretions, blood, or vomit, using a suction tip catheter (if available).

INDICATIONS

A. Oral airway insertion can be used to help control the airway, suction, ventilate, and provide oxygenation to the unconscious client.

PRECAUTIONS

A. Oral airways should be inserted only if the client is unconscious, unresponsive, and has no gag reflex. If conscious, airway insertion may stimulate vomiting and laryngospasm.

B. Oral airways should not be inserted if the client has sustained oral trauma, such as broken teeth, or has recently had oral surgery.

C. It is important to measure for the correct size of the oral airway. If the airway is too long, it could press the epiglottis against the entrance of the larynx and produce a complete airway obstruction.

D. If the airway is not inserted properly, it can push the tongue posteriorly and worsen an upper airway obstruction.

E. If the tongue and/or lips are between the teeth, trauma will result.

EQUIPMENT REQUIRED

A. An oropharyngeal airway (selection/measurement of the airway size is measured from the earlobe to the corner of the mouth).

B. A tongue blade.

C. Gloves.

D. A suction tip and canister (if available).

PROCEDURE

A. Place the head/neck in the correct position (Figure II.3).

B. The best way to insert an oral airway is to turn it backward (upside down) as it enters the mouth.

C. As the airway transverses the oral cavity and approaches the posterior wall of the pharynx, rotate it into proper position.

D. Open the client's mouth using a cross-finger technique. An alternate method is to move the tongue out of the way with a tongue blade before insertion of the airway.

E. The end flared flange of the airway should rest on the client's lips.

F. Suction if necessary.

G. If the client vomits, the airway should be removed. The mouth and airway should be cleared and the oral airway reinserted only if the client remains unconscious and has no gag reflex.

EVALUATION/RESULTS OF PROCEDURE

A. The airway is in the proper position and of the proper size when you hear clear breath sounds on auscultation of the lungs during ventilation.

B. Resuscitation will be necessary to ventilate if there is no spontaneous breathing.

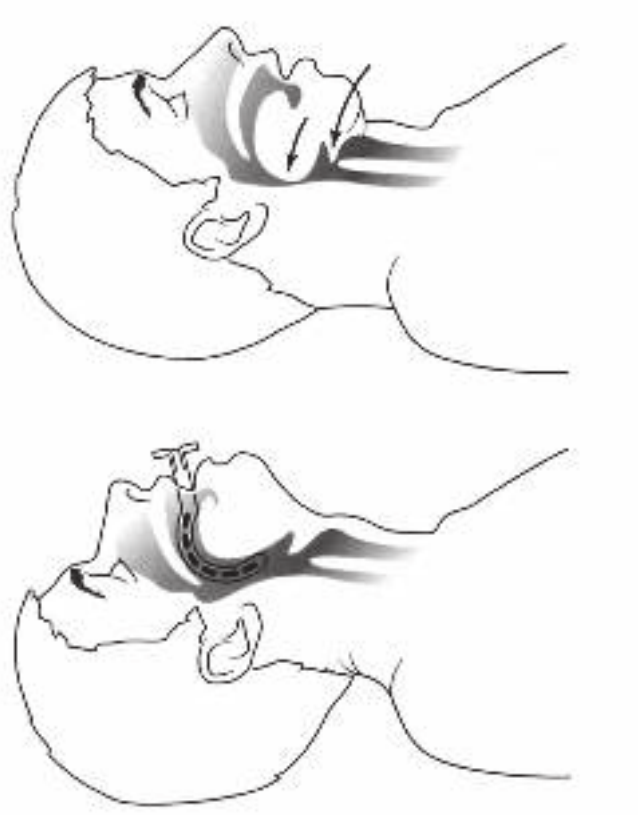


FIGURE II.3 Insertion of an oral airway.

INTRAUTERINE DEVICE INSERTION

Note: Only trained healthcare providers who carefully reviewed the selected device's manufacturer's package insert for indications and use as well as specific insertion instructions should place intrauterine devices (IUDs).

DESCRIPTION

A. The long-acting reversible contraceptive (LARC) is a long-acting method of contraception that is inserted into the uterine cavity:

1. Copper T 380 A (ParaGard):
   a. T-shaped bound in fine copper wire.
   b. **Approved for 10 years.**
   c. Clear or white knotted double string.
2. Levonorgestrel-releasing intrauterine system (LNG-IUS):
   a. Levonorgestrel (Mirena):
      i. T-shaped polyethylene frame with levonorgestrel (LNG) reservoir coating.
      ii. 52 mg: Initial release rate of LNG is 20 mcg/d; this rate is reduced by about 50% after 5 years; Mirena must be removed or replaced after 5 years.
      iii. Dark monofilament strings.
      iv. **Approved for 5 years for heavy menstrual bleeding and 7 years for contraception.**
   b. Kyleena:
      i. T-shaped polyethylene frame with LNG reservoir coating.
      ii. 19.5 mg: Release rate of LNG is 17.5 mcg/d after 24 days and declines to 7.4 mcg/d after 5 years; Kyleena must be removed or replaced after 5 years.
      iii. White monofilament strings.
      iv. **Approved for 5 years.**
   c. Skylar:
      i. T-shaped polyethylene frame with LNG reservoir coating.
      ii. 13.5 mg: Release rate of LNG is 14 mcg/d after 24 days and declines to 5 mcg/d after 3 years; Skylar must be removed or replaced after 3 years.
      iii. White monofilament strings.
      iv. **Approved for 3 years.**

INDICATIONS

A. To provide a long-acting method (3–10 years) of contraception.

B. The LNG IUDs have been therapeutic with severe dysmenorrhea and severe menorrhagia.

PRECAUTIONS

A. Pregnancy must be ruled out prior to insertion.

B. Risks at the time of insertion include uterine perforation and/or infection and a vagal response.

C. Small amounts of progestins pass into breast milk LNG IUDs.

D. To minimize the risk of expulsion, it is advisable to insert an IUD when the client is *not* on their menstrual cycle.

E. An existing IUD should be removed during the menstrual cycle.

F. Consent(s) is required by the manufacturer and by individual institutional protocols. The client should know the type of IUD device and when it should be replaced.

CONTRAINDICATIONS

A. Individuals who have a history of sexually transmitted disease and/or currently have more than one sexual partner may not be good candidates for IUD use.

B. Uterine anomaly.

C. Cervical stenosis (unable to pass a uterine sound).

D. Active pelvic infection (recent, acute, or subacute).

E. Purulent cervicitis.

F. History of ectopic pregnancy (strong relative risk).

G. Abnormal Pap smear/endometrial hyperplasia/cervical intraepithelial neoplasia (CIN)/cancer (review last recent Pap results); uterine or cervical neoplasm.

H. Concern for future fertility.

I. Allergy to copper (ParaGard) or allergy to component of LNG IUDs as applies.

J. Valvular heart disease.

K. Acute liver disease.

L. Known or suspected breast carcinoma.

Insertion of an IUD may theoretically increase the client's susceptibility to subacute bacterial endocarditis (SBE). No current evidence supports this. Optional therapy: Some clinicians require antibiotics for valvular coverage. High-risk clients may receive intravenous (IV) ampicillin and gentamicin, and moderate-risk clients may receive oral amoxicillin 2 g before insertion or removal.

EQUIPMENT REQUIRED

A. IUD.

B. Speculum.

C. Iodine solution.

D. Tenaculum.

E. Uterine sound.

F. Scissors.

G. Sterile gloves (two pairs).

H. Urine pregnancy test kit.

I. Atropine 0.5 mg should be available at the time of insertion for severe vasovagal response.

Backup staff should be available for complications such as vasovagal response and perforation of uterus.

PROCEDURE

A. Perform a urine pregnancy test.

B. Perform a baseline blood pressure and pulse.

C. Obtain the client's consent after full history and review of last Pap smear; discuss alternative methods, pros and cons of each method, procedure, side effects, and complications of IUD insertion and device.

D. With the client draped in a lithotomy position, perform a bimanual exam to determine uterine position and size.

E. Apply sterile gloves.

F. Cleanse the vagina and cervix with antiseptic solution.

G. Use the tenaculum to grasp the anterior lip of the cervix approximately 1.5 to 2.0 cm from the os:

1. Atropine should be readily available for severe vasovagal response.

2. Lidocaine (Xylocaine) gel or benzocaine (Hurricane Spray) can be used to reduce discomfort with the application and use of the tenaculum.

H. Insert the uterine sound into the cervix/uterus slowly and gently:

1. Moisture mark on the sound will indicate the depth of insertion for the IUD. (Depth <6 cm is a contraindication.)

I. Change sterile gloves.

J. Load the IUD as directed by the manufacturer.

K. Set indicator for depth of insertion as guided by the uterine sound.

L. While maintaining gentle traction with the tenaculum, insert the IUD gently to the predetermined mark on the guide sheath:

1. Spasm of the internal cervical os may occur and will resolve by waiting a few minutes.

M. Gently push the IUD through the guide into the fundal position. Excessive pain or bleeding is often a sign of perforation. Consult a physician immediately (most common perforation sites: fundus, body of uterus, and cervical wall).

N. Remove the guide sheath.

O. Remove the tenaculum.

P. Cut the IUD strings to approximately 1-in. length past the cervix.

Q. Remove the speculum.

R. Check the client's blood pressure and pulse to rule out vasovagal potential.

S. Recommend a prostaglandin inhibitor if cramping occurs/persists (Motrin 400 mg PO QID).

T. Prophylactic antibiotics may be used for SBE prophylaxis. Amoxicillin is used unless there is an amoxicillin/penicillin allergy; then erythromycin (erythromycin ethylsuccinate [EES]) or clindamycin may be prescribed. Amoxicillin 3.0 g PO 1 hour before the procedure, then 1.5 g PO 6 hours later:

1. EES 800 mg PO 2 hours before the procedure, then 400 mg PO 6 hours later.

2. Clindamycin 300 mg PO 1 hour before the procedure, then 150 mg 6 hours later.

U. Instruct the client regarding string location and periodic checks. The client should not have vaginal intercourse or use a tampon for 7 days (reduces infection).

EVALUATION/RESULTS OF PROCEDURE

A. See the client after first menses to check for expulsion.

B. If the client has missed two or more periods, perform a beta HCG (human chorionic gonadotropin) test:

1. Rule out ectopic pregnancy.

2. Explore the canal for IUD.

3. Consider ultrasound to rule out pregnancy or expulsion.

4. Consider flat plate of abdomen.

5. Consult a physician.

C. Yearly evaluation with Pap smear/annual checkup.

D. Consider hemoglobin (Hgb) for reports of heavy bleeding with ParaGard:

1. If Hgb is less than 9, remove the IUD, provide an alternative method of contraception, and prescribe iron supplement for 2 months.

2. Repeat Hgb in 1 month.

INTRAUTERINE DEVICE REMOVAL PROCEDURE

A. Remove only during menses (cervix is more dilated).

B. Apply gentle, steady traction; remove slowly.

C. Consider uterine embedding if unable to remove; needs consultation.

CLIENT SHOULD CALL THE OFFICE FOR

A. A missed period.

B. Abdominal pain (severe).

C. Temperature 100.4°F not associated with other problems (sinus infection, urinary tract infection [UTI]).

D. Foul vaginal odor/discharge, especially if bloody or greenish color.

E. Heavy bleeding with clots.

NEUROLOGIC EXAMINATION

DESCRIPTION

A. Performance of the neurologic examination is a diagnostic component of many physical examinations. Examination of all cranial nerves is not necessary for every client; it should be performed based on client-focused presenting complaints. The purpose of this support tool is to review the cranial nerves and review equipment needed for a neurologic examination.

B. A neurologic examination identifies dysfunction and assists in accurate diagnosis (Table II.2).

COMPONENTS OF THE NEUROLOGIC EXAMINATION

A. Thorough history.
B. Assessment of mental status.
C. Assessment of cranial nerve function.
D. Assessment of motor function.
E. Assessment of sensory function.
F. Assessment of gait.
G. Assessment of reflexes.

EQUIPMENT REQUIRED

A. Multiple mental examinations are available. The clock-draw test (CDT) and the Mini-Mental State Examination are two examples.
B. Pen light/ophthalmoscope: light source.
C. Ophthalmoscope.
D. Snellen chart/Rosenbaum pocket card.
E. Items to taste (salt, sugar).
F. Items to smell (vanilla, cinnamon).
G. Tongue depressor.
H. Tuning fork.
I. Ticking watch/clock
J. Cotton or soft tissue.
K. Pin or other sharp object.
L. Reflex hammer.

TABLE II.2 CRANIAL NERVE FUNCTION

Cranial Nerve Number	Cranial Nerve	Functional Test
I	Olfactory	Smell reception and interpretation
II	Optic	Vision: acuity and visual fields
III	Oculomotor	Motor: eye/lid movement, pupil constriction, visual accommodation
IV	Trochlear	Eye movement up/down
V	Trigeminal	Chewing, jaw opening and clenching, facial sensation Corneal, forehead, eyes, nose/mouth mucosa, teeth, tongue, and ears
VI	Abducens	Lateral gaze/eye movement
VII	Facial	Corneal reflex, facial expression, taste to the lateral two-thirds of tongue, and sensation to pharynx; parasympathetic secretion of tears and saliva
VIII	Acoustic	Hearing and equilibrium
IX	Glosso-medulla or glossopharyngeal	Gag reflex, cough, swallow, taste on posterior third of tongue, parasympathetic secretion of salivary glands, and carotid reflex
X	Vagus	Laryngeal muscles: muscles of phonation, swallowing, sensation behind ear and part of external canal, parasympathetic secretion of digestive enzymes/peristalsis as well as involuntary action of heart and lungs
XI	Spinal accessory	Head and shoulder movement: shoulder shrug
XII	Hypoglossal	Tongue movement/muscles, swallowing, and sound articulation

Source: Adapted from Seidel, H. M., Ball, J. W, Dains, J. E., & Benedict, G. W. (1991). *Mosby's guide to physical examination* (2nd ed.). Mosby Year-Book.

PESSARY INSERTION AND MANAGEMENT

DESCRIPTION

A. This conservative therapy option is the gold standard for pelvic organ prolapse (POP) management.

1. Pessaries are made from medical-grade silicone.

2. Many different styles and sizes are available to enable successful insertion. (Only four styles of pessaries are noted in this procedure.)

3. Pessaries can be for temporary or long-term use.

INDICATIONS

A. POP.

B. Stress urinary incontinence (SUI).

C. Client frailty prevents surgical correction of POP, or client chooses to avoid surgery for personal reasons.

D. Can be used for diagnostic purposes when evaluating for potential SUI, or when symptoms described by the client do not fit with common POP symptoms.

E. Can serve as potential reduction of prolapse progression.

PRECAUTIONS

A. Confirm client is agreeable to a trial of pessary.

B. Confirm client can be an active participant in their pessary care.

C. In the event of dementia or frailty, ensure the client has a caretaker who is willing to participate in the client's care.

D. Client and/or caregiver must be agreeable to frequent follow-up with the clinician.

CONTRAINDICATIONS

A. Client's inability or caregiver inability to collaborate in follow-up care and maintenance.

B. Severe vaginal atrophy.

C. Current vaginal infection.

D. Prolapse ulcer.

EQUIPMENT REQUIRED

A. Two to three different styles and sizes of pessaries.

B. Latex-free gloves.

C. Lubricating jelly.

PROCEDURE

A. Little evidence exists to determine the most appropriate pessary style choice.

B. Style is usually a subjective choice based on clinician comfort and familiarity.

C. Allow client to hold pessary, becoming more comfortable with it.

D. Fit with client in lithotomy position.

E. Apply water-based lubricant to gloved hand and carefully reduce the prolapse.

F. Assess vaginal length using gloved hand, two fingers in the vagina with fingertips at the vaginal cuff or posterior fornix (Figure II.4).

G. Separate the two intravaginal fingers to assess for width of the vaginal vault (Figure II.5).

H. Select appropriate type and size of pessary, cleansing with soap and water prior to insertion (Table II.3).

I. Insert pessary using gentle pressure toward the posterior vaginal wall:



FIGURE II.4 Assessment of vaginal length using two fingers placed together in the vagina.



FIGURE II.5 Separate the two intravaginal fingers to assess the width of the vaginal vault.

1. Insertion of a ring with support pessary: Fold in half; pessary will spring open in vaginal vault (Figures II.6– II.8).

2. Insertion of a Gellhorn pessary: Bend the stem or knob down against the dish (Figures II.9–II.11).

3. Insertion of a donut pessary: Compress the pessary, attempting to "squish" it (Figures II.12–II.14).

4. Insertion of a cube pessary: Compress the pessary, attempting to "squish" it (Figures II.15–II.17).

J. Check for fit by asking the client to Valsalva or cough. Perform with client in both lithotomy and standing positions.

K. Ask the client to walk, sit, and bend to determine comfort of fit.

L. Instruct client to attempt to void to determine ability to empty and to confirm proper fit.

EVALUATION OF PROCEDURE

A. If the client is comfortable with their new pessary, review removal procedures with them.

B. If the client is comfortable with practicing in the office, have them practice removal and replacement in the exam room.

REMOVAL PROCEDURE

A. A ring with support pessary should be removed by inserting the index finger into the vagina, feeling for the hole, and pulling downward toward the introitus.

TABLE II.3 EXAMPLE OF TYPES OF PESSARY AND USE WITH POP AND SUI

Pessary	Prolapse 1–2 Degree	Prolapse 3–4 Degree	Cystocele	Rectocele	SUI
Ring with support	X		X		X
Donut		X	X	X	
Cube	X	X	X	X	X
Gellhorn		X	X		

POP, pelvic organ prolapse; SUI, stress urinary incontinence.

Source: Modified from the Milex pessary guide. (n.d.). https://www.incoshop.co.uk/milex-pessaries-50-c.asp



FIGURE II.6 Ring with support pessary.

Source: Used with permission from CooperSurgical.coopersurgical.com.

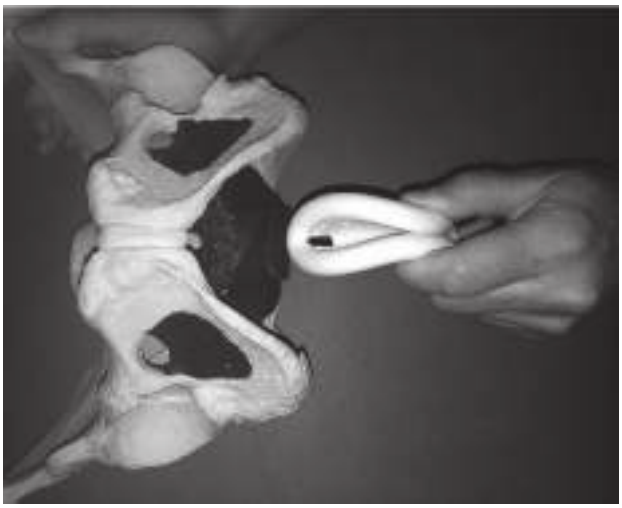


FIGURE II.7 Fold the ring pessary in half for insertion into the vaginal vault.

Source: Photograph courtesy of Barry Jarnigan, MD, Center for Pelvic Health.

B. A Gellhorn pessary can be removed by grasping the knob and gently pulling it laterally while also pulling downward toward the introitus. It may be helpful to ask the client to Valsalva during the removal.



FIGURE II.8 Position of the ring pessary in the vaginal vault.

Source: CooperSurgical (2015). Clinic and practice-based products. http ://www.coopersurgical.com/Portals/_default/Skins/ Cooper/catalogs/ ClinicCatalog.pdf.

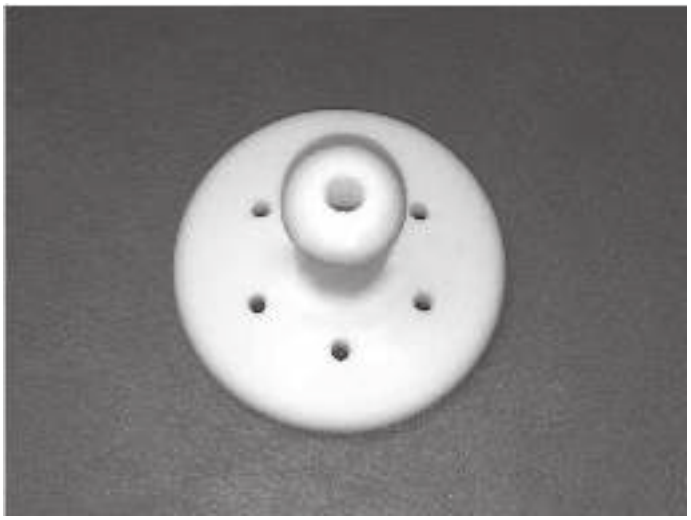


FIGURE II.9 Gellhorn pessary.

Source: Used with permission from CooperSurgical. coopersurgical.com.

1. Removal of the donut pessary can be done by inserting the index finger into the large hole in the center and gently pulling toward the introitus.

CLIENT SHOULD CALL THE OFFICE FOR

A. Pain when the pessary is in place.
B. Vaginal bleeding.
C. Inability to void or defecate.
D. Inability to retain the pessary.

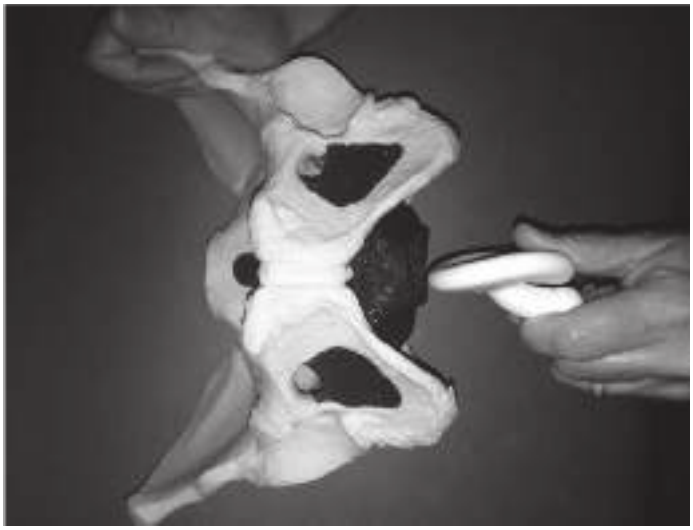


FIGURE II.10 Bend the stem or knob down against the dish for insertion of the Gellhorn pessary.

Source: Photograph courtesy of Barry Jarnigan, MD, Center for Pelvic Health.



FIGURE II.11 Position of the Gellhorn pessary in the vaginal vault.

Source: From CooperSurgical (2015). Clinic and practice-based products. http://www.coopersurgical.com/Portals/_default/Skins/Cooper/catalogs/ClinicCatalog.pdf.

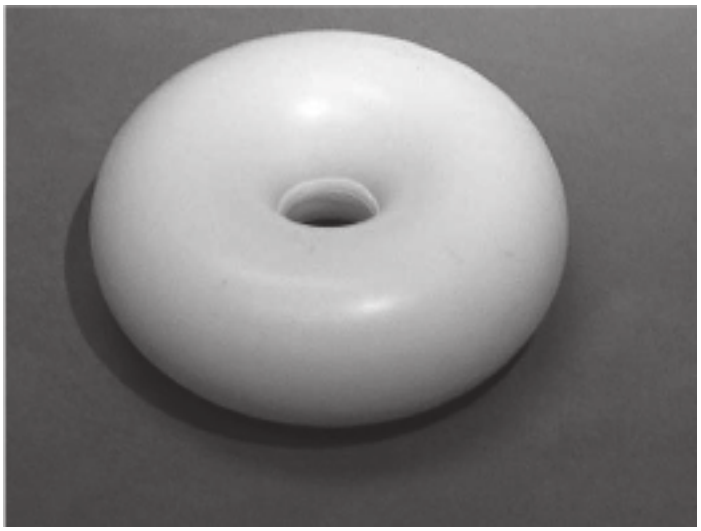


FIGURE II.12 Donut pessary.

Source: Used with permission from CooperSurgical. coopersurgical.com.

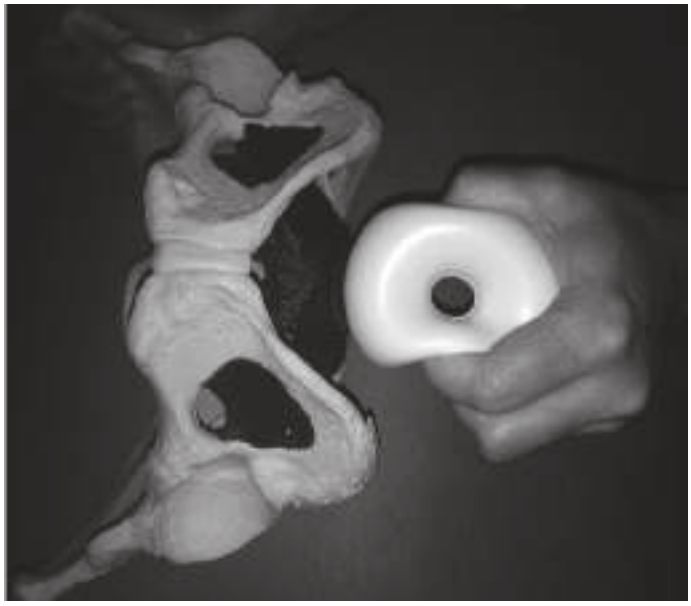


FIGURE II.13 Squish the donut pessary for insertion into the vaginal vault.

Source: Photograph courtesy of Barry Jarnigan, MD, Center for Pelvic Health.



FIGURE II.14 Position of the donut pessary in the vaginal vault.

Source: From CooperSurgical (2015). Clinic and practice-based products. http://www.coopersurgical.com.

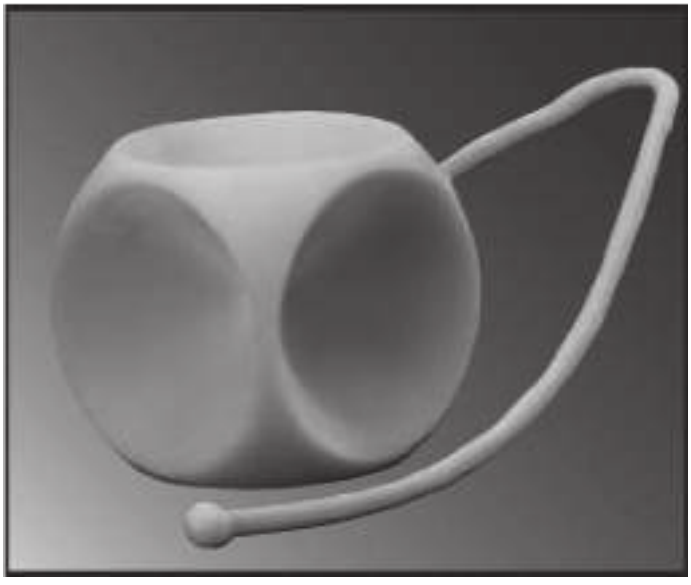


FIGURE II.15 Cube pessary.

Source: Used with permission from Cooper Surgical. (2018) Milex Cube Pessary. https://www.coopersurgical.com/Medical-Devices/Detail/milex-cube-pessary.



FIGURE II.16 Cube pessary being compressed for insertion.

Source: Photograph courtesy of Barry Jarnigan, MD, Center for Pelvic Health.



FIGURE II.17 Position of the cube pessary in the vaginal vault.

Source: From CooperSurgical (2015). Clinic and practice-based products. http://www.coopersurgical.com.

FOLLOW-UP

A. Follow up after completion of fitting session:

1. Instruct the client to return for follow-up within 2 weeks if they are unable to remove the pessary.

2. Instruct the client to return for follow-up in 4 weeks if they can remove and replace the pessary.

B. Follow up for routine maintenance:

1. If the client is unable to manage their pessary, follow-up should occur every 3 months with the clinician.

2. If the client can manage their pessary, follow-up can occur every 6 months to annually.

PESSARY SUPPLIERS

A. Bioteque America, www.bioteque.com.

B. CooperSurgical, www.coopersurgical.com.

C. MedicalExpo, www.medicalexpo.com.

PROSTATIC MASSAGE TECHNIQUE: TWO-GLASS TEST

DESCRIPTION

A. Obtaining prostatic fluid by prostate massage using the two-glass test known as the Nickel premassage and postmassage test. The Meares–Stamey four-glass test is considered too time-consuming and is rarely used by urologists.

INDICATIONS

A. Expressed prostatic secretion (EPS) is used to distinguish chronic prostatitis from acute prostatitis or urinary tract infection (UTI).

PRECAUTIONS

A. Do not perform in clients with acute prostatitis (swollen, tender, boggy prostate); may cause septicemia.

EQUIPMENT REQUIRED

A. Clean-catch specimen cups (2).
B. Labels (2) marked:
  1. Premassage.
  2. Postmassage.
C. Sterilized towelette.

PROCEDURE

A. Collection of a clean-catch midstream urine specimen for male clients: The client should retract the foreskin (if applies) and cleanse the glans penis with a sterilized towelette in order to avoid contamination.
B. The client should collect a midstream flow of urine by starting the flow of urination, then catch at least 10 mL of urine in the first clean-catch specimen cup marked premassage. The client should be instructed to then stop the flow of urine.
C. The client is asked to lean over the examining table for a digital rectal examination. With a lubricated index finger, gently enter the rectum. The prostate should be massaged by stroking it from the periphery toward the midline several times on each side (similar to a windshield wiper).
D. Following the massage, ask the client to provide more urine in the second clean-catch specimen cup labeled postmassage.
E. Send both the premassage and postmassage urine specimens for urine cultures.

TREATMENT

A. See Table II.4.

TABLE II.4 EVALUATION AND RESULTS OF PROSTATIC SECRETIONS

Diagnosis	Microscopic Evaluation	Treatment
Urethritis	WBCs, bacteria in VB_1; in no other specimens	<40 years, probably STI Rx: ceftriaxone 250 mg IM in a single dose PLUS azithromycin 1 g PO in a single dose >40 years, probably coliform bacteria nongonococcal urethritis Rx: azithromycin 1 g PO in a single dose OR doxycycline 100 mg BID × 7 days
Cystitis	WBCs, RBCs, bacteria in VB_2	First-line therapy: trimethoprim-sulfamethoxazole PO BID × 7–10 days Second-line therapy: quinolones: ciprofloxacin 500 mg BID for 3 days Alternative therapy: amoxicillin-clavulanate 875/125 mg BID × 7 days OR amoxicillin-clavulanate 500/125 mg TID × 7 days
Acute prostatitis	WBCs, bacteria in VB_2 and/or EPS, VB_3	Trimethoprim-sulfamethoxazole PO BID for 14–28 days First-line therapy: quinolones: Ciprofloxacin 500 mg BID for4 weeks, OR Ofloxacin 400 mg BID × 4–6 weeks, OR Levofloxacin 500–750 mg daily × 4 weeks Second-line therapy: trimethoprim-sulfamethoxazole DS BID × 14 days May need hospitalization for IV antibiotics
Chronic prostatitis	Bacteria in EPS and/or VB_3, but not in VB_1 or VB_2	Trimethoprim-sulfamethoxazole BID for 3–4 months PO, OR Ciprofloxacin BID 500 mg BID for 3–4 months, OR Tetracyclines: doxycycline 100 mg BID × 4 weeks

EPS, expressed prostatic secretion; IM, intramuscular; IV, intravenous; RBCs, red blood cells; Rx, prescription; STI, sexually transmitted infection; VB_1, voided bladder 1, represents the urethra; VB_2, voided bladder 2, represents the bladder; VB_3, voided bladder 3, represents the prostate; WBCs, white blood cells.

RECTAL PROLAPSE REDUCTION

DESCRIPTION

A. There are three types of rectal prolapse:

1. Partial: Anus is inverted; rectal mucosa protrudes 1 to 3 cm out of rectal sphincter.

2. Complete: All layers of rectum have intussuscepted through the anus. (Rare in adults; most often seen in children and the elderly.)

3. Internal: Client complains of a protrusion, but no protrusion is noted on examination.

INDICATIONS

A. Diagnosis of rectal prolapse is made by inspection. Several populations are at risk of rectal prolapse, including infants, females, and the elderly. Other factors associated with rectal prolapse include malnutrition, severe constipation, digital removal of stool impactions, severe chronic diarrhea, obstetrical injuries, rectal intercourse, and multiple sclerosis as well as other neuromuscular diseases.

B. Hemorrhoids, mucosal prolapse, polyps, and cancer must be ruled out. Conditions associated with rectal prolapse include cystic fibrosis, ulcerative colitis, intussusception, and Hirschsprung disease.

PRECAUTIONS

If a manual reduction fails, an emergent surgical referral should be done.

A. Refer clients with complete prolapse to a physician for evaluation and treatment. After a rectal prolapse reduction by proctoscope, follow-up testing by a barium enema, endoscopy, manometry, defecography, or MRI if indicated.

B. Surgery may also be indicated, depending on the type of prolapse, recurrence, and risk of strangulation.

C. Clients with a history of surgery for rectal prolapse should be evaluated and assessed for hemorrhage, bowel obstruction, pelvic abscess, fecal impaction, and recurrent prolapse.

D. Instruct the client on a high-residue diet and adequate fluid intake to prevent constipation. Instruct the client to avoid straining to induce a bowel movement. Stool softeners, laxatives, and enemas may need to be used to prevent constipation. To reduce the recurrence of prolapse, pelvic floor exercises (Kegel) that strengthen the pelvic floor muscles may also be used.

EQUIPMENT REQUIRED

A. Sterile gauze.
B. Normal saline (NS).
C. Anoscope.
D. Water-soluble lubricant.
E. Gloves.
F. Chux pads and tissue.
G. Light source.
H. Examination table.

PROCEDURE

A. Explain the procedure to the client and discuss all steps just before performing. Explain that during the procedure, relaxation of the anal sphincter is key.

B. Obtain the client's consent.

C. Ask the client to void. (Urinary stress incontinence is common during the procedure.)

D. Apply gloves.

E. Have the client lie in the left lateral position and give extra tissues for urinary incontinence.

F. Ask the client to strain (perform Valsalva maneuver). This allows the practitioner to fully evaluate the prolapse through the anal sphincter. If this does not produce the prolapse, have the client squat and/or sit on the toilet to strain.

G. With a good light source, do a detailed visual examination to rule out hemorrhoids versus a complete/partial prolapse.

H. Gentle manual pressure should be applied distally to slide the distal end of the prolapse into the lumen and through the anal sphincter.

I. A rectal prolapse that is large and/or difficult may be reduced by an application of sugar to reduce swelling.

J. As the procedure is being done, give the client and/or caregiver instructions on how reduction is done in the event of another prolapse:

1. Provide the client with gloves and lubricant for future need.

2. Prolapses may also reduce spontaneously without any manual reduction.

REMOVAL OF A FOREIGN BODY FROM THE NOSE

DESCRIPTION

A. This is the technique for removing a nasal foreign body (NFB) that is anterior to the pharynx and can be visualized with a nasal speculum.

INDICATIONS

A. Clients with retained NFB often present with unilateral, foul rhinorrhea, nosebleeds, or the request for foreign body removal.

PRECAUTIONS

A. Try to identify the type of object, organic or inorganic:

1. Organic foreign objects include food items (e.g., peas and beans), sponges, or rubber.

2. Inorganic foreign objects include a rock, pearl, battery, or small toy parts.

B. If you cannot identify the object, do not irrigate the nares before removal.

C. Vegetable matter will swell if hydrated, so remove it in a dry environment when possible.

D. Small "button" batteries from toys are common obstructions and should not be irrigated due to the destruction from the low-volt current and potential spread of alkaline content.

E. When the object is too far into the nasal turbinate/cavity or when in doubt, do not attempt the procedure and refer to an otolaryngologist:

1. The procedure may need to be done under sedation.

2. The object may need to be surgically removed.

EQUIPMENT

A. Proper lighting (headlight or head mirror).

B. Nasal speculum.

C. Topical anesthetic for use prior to procedure (topical 1% lidocaine with 0.5% phenylephrine).

D. Suction and several suction tips.

E. Other equipment should be available, depending on the type of foreign body: wire loop or curette, alligator or Hartman forceps, bayonet forceps, cyanoacrylate glue (Dermabond), right-angle hooks, a Foley catheter, and suction.

PROCEDURE

A. Determine the type of object in the nostril during history-taking in order to determine the best approach for removal.

B. Explain the procedure to the client.

C. Obtain the client's consent for removal.

D. To best visualize the nasal cavity, have the client lie down or sit erect with the head slightly tilted.

E. Visualize the nostril with the nasal speculum (insert speculum into nares with handles horizontal).

F. If tissue edema is present in the nares, you may use a topical anesthetic with vasoconstrictor to the affected nares to help prevent bleeding.

G. If the client is willing, have them dislodge the foreign body with forceful nose blowing while occluding the unaffected nostril and keeping the mouth closed.

H. The client should be supine or with a slight elevation of the head with assistance to stabilize their head.

I. If forceful blowing is not successful, attempt to remove the foreign object using the most appropriate instrument or technique (wire loop or curette, alligator or Hartman forceps, bayonet forceps, right-angle hooks, and suctioning). The choice of instrument for removal is dependent upon the type of object, location, and how well the client is able to cooperate during the procedure.

J. Use of a catheter involves lubricating the tip, inserting past the foreign body, and inflating the balloon with approximately 2 mL of air or water.

K. Gentle withdrawal will pull out the foreign object.

L. Use of cyanoacrylate glue involves placing a drop of the adhesive on the wooden tip of a cotton-tipped applicator, touching the foreign body with the wooden tip, and gently pulling the object out of the nares.

M. Simple external pressure should be applied to prevent bleeding.

EVALUATION/RESULTS OF PROCEDURE

A. Evaluate the other nares and ears if one foreign object is found.

B. Bleeding is common after the removal of a foreign object. Pressure should be applied to stop the bleeding.

C. Follow up in 2 days to evaluate the mucosa.

D. Consult a physician if you are unable to dislodge or remove the object.

E. Button batteries require prompt removal and inspection due to the destruction to the mucosa.

REMOVAL OF A TICK

DESCRIPTION

A. Ticks are vectors for Lyme disease, deer tick fever, and Rocky Mountain spotted fever. Ticks should be removed as soon as possible since they pass bacteria that cause the fevers and rashes.

INDICATIONS

A. There are several populations at risk for tickborne transmission diseases, including those involved in any outdoor recreational activity; those in contact with heavy brushy areas such as hunters, campers, and landscapers; and persons in contact with dogs, deer, rodents, and other animals with ticks.

EQUIPMENT REQUIRED

A. Fine-tip tweezers.
B. Antiseptic or rubbing alcohol.
C. Gloves; nonsterile may be used.
D. Office setting: scalpel if the tick head remains.

PROCEDURE

A. Use gloves.
B. To remove the tick, care should be taken to avoid squeezing the body of the tick:

1. It should be grasped with a fine-tip tweezer close to the skin.
2. Remove by gently pulling the tick upward straight out without using any twisting motions (Figures II.18 and II.19).
3. Do not crush the tick during removal.

C. Examine for the entire head and body removal; if the head is embedded, a small incision may be required for the removal.
D. Cleanse the skin with antiseptic or rubbing alcohol.
E. Save the tick for identification if required by your office protocol. Write the client's name, date of removal, and date of the tick bite on paper, and place the paper and the tick in a resealable baggie and place it in the freezer.
F. If your office protocol does not require saving the tick for identification, dispose by placing the tick in alcohol, place it in a sealed bag/container, and dispose.

EVALUATION/RESULTS OF PROCEDURE

A. Examine the rest of the body for other ticks.
B. Try to determine the length of time the tick was on the skin.
C. Assess for symptoms of Lyme disease, Rocky Mountain spotted fever, and deer tick fever.
D. Medications and consultants as applicable.

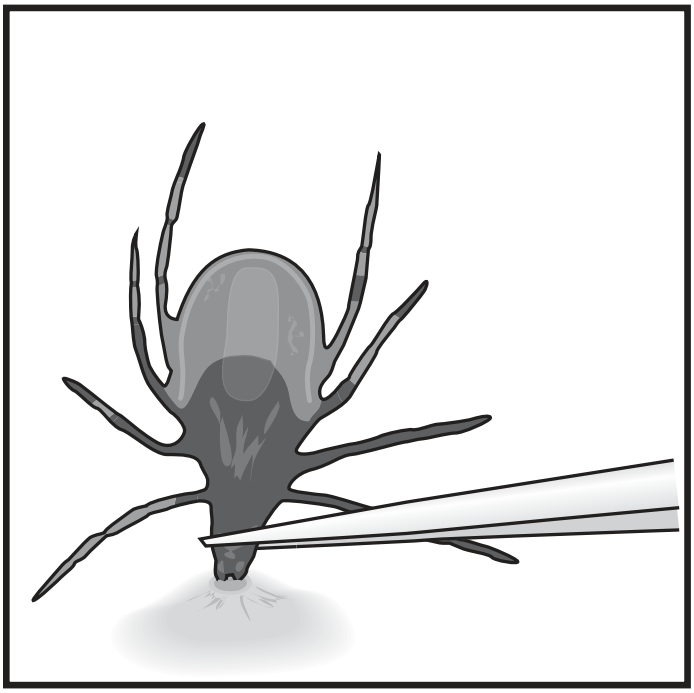


FIGURE II.18 Grasp the tick's body close to the skin using a fine-tip tweezer. Avoid squeezing the body.

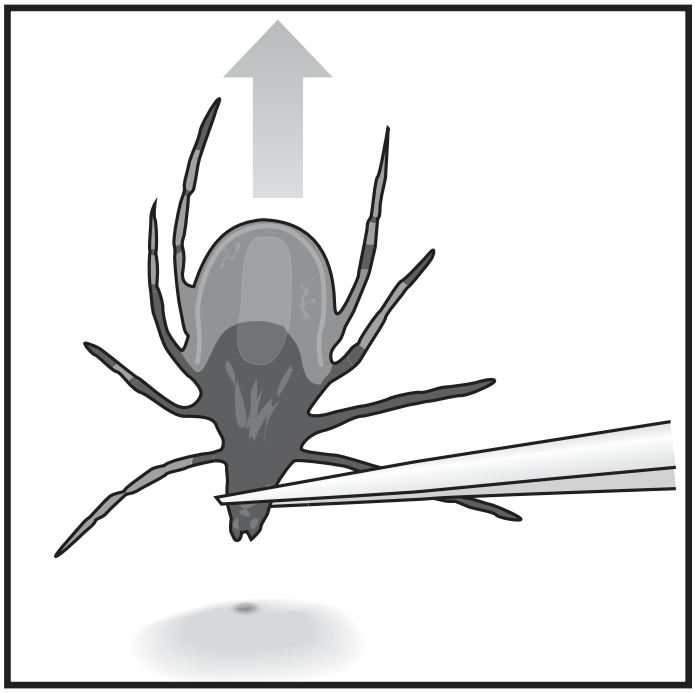


FIGURE II.19 Remove by pulling the tick straight upward without using twisting motions.

TRICHLOROACETIC ACID/PODOPHYLLIN THERAPY

DESCRIPTION

A. Trichloroacetic acid (TCA) and podophyllin are substances used to eliminate exophytic warts on the external genitalia and perianal area.

INDICATIONS

A. External genitalia and perianal warts.

PRECAUTIONS

A. Podophyllin use is contraindicated in pregnancy.

B. Either substance should be applied sparingly to avoid contact with unaffected skin.

EQUIPMENT REQUIRED

A. Examining table with stirrups for positioning the female client in the lithotomy position.

B. Light source for examining the genitalia and perianal area.

C. Gown/drape for the client.

D. Gloves: Nonsterile glove may be used.

E. Cotton-tipped applicators.

F. Petroleum jelly.

G. Sodium bicarbonate (baking soda) or powder with talc.

H. Plastic medicine cups.

I. TCA 80% to 90%. OR

J. Podophyllin 10% to 25% in compound tincture of benzoin.

PROCEDURE

A. Assist the female client to the lithotomy position, or the male client to a sitting or supine position, on the exam table.

B. Inspect the external genitalia and perianal areas for exophytic warts.

C. Pour a small amount (0.5–1.0 mL) of solution (TCA or podophyllin) to be used in a plastic medicine cup.

D. Use the cotton-tipped applicator to apply petroleum jelly on unaffected skin surrounding areas identified for therapy.

E. Use the wooden end of the cotton-tipped applicator to apply a small amount of solution (TCA or podophyllin) to the warts.

F. If TCA is used, apply sodium bicarbonate or powder with talc to absorb unreacted acid:

1. The client should not sit or stand and dress until the treatment has dried.

G. If podophyllin is used, the area should air-dry before the client dresses; instruct the client to thoroughly wash off solution in 1 to 4 hours.

H. Repeat weekly if necessary. If warts persist after six applications, consider other therapeutic methods.

I. Advise the client that mild to moderate pain or local irritation may occur after treatment.

WET MOUNT/CERVICAL CULTURES

DESCRIPTION

A. The wet mount procedure is a technique used to identify vulvovaginitis, commonly caused by trichomoniasis, bacterial vaginosis, and vulvovaginal candidiasis. Any client complaining of vaginal discharge, irritation, vaginal pain, and/or a vaginal odor should have a wet mount performed to aid in identifying the organism to assist in the diagnosis.

INDICATIONS

A. Vaginal discharge.
B. Vulvar/vaginal irritation or pain.
C. Vaginal discharge with an odor.
D. Prior to the examination: Take a thorough history prior to the physical examination to determine if other testing needs to be performed.
E. Presenting complaints (refer to Table II.5 for a comparison of bacterial vaginosis, candidiasis, and trichomoniasis):

1. Bacterial vaginosis: complaints of a thin off-white "grayish" discharge and, musty "fishy" amine odor. The odor increases after intercourse.
2. Vulvovaginal candidiasis: thick, white "cottage cheese" discharge, no odor, pruritus, and some dysuria.
3. Trichomoniasis (parasite *Trichomonas vaginalis*): copious, frothy, yellow-gray to green discolored, malodorous vaginal discharge, pruritus, and vaginal irritation. *The client may also be asymptomatic*.

PRECAUTIONS

A. Use universal precautions when obtaining specimens.
B. All specimens need to be disposed of as biohazard waste.

EQUIPMENT REQUIRED

A. Examining table with stirrups for positioning the client in the lithotomy position.
B. Light source for examining the cervix.
C. Gown/drape for the client.
D. Gloves, two pairs (nonsterile).
E. Speculum: Always have several different types and sizes of speculums on hand for appropriate sizing, visualization, and comfort.
F. Condoms available, if needed.
G. Q-tip/cotton-tipped applicators.
H. Gen-Probe, or other collecting tubes for *Neisseria gonorrhoeae* and *Chlamydia* specimens.
I. Small test tube/plastic collection tube.
J. Normal saline (NS) and 10% potassium hydroxide (KOH).
K. Glass slides (2): one marked KOH and one marked NS.
L. Cover slips for slides (2).
M. Nitrazine pH test tape.
N. Microscope with 10× and 40× objectives.

PROCEDURE

A. After the client has voided and changed into a gown, assist the client into the lithotomy position.
B. Apply gloves.
C. Prepare the test tube/specimen tube with 1 mL of NS. Prepare slides (one marked KOH and one marked NS); apply one drop of each solution to a clean slide and set aside.
D. Inspect the perineum for lesions, erythema, fissures, condyloma, and lacerations:

1. Bacterial vaginosis: usually normal-appearing tissue.
2. Vulvovaginal candidiasis: vulvar and vaginal erythema, edema, and fissures.
3. Vulvar and vaginal edema and erythema.

E. Gently insert the speculum into the vagina. Do not use lubricant since this may interfere with the quality of the specimen. Use warm water for lubrication.
F. While inserting the speculum, visualize the vaginal vault, walls, and cervix. Note any irregularities such as lesions, masses, and lacerations.
G. If the lateral sidewalls collapse and decrease visualization of the cervix, withdraw the speculum. Apply a condom to the speculum and cut the condom tip. Reinsert the condom-covered speculum to visualize the cervix.

TABLE II.5 VAGINITIS DIFFERENTIATION

	Normal	Bacterial Vaginosis	Candidiasis	Trichomoniasis
Symptom presentation		Odor, discharge, itch	Itch, discomfort, dysuria, thick discharge	Itch, discharge, 70% asymptomatic
Vaginal discharge	Clear to white	Homogeneous, adherent, thin, milky white; malodorous "**foul fishy"**	Thick, clumpy, white **"cottage cheese"**	Frothy, gray or yellow-green; malodorous
Clinical findings			Inflammation and erythema	Cervical petechiae "**strawberry cervix"**
Vaginal pH	3.8–4.2	>4.5	Usually <4.5	>4.5
KOH "whiff" test	Negative	Positive	Negative	Often positive
NaCl wet mount	Lactobacilli	Clue cells (>20%), no/few WBCs	Few to many WBCs	Motile flagellated protozoa, many WBCs
KOH wet mount			Pseudohyphae or spores if non-*albicans* species	

KOH, potassium hydroxide; NaCl, sodium chloride; WBCs, white blood cells.

H. Observe for vaginal discharge, noting the amount, color, consistency, and odor:

1. Bacterial vaginosis: discolored discharge with odor, homogeneous discharge that adheres to the vaginal walls.

2. Vulvovaginal candidiasis: thick white discharge that adheres to the vaginal walls.

3. Frothy, purulent discharge and a "strawberry" cervix can be identified.

I. Collect a sample of discharge with the two Q-tips:

1. Place one Q-tip into a prefilled test tube/specimen tube and make your slide later. OR

2. Collect a sample of discharge with the Q-tip and roll the Q-tip into the NS-prepared slide cover with a cover slip.

3. *Then* roll the Q-tip into the KOH-prepared slide.

4. Note any "musty/fishy" amine odor prior to placing the cover slip on the slide. (Whiff test is positive if amine odor is noted.)

J. Using the second Q-tip, roll it over a strip of nitrazine test tape to evaluate pH.

K. Using the Gen-Probe, insert the tip onto the cervical os and rotate the tip in the os several times to obtain an adequate specimen (sample for a minimum of about 30 seconds).

L. Withdraw the Gen-Probe applicator and place in the appropriate specimen container.

M. Gently remove the speculum and perform a bimanual examination, assessing for cervical motion tenderness and pain.

N. After completing this exam, take the specimens to the lab area for evaluation (while the client is dressing).

O. Prepare your slides (if you did not follow steps I.1–I.4):

1. Slide 1: Place one drop of NS to the slide, roll the Q-tip, and apply the cover slip.

2. Slide 2: Place one drop of KOH to the slide and roll the Q-tip; note any "musty/fishy" amine odor from the slide prior to placing the cover slide.

P. Observe the NS slide, first under the microscope. Start on low power, 10×, and adjust the focus until the specimen is clearly seen under the microscope.

Q. Observe the entire field and note the number of squamous cells.

R. Switch to the high-powered field, 40×, making sure the light source and the visual focus are adequate.

S. Observe the slide for:

1. Vaginal squamous epithelial cells: appear flat, with clear edges. Red blood cells (RBCs), sperm, or polymorphonuclear neutrophils (PMNs) may also be identified (Figure II.20).

2. Clue cells: epithelial cells with irregular borders and a granular appearance; may see the presence of coccobacilli bacteria (not present on slide; Figure II.21).

3. Bacteria.

4. *Lactobacilli:* appear as rods (Figure II.22).

5. White blood cells (WBCs): Note: a large number of WBCs is not normal.

6. Trichomonads: appear as ovoid mobile organisms that dart around on the slide (Figures II.23 and II.24).

7. *Candida:* appear as branching pseudohyphae or budding yeast (best seen under the KOH slide; Figure II.25).

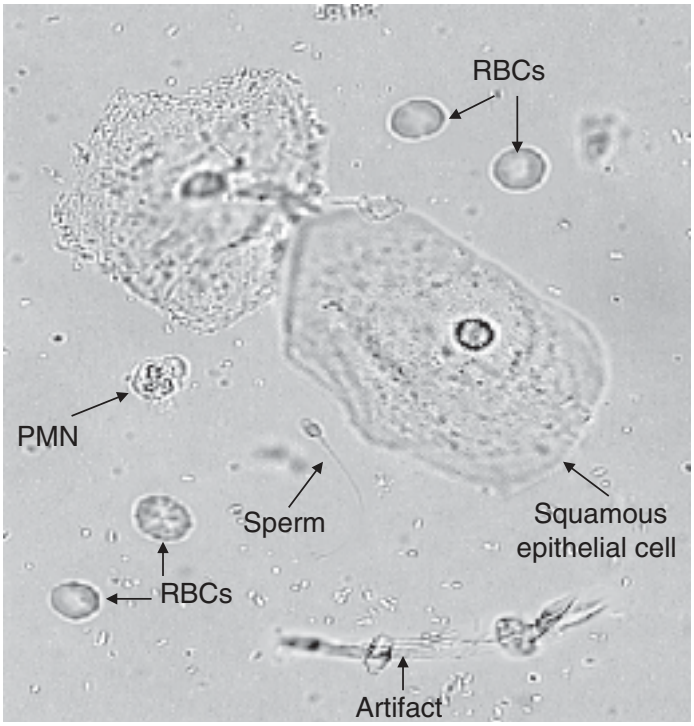


FIGURE II.20 Epithelial cells. PMN, polymorphonuclear neutrophils; RBCs, red blood cells.

Source: Used with permission from the University of Washington STD Prevention Training Center.

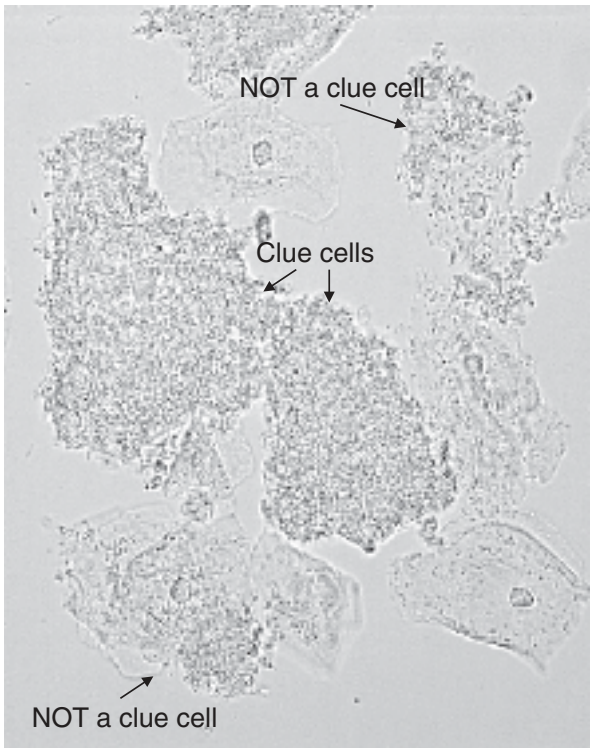


FIGURE II.21 Clue cells.

Source: Used with permission from the University of Washington STD Prevention Training Center.

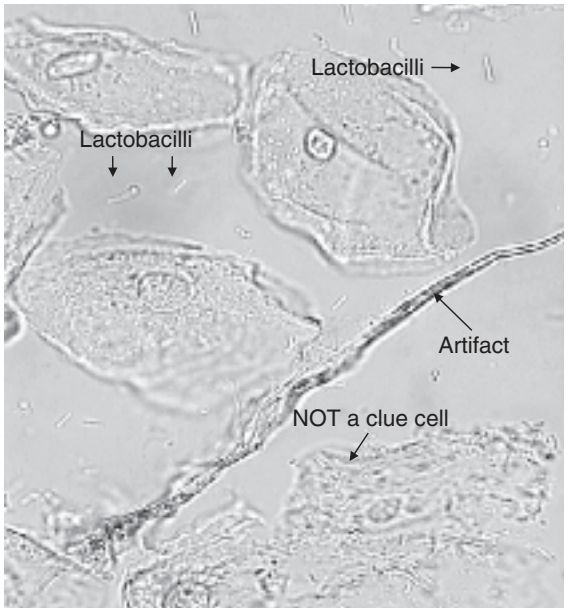


FIGURE II.22 *Lactobacilli.*

Source: Used with permission from the University of Washington STD Prevention Training Center.

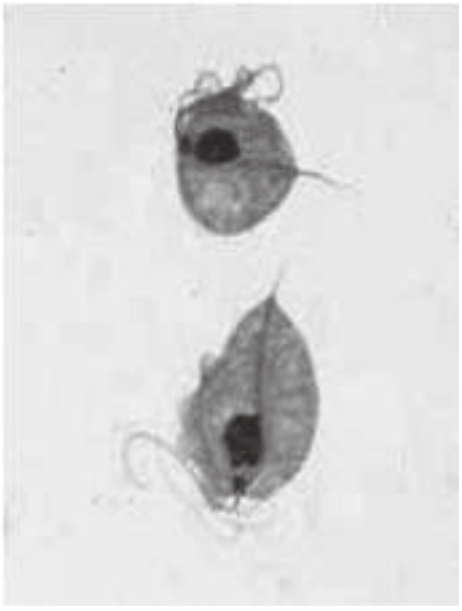


FIGURE II.23 *Trichomonas vaginalis.*

Source: Used with permission from the University of Washington STD Prevention Training Center.

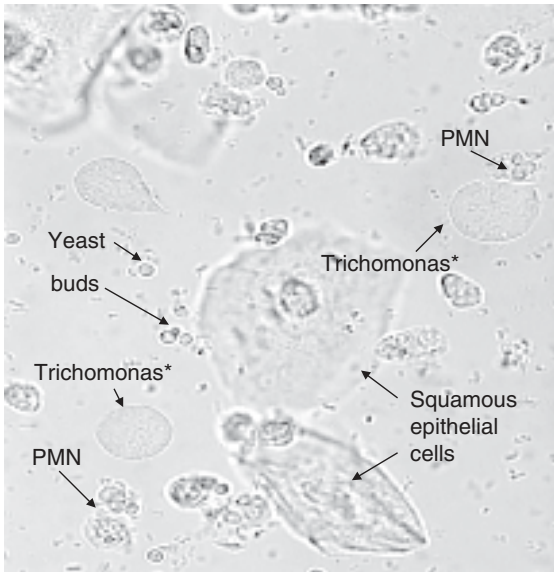


FIGURE II.24 *Trichomonas* (1,000×). PMN, polymorphonuclear neutrophils.

Note: Organism is enlarged.
Source: Used with permission from the University of Washington STD Prevention Training Center.

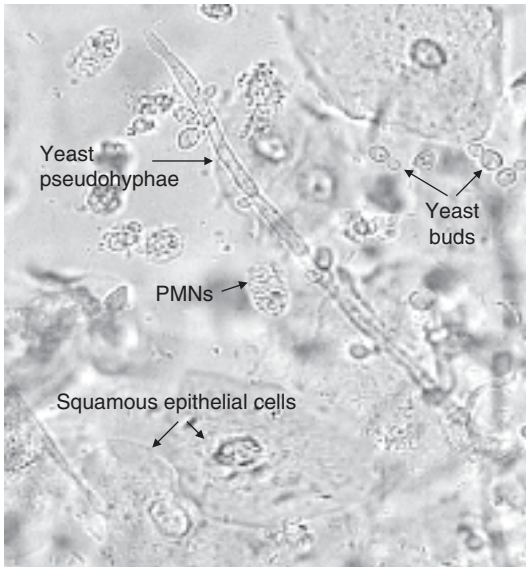


FIGURE II.25 Budding yeast cells, pseudohyphae, and septate hyphae and spores. PMNs, polymorphonuclear neutrophils.

Source: Used with permission from University of Washington STD Prevention Training Center.

TREATMENT

A. Refer to specific chapters for treatment recommendations and medication dosages.

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SECTION III

APPENDICES

A. Normal Laboratory Values
B. Dietary Recommendations (New Section: FODMAP)
C. 2019 Beers Criteria
D. Deprescribing Algorithms

APPENDIX A

NORMAL LABORATORY VALUES

Normal laboratory values are presented here. However, ranges of laboratory value differ from laboratory to laboratory. They differ due to age and sex. Different values are presented; all normal values are listed under the Females column (Table A.1). Values for males are the same unless otherwise noted. Legend: 10^6 = 1,000,000; 10^3 = 1,000.

TABLE A.1 NORMAL LABORATORY VALUES

	Females	Males
Complete blood count		
RBCs	3.9–5.03 10^6/μL	4.3–5.72 10^6/μL
Hgb ↑ polycythemia, dehydration, ↓ blood loss, severe anemia, low production, or death of blood cell	12–15.5 g/dL	13.5–17.58 g/dL
Hct ↓ anemia, massive blood loss, ↑ dehydration, or hemoconcentration with shock	34.9%–44.5%	38%–50%
MCV RBC size: normocytic, microcytic, macrocytic	80–96 fL	
MCH RBC color: normochromic, hypochromic	27–33 pg	
MCHC	33.4–35.5 g/dL or %	
Reticulocyte count: measures the number of new RBCs produced by the bone marrow	33–137 × 10/μL	
WBCs		
↑ Bacterial infections, infectious diseases (mononucleosis), ↓ stress, tissue death, leukemia, cancer, hemorrhage	3.8–11.0 10^3/ mm^3	
Differential		
Segmented neutrophils ↑ stress, trauma, inflammatory disorders, ↓ viral infections, severe bacterial infections, aplastic anemia	50%–81%	
Band neutrophils	1%–5%	
Lymphocytes ↑ chronic bacterial infection, viral infections, ↓ leukemia, AIDS, lupus erythematosus	14%–44%	
Monocytes ↑ chronic inflammatory disorders, viral infections, chronic conditions, ↓ steroid therapy	2%–6%	
Eosinophils ↑ allergies, parasite infections, leukemia	1%–5%	
Basophils ↑ leukemia ↓ allergic reactions, stress, hyperthyroidism	0%–1.0%	
Serum iron concentration of iron bound to transferrin	60–160 mcg/dL	80–180 mcg/dL
TIBC Amount of iron transferrin can bind	250–460 mcg/dL	
ESR	Females <50	Males <50
↑ Pregnancy, inflammatory conditions, ↓ sickle cell anemia	<20 mm/hr Females >50 <30 mm/hr	<15 mm/hr Males >50 <20 mm/hr

(*continued*)

TABLE A.1 NORMAL LABORATORY VALUES (*CONTINUED*)

	Females	Males
Electrolytes		
Calcium	8.8–10.3 mg/dL	
Chloride	95–107 mEq/L	
Magnesium	1.6–2.4 mEq/L	
Phosphate	2.5–4.5 mg/dL	
Potassium	3.5–5.2 mEq/L	
Sodium	135–147 mEq/L	
BUN/Creatinine renal function tests		
BUN > 20 years old	7–30 mg/dL	0.2–0.6 mg/dL
Creatinine	<1.3 mg/dL	
Uric acid	2.5–7.5 mg/dL	4.0–8.5 mg/dL
Creatinine by age and sex • 20–49 y • 50–59 y • 60–69 y • 70–79 y • >80 y	 0.50–1.10 mg/dL 0.50–1.05 mg/dL 0.50–0.99 mg/dL 0.60–0.93 mg/dL 0.60–0.88 mg/dL	 0.60–1.35 mg/dL 0.70–1.33 mg/dL 0.70–1.25 mg/dL 0.70–1.18 mg/dL 0.70–1.11 mg/dL
Thyroid studies		
TSH	2.0–10.5 mcg/mL	
↑ Hypothyroidism, ↓ hyperthyroidism	0.8–1.1 mcg/dL	
T3 ↑ pregnancy and oral contraceptive use	5.0–13.0 mcg/dL	
Thyroxine (T4) ↑ hyperthyroidism, ↓ hypothyroidism		
Aminotransferases		
↑ Liver damage and medications	4–35 U/L	7–46 U/L
ALT	6–18 U/L	7–21 U/L
AST		
Bilirubin (measures bile salt conjugation and excretion)		
Direct bilirubin	0.0–0.2 mg/dL	
Indirect bilirubin	Total bilirubin minus direct bilirubin	
Total bilirubin >20 y	0.1–1.3 mg/dL 3.0 mg/dL indicates hepatic disease, jaundice is visible	

(*continued*)

TABLE A.1 NORMAL LABORATORY VALUES (*CONTINUED*)

	Females	Males
Glucose Fasting 65–100 mg/dL 2-hour postprandial 65–140 mg/dL **Serum albumin (measures protein synthesis)** 3.2–5.0 g/dL ↑ Dehydration, inflammatory illness, liver insufficiency, malnutrition, and cancer **Lipid profile: adults** Fasting cholesterol: Less than 200 mg/dL indicates low risk for CHD 200–239 mg/dL: Borderline risk for CHD >240 mg/dL: High risk for CHD Triglycerides: <140 mg/dL: Low risk for CHD 150–199 mg/dL: Borderline risk for CHD >200 mg/dL: High risk for CHD >500 mg/dL: Very high risk for CHD HDL: females >50 mg/dL; males >4 mg/dL desirable levels higher than 60 mg/dL		
LDL: <100 mg/dL is optimal 100–159 mg/dL: Borderline CHD >160 mg/dL: High risk for CHD >190 mg/dL: Very high risk for CHD **Triglycerides** Ages 0–9 y: <75 mg/dL; ages 10–19 y: <90 mg/dL Ages 0–9 y: 75–99 mg/dL; ages 10–19 y: 90–129 mg/dL borderline risk for CHD Ages 0–9 y: >100 mg/dL; ages 10–19 y: >130 mg/dL high risk for CHD HDL: >45 mg/dL 40–45 mg/dL: Borderline risk for CHD <40 mg/dL: High risk for CHD LDL: <110 mg/dL 110–129 mg/dL: Borderline risk for CHD >130 mg/dL: High risk for CHD		
Coagulation PT: 9–12.5 sec PTT: 20–36 sec Fibrinogen: 160–450 mg/dL Bleeding time: 3–7 min Thrombin time: 11–15 sec Platelets: 140,000–450,000/mL ↓ Thrombocytopenia, acute leukemia/aplastic anemia during chemotherapy, interferon, infections, and drug reactions ↑ Myeloproliferative disease, cancer, and RA **Sickle cell disease** HgbS: Normal, none present Homozygous HgbS: Sickle cell disease Heterozygous HgbS: Sickle cell trait Hemoglobin electrophoresis: Determines hemoglobin types and percentages **Hepatitis B** Hepatitis B surface antigen: Negative Positive: Acute or chronic infection, further liver function tests needed Hepatitis B surface antibody: Positive indicates immunity to infection (previous infection or hepatitis B vaccine) **Serum ALP (marks liver disease)** 40–120 IU/L Pregnancy value 30–200 IU/L ↑ Bile stones, biliary and pancreatic cancer, viral hepatitis, cirrhosis, Paget's disease, osteomalacia, bone metastasis **D-dimer** <250 μ/L: Objective evidence of increased fibrinolysis/evidence of intravascular coagulation and thrombotic disease; routine first-line assessment of clients with a suspected VTE, which can be present as a DVT or PE **Urinalysis** Color: Pale yellow/straw to amber Clarity: Clear Specific gravity: 1.001–1.035 pH: Normal 4.6–8.0 (acidic) Glucose: Absent Ketones: Absent Protein: Negative **Urine dipstick U/A** Nitrites: Negative (positive = bacteria, infection) Leukocytes oxidase: Negative (positive = WBCs)		

(*continued*)

TABLE A.1 NORMAL LABORATORY VALUES (*CONTINUED*)

	Females	Males
Arterial blood gas values pH: 7.35–7.45 $PaCO_2$: 35–45 mmHg HCO_3: 22–26 mEq/L O_2 saturation: 94%–100% PaO_2: 80–100 mmHg BE: −2 to +2 mmol/L **Venous blood gas values** pH: 7.32–7.42 $PaCO_2$: 38–52 mmHg HCO_3: 19–25 mEq/L O_2 saturation: 40%–70% PaO_2: 28–48 mmHg BE: 0 to +4 mmol/L		
HIV ELISA: Negative ELISA: Positive: Repeat; if second ELISA positive, perform western blot Western blot negative: No evidence of HIV Western blot positive: Evidence of HIV **Rubella** Titer: >1:10 immune Titer: <1:7 nonimmune, administer rubella vaccine **Female hormone levels**		

	FSH	LH	Progesterone	Prolactin
Follicular phases	2–25 mIU/mL	5–30 mIU/mL	0.2–6.0 mIU/mL	<23 ng/mL
Midcycle phase	10–90 mIU/mL	75–150 mIU/mL	6–30 mIU/mL	<28 ng/mL
Luteal phase	2–25 mIU/mL	3–40 mIU/mL	5.7–28.1 mIU/mL	5–40 ng/mL
Postmenopausal	40–350 mIU/mL	30–200 mIU/mL	0–0.2 mIU/mL	<12 ng/mL

ALP, alkaline phosphatase; ALT, alanine transaminase; AST, aspartate aminotransferase; BE, base excess; BUN, blood urea nitrogen; CHD, coronary heart disease; DVT, deep vein thrombosis; ELISA, enzyme-linked immunosorbent assay; ESR, erythrocyte sedimentation rate; FSH, follicle-stimulating hormone; Hct, hematocrit; HDL, high-density lipoprotein; Hgb, hemoglobin; HgbS, sickle cell hemoglobin; IU, international unit; LDL, low-density-lipoprotein; LH, luteinizing hormone; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; PE, pulmonary embolism; PT, prothrombin time; PTT, partial thromboplastin time; RA, rheumatoid arthritis; RBC, red blood cells; TIBC, total iron-binding capacity; TSH, thyroid-stimulating hormone; U/A, urinalysis; VTE, venous thromboembolism; WBC, white blood cell.

APPENDIX B

DIETARY RECOMMENDATIONS

- Bland Diet
- DASH Diet: Dietary Approaches to Stop Hypertension
- Foods to Avoid While Taking Warfarin (Coumadin, Jantoven)
- Fermentable Oligosaccharides, Disaccharides, Monosaccharides, and Polyols (FODMAP) Diet
- Gluten-Free Diet
- High-Fiber Diet
- Lactose-Intolerance Diet
- Low-Fat/Low-Cholesterol Diet
- Nausea and Vomiting Diet Suggestions
- Nutrition for Wound Healing
- Food Sources and Recommendations for Common Vitamin and Mineral Deficiencies
- Food Sources and Recommendations for Vitamin D and Calcium

The contributions of Cheryl A. Glass to this appendix in prior editions are acknowledged here.

BLAND DIET

A. You have been prescribed a bland diet. This diet provides adequate nutrition along with the treatment of gastrointestinal problems, such as ulcerative conditions or inflammatory problems of the stomach and intestines. It is intended to decrease irritation in the lining of the stomach and intestines.

FOOD TIPS

FOODS TO AVOID

A. Garlic, onions, alcohol, fatty foods, fried foods, chocolate, cocoa, coffee (even decaffeinated), dried fruits, citrus fruit and juices (orange, pineapple, and grapefruit), tomato products, peppermint, whole grain breads and cereals, prespiced foods such as processed lunch meats and ham, pepper, and chili powder.

GENERAL INSTRUCTIONS

A. Eat at least three small meals a day.
B. Avoid alcohol and beer.
C. Avoid caffeinated drinks/colas.
D. Avoid fried, greasy foods.
E. Bake or broil your foods.
F. Trim the fat from meats before cooking.
G. Bake, broil, mash, or cream potatoes.
H. Avoid raw fruits and vegetables such as corn on the cob and other gas-forming vegetables such as cabbage, dried beans, and peas.
I. Avoid rich desserts.
J. Avoid bedtime snacks—they may increase acid production and cause discomfort at night.
K. Avoid eating 2 hours before you go to bed.
L. Ask your healthcare provider if nutritional supplements are necessary.

APPROVED FOODS BY FOOD GROUP

A. Dairy products:
  1. Whole milk.
  2. Low fat or 2% milk.
  3. Skim milk.
  4. Evaporated milk.
  5. Buttermilk.
  6. Cottage cheese.
  7. Yogurt.
  8. Cheese (avoid stronger cheeses that are darker yellow, and blue cheese).

B. Meat:
  1. Beef.
  2. Veal.
  3. Fresh pork.
  4. Turkey.
  5. Chicken.
  6. Fish (canned or fresh).
  7. Liver.
  8. Egg (as a meat substitute).
  9. Tofu.
  10. Tempeh soy product.

C. Breads/grains:
  1. Enriched breads (plain toast).
  2. Oats.
  3. Cereal.
  4. Tortillas.
  5. English muffins.
  6. Saltine crackers.
  7. Pasta (all types).

D. Fruits/vegetables:
  1. All vegetables.
  2. All fruits and juices (except citrus).

E. Desserts:
  1. Custard.
  2. Pudding.
  3. Sherbet.
  4. Ice cream (except peppermint and chocolate).
  5. Gelatin.
  6. Angel food cake.
  7. Pound cake.
  8. Sugar cookies.
  9. Jams and jellies.
  10. Honey.

F. Drinks:
  1. Decaffeinated tea.
  2. Juices (except citrus).
  3. Caffeine-free sodas.

G. Spices:
  1. Salt.
  2. Thyme.
  3. Sage.
  4. Cinnamon.
  5. Paprika.
  6. Apple cider vinegar.
  7. Prepared mustard.
  8. Lemon and lime juices.

DASH DIET: DIETARY APPROACHES TO STOP HYPERTENSION

A. The Dietary Approaches to Stop Hypertension (DASH) diet is based on a combination of different types of foods and is recommended to help control high blood pressure. It is a food plan that is based on foods that are low in cholesterol and high in dietary fiber, potassium, calcium, and magnesium; it is moderately high in protein. The DASH eating plan is high in vegetables, fruits, low-dairy products, whole grains, poultry, fish, beans, and nuts. DASH eating has a reduction in lean red meats, added sugar, and sugar-containing sodas. The DASH diet's dairy food portions make the diet high in calcium and vitamin D. Overall, Americans, especially African Americans, are deficient in vitamin D.

B. The DASH diet, along with weight loss and exercise, is used to control other health problems such as type 2 diabetes and heart disease. If your ethnic background is Hawaiian, Native American, Eskimo, Hispanic, or African American, you are at higher risk for high blood pressure. Following a DASH eating plan also will help lower the bad cholesterol, or low-density lipoprotein (LDL), which can reduce heart disease.

C. A major way the DASH diet helps lower blood pressure is to limit the amount of salt (sodium). The recommendations are limiting sodium to 2,300 mg a day or less. If you have high blood pressure, you may be limited to 1,500 mg or less a day. **One teaspoon of salt contains 2,000 mg of sodium.**

D. Foods high in sodium make you retain extra fluid. If you notice that your hands are swelling (rings are tight) or your feet and legs swell (sock rings or shoes feel tight), you are getting too much salt. Fluid retention is bad if you have heart failure. This diet is rich in potassium, which can help you get rid of the extra sodium and decrease the fluid retention.

E. Sea salt, even though it may contain less sodium, is not low enough in sodium to use as a substitute for regular salt. Salt substitutes contain potassium chloride; this can cause more fluid retention. Potassium chloride salt substitutes may also interfere with your blood pressure medications, so check with your healthcare provider before using them.

TIPS FOR REDUCING SODIUM

A. Slowly cut back on your salty foods and begin to use healthier products. Take the salt shaker off the table.

B. Eat fresh foods:
- **1.** Avoid prepackaged foods.
- **2.** Eat fresh vegetables instead of canned vegetables. If you use canned vegetables, choose the low-sodium option and/or rinse the vegetables.

C. Read food labels:
- **1.** Sodium is in almost all processed foods, including milk. Sodium is listed different ways on labels; see Table B.1.
- **2.** Focus on the amount of sodium per food serving.
- **3.** Do not forget to read labels on soda and sports drink bottles.
- **4.** Choose your favorite food brand with low-salt or low-sodium labels on the package instead of the same product with more salt.

D. Avoid salty snacks and foods, including the following:
- **1.** Crackers, chips, and pretzels.
- **2.** Cheeses.
- **3.** Olives, pickles, pickled okra, and other salty foods.
- **4.** Processed foods, including jerky, hot dogs, bacon, deli meats, canned fish, and canned meats, which contain a large amount of sodium.

E. Limit using soy sauce, seasoned salts, and meat tenderizers.

F. Many seasonings, including ketchup and sauces, contain a lot of sodium. Substitute with other flavors such as fresh herbs (e.g., rosemary, thyme, oregano, cilantro, and basil). Use garlic powder, lemon and lime juice, and crushed red peppers, as well as ginger.

G. Try making your own salt-free herb blend to use on your foods. Ingredients that can add flavor without adding salt include the following:
- **1.** Peppers such as cayenne, black pepper, and lemon pepper.
- **2.** Dried herbs such as thyme.
- **3.** Garlic powder.
- **4.** Paprika.
- **5.** Celery seed.

H. Helpful websites for salt-free herb blends, products, and recipes are the following:
- **1.** Salt-free seasoning recipes:
  - a. www.tasteofhome.com/Recipes/Salt-Free-Seasoning-Mix.
- **2.** Websites with low-salt recipes:
  - a. Mrs. Dash: www.mrsdash.com/recipes.
  - b. Foodnetwork: www.foodnetwork.com/topics/low-sodium.
  - c. Mayo Clinic: www.mayoclinic.org/healthy-lifestyle/recipes/low-sodium-recipes/rcs-20077197.
- **3.** DASH Diet Food Tracker Apps are available for download in the Apple App Store.

TIPS ON EATING THE DASH WAY

A. Start small. Make gradual changes in your eating habits, such as eating smaller portions (Tables B.2 and B.3).

B. Center your meal around carbohydrates, such as pasta, rice, beans, or vegetables.

C. Treat meat as only part of a whole meal instead of the main focus of the meal.

D. Use fruits or low-fat, low-calorie foods such as sugar-free gelatin for desserts and snacks.

E. Choose "whole" grains in breads and cereals.

F. Choose to eat vegetables without butter or sauce.

G. Choose lean cuts of meat. Use fresh poultry, for example, skinless turkey and chicken.

H. Choose ready-to-eat breakfast cereals that are lower in sodium.

I. Eat fruits for dessert. Use fruits that are canned in their own juice.

J. Add fruit to plain yogurt.

K. To increase eating vegetables, stir-fry with 2 oz of chicken and use 1 cups of raw vegetables.

L. Snack on vegetables, bread sticks, graham crackers, or unbuttered/unsalted popcorn.

M. Drink water or club soda.

N. Eating out:
- **1.** Ask the restaurant to prepare your meal without salt, if possible.
- **2.** Review the menu to avoid foods that are listed as pickled, cured, or smoked, or contain soy sauce or broth.

TABLE B.1 WAYS SODIUM IS LISTED ON A FOOD LABEL

Phrase on Label	Meaning of Sodium Content
Sodium-free or salt-free	<5 mg per serving
Very low sodium	35 mg or less of sodium per serving
Low sodium	140 mg or less of sodium per serving
Low-sodium meal	140 mg or less sodium per 3.5 oz (100 g)
Reduced or less sodium	At least 25% less sodium than the regular version
Light in sodium	50% less sodium than the regular version
Unsalted or no salt added	No salt added to the product during processing (this is not a sodium-free food)

Source: Adapted from U.S. Food and Drug Administration. (2018). Use the nutrition facts label to reduce your intake of sodium in your diet. https://www.fda.gov/media/84262/download.

TABLE B.2 SERVING PORTION SIZES

Food	Portion Size	Food	Portion Size
1 baked potato/1 cup of flaked cereal	Fist	½ cup of fresh fruit (of baseball); cup of pasta, rice, or potato (of baseball)	Baseball
1½ cheese (4 dice) ½ tsp of margarine (1 die)	Dice	½ cup of ice cream	
¼ cup of raisins	Egg	3 oz of meat, includes fish, meat, and chicken	Deck of cards
½ cup almonds	Golf ball	1 pancake	Compact disc
2 tbsp peanut butter	Ping-pong ball	1 piece of cornbread	Bar of soap
1 cup of salad/1 cup of popcorn (unbuttered)	Baseball	3 oz of grilled or baked fish	Checkbook

Source: Adapted from National Heart, Lung, and Blood Institute. (2009). *Aim for a healthy weight: Keep an eye on portion size*. https://www.nhlbi.nih.gov/files/docs/public/heart/PortionSize_ZCard_taggd.pdf.

TABLE B.3 DASH DIET DAILY SERVINGS

Food Group	Daily Servings	Serving Sizes	Examples and Notes	Significance of Each Food Group to the DASH Diet Pattern
Grains and grain product	6–8	1 slice bread; cup dry cereal; cup cooked rice, pasta, or cereal	Whole wheat bread, English muffin, pita bread, bagel, cereals, grits, oatmeal, couscous	Major sources of energy and fiber
Vegetables	4–5	1 cup raw leafy vegetables; cup cooked vegetables; 6 oz vegetable juice	Tomatoes, potatoes, carrots, peas, squash, broccoli, turnip greens, collards, kale, spinach, artichokes, beans, sweet potatoes	Rich sources of potassium, magnesium, and fiber
Fruits	4–5	6 oz fruit juice; 1 medium fruit; ¼ cup dried fruit; ¼ cup fresh, frozen, or canned fruit	Apricots, bananas, dates, grapes, oranges, orange juice, grapefruit, grapefruit juice, mangoes, melons, peaches, pineapples, prunes, raisins, strawberries, tangerines	Important sources of potassium, magnesium, and fiber

(*continued*)

TABLE B.3 DASH DIET DAILY SERVINGS (*CONTINUED*)

Food Group	Daily Servings	Serving Sizes	Examples and Notes	Significance of Each Food Group to the DASH Diet Pattern
Low-fat or fat-free milk and dairy foods	2–3	1 cup milk; 1 cup yogurt; 1.5 oz cheese	Skim or 1% milk, skim or low-fat buttermilk, nonfat or low-fat yogurt, part-skim mozzarella cheese, nonfat cheese	Major sources of calcium and protein
Lean meats, poultry, and fish	6 or less	1 oz cooked meats, poultry, or fish; 1 egg	Select only lean; trim away visible fat; broil, roast, or boil instead of frying; remove skin from poultry	Rich sources of protein and magnesium
Nuts, seeds, and legumes	4–5 per week	½ oz of nuts; ½ oz or 2 tbsp seeds; 2 tbsp of peanut butter; ½ cup cooled legumes	Almonds, filberts, mixed nuts, peanuts, walnuts, sunflower seeds, kidney beans, lentils	Rich sources of energy, magnesium, potassium, protein, and fiber
Sweets and added sugars	5 or less per week	½ tbsp sugar; 1 tbsp jelly or jam; ½cup sorbet or gelatin; 1 cup lemonade	Fruit-flavored gelatin, fruit punch, hard candy, jelly, maple syrup, sugar	Sweets should be low in fat

DASH, Dietary Approaches to Stop Hypertension.

Source: Adapted from National Heart, Lung, and Blood Institute. (n.d.). DASH eating plan. https://www.nhlbi.nih.gov/health-topics/dash-eating-plan.

3. Limit condiments, including mustard, ketchup, and pickles.

O. Table B.4 presents the number of servings, and Table B.5 illustrates the caloric adjustment for 2,000 calories a day using the DASH diet.

TABLE B.4 TOTAL NUMBER OF SERVINGS IN 2,000 CAL/D

Food Group	Servings
Grains	6
Vegetables	5
Fruits	4
Dairy: fat-free or low-fat milk	3
Poultry, meat, and eggs	8–9 per week
Fish and other seafood	2–3 servings a week
Nuts, seeds, and legumes	1
Fats and oils	3
Sweets and added sugars	1

Source: Adapted from National Heart, Lung, and Blood Institute. (n.d.). DASH eating plan. https://www.nhlbi.nih.gov/health-topics/dash-eating-plan.

RESOURCES

DASH Diet Calorie Adjustments for 1,200, 1,600, 2,000, and 2,400 Calorie Diets: dashdiet.org/images/calories.pdf

DASH Diet Eating Plan: dashdiet.org/dash_diet_recipes.asp

DASH Diet Recipes: www.mayoclinic.com/health/dash-diet-recipes/RE00089

DASH for Health: www.dashforhealth.com

TABLE B.5 DASH EXAMPLE MENU (2,000 CAL)

Food	Amount	Servings Provided
Breakfast		
Orange juice	6 oz	1 fruit
1% low-fat milk	8 oz (1 cup)	1 dairy
Cornflakes (with 1 tsp sugar)	1 cup	2 grains
Banana	1 medium	1 fruit
Whole wheat bread (with 1 tbsp jelly)	1 slice	1 grain
Soft margarine	1 tsp	1 fat
Lunch		
Chicken salad	¾ cup	1 poultry
Pita bread	½ large	1 grain
Raw vegetable medley:		
Carrot and celery sticks	3–4 sticks each	
Radishes	2	1 vegetable
Loose-leaf lettuce	2 leaves	
Part-skim mozzarella cheese	1.5 slice (1.5 oz)	1 dairy
1% low-fat milk	8 oz (1 cup)	1 dairy
Fruit cocktail in light syrup	½ Cup	1 fruit
Dinner		
Herbed baked cod	3 oz	1 fish
Scallion rice	1 cup	2 grains
Steamed broccoli	½ cup	1 vegetable
Stewed tomatoes	½ cup	1 vegetable
Spinach salad:		
Raw spinach	½ cup	
Cherry tomatoes	2	1 vegetable
Cucumber	2 slices	
Light Italian salad dressing	1 tbsp	½ fat
Whole wheat dinner roll	1 small	1 grain
Soft margarine	1 tsp	1 fat
Melon balls	1/2 cup	1 fruit
Snacks		
Dried apricots	1 oz (¼ cup)	1 fruit
Mini-pretzels	1 oz (¾ cup)	1 grain
Mixed nuts	1.5 oz (cup)	1 nuts
Diet ginger ale	12 oz	0

DASH, Dietary Approaches to Stop Hypertension.

Source: National Heart, Lung, and Blood Institute. (2018). DASH eating plan. https://www.nhlbi.nih.gov/health-topics/dash-eating-plan.

FOODS TO AVOID WHILE TAKING WARFARIN (COUMADIN, JANTOVEN)

A. Warfarin is a medication that thins the blood to prevent blood clots. Some nutrients and foods can interfere with warfarin and make the warfarin less effective or not work as well. It is important to closely monitor the foods that you eat daily to make sure your medication is working for you the same way every day. One nutrient that causes this problem is vitamin K. Vitamin K interferes with the warfarin and can cause problems with thinning the blood. Therefore, it is important to avoid foods and nutrients that would cause this problem. It is suggested to eat foods that contain the same amount of vitamin K every day. This will help to prevent problems with thinning your blood.

B. The recommended intake of vitamin K for the adult male is 120 mcg and for adult female is 90 mcg.

A. The following is a list of foods that have a **MODERATE to HIGH level of vitamin K and should be avoided:**

1. Broccoli (cooked).
2. Brussels sprouts.
3. Chard.
4. Collard greens.
5. Green tea.
6. Kale.
7. Mustard greens.
8. Parsley.
9. Spinach.
10. Turnip greens.

B. Some drinks can also interfere with warfarin and **should be avoided**. These drinks include the following:

1. Cranberry products (can increase risk of bleeding).
2. Alcohol (can increase risk of bleeding).
3. Grapefruit juice (can increase risk of bleeding).
4. Green tea (lowers the effectiveness of warfarin).

C. Foods that have a **LOWER level of vitamin K** and are safer to eat in moderate portions include the following:

1. Asparagus.
2. Avocado.
3. Blackberries/blueberries.
4. Cabbage.
5. Carrots.
6. Cauliflower.
7. Cucumbers.
8. Lettuce, iceberg, and romaine.
9. Peas.
10. Peppers.
11. Potatoes.
12. Prunes.
13. Squash (summer and winter).
14. Sweet potatoes.
15. Tomatoes.
16. Tuna.

D. Medications (prescribed and over-the-counter [OTC] medications, multivitamins, supplements, and herbal supplements) may also interfere with warfarin and should only be taken after discussing the medication, vitamin, or supplement with your healthcare provider for safety.

E. If you have questions about certain foods or medications, you need to discuss this with your healthcare provider.

FOODS RICH IN VITAMIN K

A. If you are taking a blood thinner, you should be aware that certain foods are high in vitamin K.

B. Vitamin K can interfere with how blood thinners work.

C. You do not necessarily have to stop eating these foods, just be consistent and maintain your regular eating habits.

D. Please discuss these foods further with your healthcare provider.

1. **Vitamin K-rich foods include the following:**
   a. Broccoli.
   b. Brussels sprouts.
   c. Collard greens.
   d. Endive.
   e. Kale.
   f. Mustard greens.
   g. Parsley.
   h. Swiss chard.
   i. Spinach.
   j. Turnip greens.
2. **Foods that are low in vitamin K:**
   a. Asparagus.
   b. Avocado.
   c. Blackberries.
   d. Blueberries.
   e. Cabbage.
   f. Cranberry juice.
   g. Green tea.
   h. Lettuce (iceberg and leafy).
   i. Liver.
   j. Peas.
   k. Prunes.
   l. Tuna.

FERMENTABLE OLIGOSACCHARIDES, DISACCHARIDES, MONOSACCHARIDES, AND POLYOLS (FODMAP) DIET

A. Irritable bowel syndrome (IBS) symptoms might be better following a strict low-fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (FODMAP) diet and then reintroducing foods one at a time. You have been recommended to follow a change in your diet by decreasing carbohydrates such as fructose, fructans, lactose, and others called FODMAPs. FODMAPs are also found in certain grains, vegetables, fruits, and dairy product.

GENERAL INSTRUCTIONS

A. Keep a food diary of what you are eating and how you feel.

B. When your IBS symptoms are getting better, reintroduce one food at a time to see if it makes your IBS symptoms come back or get worse:

1. After you feel that the new food does not cause increased IBS symptoms, try a second food.

2. Foods that make your IBS symptoms worse should continued to be avoided.

LOW FODMAP FOODS (GOOD TO EAT)

A. Vegetables (bean sprouts, kale, tomato, zucchini, spinach, eggplant, carrots).

B. Legumes (eggs, nuts, seeds, tofu).

C. Fruits (higher glucose than fructose). Eat only 1 serving per meal, 3 oz (bananas, blueberries, pineapple, strawberries, oranges, mandarins, kiwi).

D. Meats, poultry, and meat substitutes.

E. Fish and seafood.

F. Cereals, grains, breads, biscuits, pasta, nuts, and cakes (brown rice, oats, polenta, quinoa, and tapioca).

G. Condiments, dips, sweets, sweeteners, and spreads (glucose, maple syrup, sugar, artificial sweeteners including aspartame, saccharin, and stevia).

H. Dairy foods and eggs (cheddar cheese, feta cheese, Parmesan cheese, cream, lactose-free milk).

I. Beverages (black tea, coffee, gin, green tea, vodka, water, white tea)

HIGH FODMAP FOODS (AVOID EATING THESE FOODS)

A. Wheat (main ingredient in food), barley, and rye.

B. Garlic.

C. Onions.

D. Fruits (higher in fructose than glucose, including apples, apricots, peaches, pears, watermelon, plums, nectarines, mangoes, cherries).

E. Vegetables (asparagus, Brussels sprouts, artichokes, leeks, mushrooms, snow peas, cauliflower).

F. Legumes (baked beans, black-eyed peas, butter beans, kidney beans, lentils, soybeans, split peas).

G. Sweeteners (agave nectar, high-fructose corn syrup, honey, sugar-free gum/mints).

H. Dairy (cottage cheese, cream cheese, milk, ricotta, yogurt).

I. Beverages (chai tea, chamomile tea, coconut water, dessert wine, and rum).

GLUTEN-FREE DIET

GLUTEN-FREE DIET TIPS

A. The symptoms from celiac disease are triggered from glutens in your diet. Three cereals that contain gluten are wheat, rye, and barley. Glutens are also in other products, such as food additives, so it is **very important to read all the ingredients on food labels** (Table B.6).

DIETARY RECOMMENDATIONS FOR CELIAC DISEASE

A. You may also be told to follow a lactose-free diet for a short time to alleviate your symptoms.

B. Most bread sold in the grocery aisle is not allowed on a gluten-free eating plan.

C. All vegetables and fruits are gluten-free. However, frozen and canned fruits and vegetables may contain an additive with gluten.

D. Although you should not enjoy beer, wine is still on the menu when you go to dinner.

E. Specialty bakeries are able to make gluten-free cakes for special occasions. Plain hard candy, marshmallows, and other candies are usually gluten-free.

F. Caution should be taken when ordering any breaded foods such as chicken nuggets or breaded fish.

G. Deli meats may also contain gluten.

H. Gluten-free foods are often not fortified with vitamins and minerals. It is recommended to take a daily multivitamin.

GLUTEN-FREE RECIPES

A. Gluten-Free Recipes: allrecipes.com/Recipes/Healthy-Cooking/Gluten-Free/Main.aspx.

B. Betty Crocker: www.bettycrocker.com/Search?term=gluten%20free.

C. Chex: www.chex.com/gluten-free-recipes.

RESOURCES

Celiac Disease Foundation: www.celiac.org
www.celiac.com

TABLE B.6 EXAMPLES OF FOODS THAT ARE ALLOWED AND TO BE AVOIDED ON A GLUTEN-FREE DIET

Allowed	Avoid
Fresh fruits and vegetables without any processing or additives	Wheat, wheat berry, wheat bran, wheat germ, wheat grass, whole wheat berries
Meat	Flours, bread flour
Soy, soybean, tofu	Bulgur (bulgur wheat, bulgur nuts)
Brown rice	Rye
Enriched rice/instant rice/ wild rice	Barley
Buckwheat	Barley malt/barley extract
Millet	Oats, oat bran, oat fiber
Sorghum	Cereals
Alfalfa	Matzo
Almond	Beer, ale, porter, stout
Canola	Farina
Chickpea	Croutons
Corn, corn flour, cornmeal	Bran
Brown rice flour	Tabbouleh
Tapioca	Soy sauce
Coffee with no grains added	Bouillon cubes
Milk	Cold cut meats
Wine (check for preservatives and dyes)	Energy bars
Gluten-free beers	Instant coffee and teas
	Malted milk
	Malted wine coolers
	Beer, ales, and lagers

HIGH-FIBER DIET

A. Fiber is a plant cell-wall component that is not broken down by the digestive system. An old term for fiber is "roughage" because it absorbs fluid and moves waste faster through the intestines in a bulky mass. The Food and Drug Administration defines as high-fiber those products that contain 25% of the daily fiber value. High-fiber diets are used to help prevent constipation as well as diarrhea. Fiber has been used to help several medical conditions such as diabetes, diverticulosis, IBS, and high cholesterol, as well as weight loss. If you have a chronic health condition, check with your healthcare provider about starting any new dietary change.

B. Fiber provides a full feeling that can help with spacing meals further apart (3–4 hours). Fiber recommendations change with your age (Table B.7).

TIPS FOR INCREASING FIBER IN YOUR DIET

A. Read the Nutrition Facts food labels for fiber content per food serving:

1. Cereals that provide 5 g of fiber per serving give you 20% of your daily fiber.

2. Look for "whole grain" on the label. Just because bread is brown does not mean it is whole grain.

B. Increase fiber in your diet gradually to prevent gas. Adding too much fiber too quickly may give you abdominal pain, bloating, and constipation. Increase your fiber over several weeks so that it gives time for you to adjust.

C. As you increase fiber, it is also important to increase the amount of fluids you drink up to six to eight glasses a day, including tea, milk, fruit juices, coffee, and even soft drinks. The extra fluids that you drink along with the extra fiber makes you feel fuller, which can help control snacking.

D. Keep a food diary and review it periodically to decide on other diet adjustments that may need to be made.

E. Several fiber supplements are available over the counter (OTC) to help you get your daily recommendation of fiber.

GOOD FOOD SOURCES OF FIBER

A. Bran: Add one teaspoon of whole grain bran to food three times a day or take an OTC fiber supplement such as psyllium (Metamucil), as directed.

B. Whole grain cereals and breads: Eat oat, bran, multigrain, light, wheat, or rye breads rather than pure white bread or breads that list eggs as a major ingredient. Grains are not only a good source of fiber but also contain vitamins and minerals. Folic acid has been added to breads and cereals to help reduce neural tube defects.

C. Fresh or frozen fruits and vegetables: Citrus fruits are especially good sources of fiber. Eat raw or minimally cooked vegetables, especially squash, cabbage, lettuce, other greens, and beans. Leave the skins on fruits and vegetables; eating the whole fruit is better than drinking the juice. Whole tomatoes offer more fiber than peeling the skin off. The more colorful the fruit and vegetable (dark green, reds, blue, yellow) the better; they provide a good source of antioxidants that are good for the heart and the prevention of some cancers. Apples are a good source of both fiber and water.

D. Legumes (pods): Peas and beans are a good source of fiber. Add chickpeas and kidney beans to salads for extra fiber and flavor. Add baked beans as a delicious side item to your meal.

E. Coffee is another source of fiber.

F. Nuts are an excellent source of fiber. They are considered nutrient-dense and are a good source of vitamins and folic acid. Sprinkle sunflower seeds on a salad to add flavor and fiber. The amount of nuts eaten should be limited to 1 to 2 oz because they are also high in calories.

G. If you have diverticulosis, avoid foods with seeds or indigestible material that may block the neck of a diverticulum, such as nuts, corn, popcorn, cucumbers, tomatoes, figs, strawberries, and caraway seeds.

TABLE B.7 FIBER RECOMMENDATIONS BY AGE

Group/ Sex	Age	Fiber Recommendations
Male	19–50 y	38 g of fiber each day
Female	19–50 y	25 g of fiber each day
Male	≥51 y	30 g of fiber each day
Female	≥51 y	21 g of fiber each day
Male	>70 y	30 g of fiber each day
Female	>70 y	21 g of fiber each day

Source: Adapted from Institute of Medicine. (2005). *Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids*. National Academies Press. https://www.nap.edu/read/10490/chapter/9#389.

LACTOSE-INTOLERANCE DIET

A. Lactose is the sugar present in milk. Lactose intolerance is very common; it occurs when the body is not able to appropriately digest the milk sugar content and the result is diarrhea. You have been diagnosed as having difficulty digesting milk (lactose) products or you have problems with malabsorption. Lactose intolerance can cause gas, bloating, abdominal cramps, diarrhea, and nausea or vomiting. You may be able to eat small portions without problems, or you may be unable to tolerate any foods containing lactose (Table B.8).

B. Lactose intolerance with milk products often causes a problem with not getting enough calcium and vitamin D that are needed for bones and other health needs.

C. Lactose-free food is available in most grocery stores.

D. Keeping a food diary is important to see what kinds of foods cause the symptoms of stomach pain, diarrhea, gas, and bloating. Some symptoms start after 30 minutes of eating and other symptoms may not be felt for 2 hours:

1. You may be instructed to take out all milk and lactose foods and slowly restart them.

2. Yogurt may be better than milk.

3. Small amounts of cheddar cheese on crackers may be tried.

4. Small amounts of milk in cereal may be tolerated.

E. There are lactase medicines available that can be bought in grocery and drug stores.

TIPS FOR FOLLOWING A LACTOSE-INTOLERANT DIET

A. Limit or omit foods that contain milk, lactose, whey, or casein.

B. Lactose-controlled diets allow up to 1 cup of milk per day for cooking or drinking, if you can tolerate it.

C. Read labels carefully. If you cannot tolerate any lactose, choose lactose-free foods with lactate, lactic acid, lactalbumin, whey protein, sodium caseinate, casein hydrolysates, and calcium compounds.

D. You may also choose kosher foods marked "pareve" or "parve," which do not contain lactose. Read labels carefully.

E. A low-fat diet is also important if you have fat malabsorption.

F. To help with diarrhea caused by malabsorption, avoid more than one serving a day of caffeine-containing drinks.

G. Beverages with high sugar content, such as soft drinks and fruit juices, may increase diarrhea. Juices and fruits with high amounts of fructose include apples, pears, sweet cherries, prunes, and dates.

H. "Sugarless" sorbitol-containing candies and gums may cause diarrhea.

TABLE B.8 RECOMMENDED FOODS FOR LACTOSE INTOLERANCE

Foods	Recommended	Not Recommended
Milk	Soybean milk, milk treated with lactase, nondairy creamers, whipped topping; up to 1 cup/d of buttermilk, yogurt, sweet acidophilus milk, and whole, low-fat, or skim milk	Milk products in excess of 1 cup/d, malted milk, milk shakes, hot chocolate, cocoa
Meat and protein foods	All meats, fish, poultry, eggs, peanut butter, tofu, and hard, aged, and processed cheese, if tolerated	Sandwich meat, hot dogs that contain lactose, cottage cheese, any meat prepared with milk products
Vegetables	All fresh, frozen, canned, buttered, and/or breaded vegetables	Any vegetable prepared with milk or milk products in excess of allowance (1 cup)
Fruits	All fresh, frozen, or canned fruits	Any fruits processed with lactose
Breads, cereal, and starchy food	White, wheat, rye, or other yeast breads, crackers, macaroni, spaghetti, popcorn, dry or cooked cereals	Commercial bread products (French toast, bread mixes, pancakes, biscuits), cakes/cookies containing milk or milk products
Fats and oils	Butter/margarine, salad dressing, mayonnaise, all oils, nondairy creamers, bacon	Sour cream, salad dressings with milk products in excess of 1 cup allowance
Soups	Vegetable and meat soups, broth, and bullion	Dried soups, creamed soups made with milk
Desserts	Plain and fruit-flavored gelatins, sherbet, fruit pies, cakes, pudding, pastries, angel food cake, sponge cake	Ice cream, ice milk, cream pie, puddings, custards, cakes, and pastries with milk (unless counting as day's allowance)
Beverages	Coffee, tea, soft drinks, fruit juices, carbonated and mineral waters	Beverages with milk over the 1 cup allowance
Miscellaneous condiments	Catsup, mustard, soy sauce, vinegar, steak sauce, Worcestershire sauce, chili sauce	None
Seasonings	Salt, pepper, spices, herbs, and seasonings	None
Sweets	Sugar, jelly, honey, molasses, preserves, marmalade, syrups, hard candy, baker's cocoa, carob powder, artificial sweeteners	Cream or chocolate candies containing milk or milk products (unless counting as day's allowance), caramels, toffee, and butterscotch

Source: Cash, J. C., and Glass, C. A. (2021). *Family practice guidelines* (5th ed.). Springer Publishing Company.

LOW-FAT/LOW-CHOLESTEROL DIET

A. The connection between fat in the diet and heart attack is cholesterol. Cholesterol is a fat-like substance produced by your liver and also found in many foods. Too much cholesterol causes heart attacks by clogging the arteries that deliver blood to your heart.

B. Exercising and following a low-fat/low-cholesterol diet can help control your blood cholesterol and reduce your risk of heart attack.

TIPS

A. Read food labels. Use the ingredient list on labels to identify products containing saturated fat. High-fat ingredients may have many names. Remember: Foods can say "no cholesterol" and still be high in saturated vegetable fat and calories.

B. Train yourself to think "low fat" in your food and use the following cooking methods:

1. Bake.
2. Broil.
3. Grill.
4. Stir-fry.

C. Eat less fried food, fast food, and baked products.

D. Eat more fruits and vegetables.

E. Organize your shopping around low-fat foods.

F. Add flavor to foods by using herbs and spices instead of butter and sauces.

G. Choose coleslaw, sliced tomatoes, or a dill pickle instead of fries and chips.

FOODS TO AVOID OR LIMIT

A. Proteins/meats:

1. Shrimp.
2. Fried meats, fish, or poultry.
3. Fatty ground meat.
4. Prime or heavily marbled meats.
5. Bacon, sausage, high-fat deli meats, and cheeses.
6. Liver and organ meats.

B. Breads/cereals:

1. High-fat baked foods, such as Danish pastries, croissants, and doughnuts.
2. Fried rice, crispy chow mein noodles.
3. Granola bars with coconut or coconut oil.
4. Chips, cheese, or butter crackers.
5. High-fat cookies and cakes.

C. Fruits and vegetables:

1. Coconut.
2. Fried vegetables such as onion rings and breaded fried pickles, mushrooms, and okra.
3. Cream, cheese, or butter sauces on vegetables.

D. Milk/dairy products:

1. Whole or 2% milk.
2. Cream, half-and-half, nondairy creamers.
3. Ice cream, whipped cream, nondairy whipped toppings.
4. Whole milk yogurt, sour cream.
5. Cheeses: cheddar, American, Swiss, cream cheese, Brie, Muenster.

E. Very high-fat foods:

1. Butter or margarine made with partially hydrogenated oils.
2. Lard, meat fat, and coconut or palm oils.
3. Salad dressings made with sour cream or cheese.
4. Chocolate.
5. Beef tallow.
6. Hydrogenated or partially hydrogenated vegetable shortening.
7. Cream.
8. Cocoa butter.

FOODS THAT ARE ALLOWED

A. Proteins/meats:

1. Fish and shellfish.
2. Chicken and turkey cooked without the skin.
3. Ground turkey.
4. Eggs: limited to two yolks per week.
5. Dried beans, lentils, tofu.
6. Small amounts of meat and seafood.

B. Breads/cereals:

1. Plain bread and English muffins and bagels.
2. Plain pasta, rice.
3. Cereals, oatmeal.
4. Pretzels, air-popped popcorn, rice cakes, Melba toast.
5. Low-fat baked goods: angel food cake, graham crackers, fruit cookies, and gingersnaps.

C. Fruits and vegetables:

1. Eat several servings per day of high-nutrition, low-fat fruits and vegetables.
2. Prepare vegetables by steaming, broiling, baking, or stir-fry.

D. Milk/dairy products:

1. Skim or 1% milk.
2. Low-fat milk, evaporated milk, nonfat dry milk powder.
3. Frozen yogurt, ice milk, sherbet, sorbet.
4. Low-fat yogurt.
5. Low-fat cheeses: 1% cottage cheese, skim milk ricotta, mozzarella, and American cheeses.

E. Allowed high-fat foods:

1. Margarine made with liquid safflower, corn, or sunflower oils.
2. Olive, canola, or peanut oils.
3. Nut snacks in moderation (high fat and calories).
4. Salad dressings made with saturated oils.

RESOURCES

The National Heart, Lung, and Blood Institute website has examples of low-fat and low-calorie food alternatives for dairy, meat, fish, poultry, snacks and sweets, fats, oils and salad dressings, and other miscellaneous foods at https://www.nhlbi.nih.gov/health/educational/lose_wt/eat/shop_lcal_fat.htm

NAUSEA AND VOMITING DIET SUGGESTIONS

A. For simple nausea and vomiting with an upset stomach, follow these steps:

STEP 1: REPLACE LOST FLUIDS

A. Rest your stomach for 1 to 2 hours.
B. Older child and adults:
1. After vomiting stops, take sips of clear liquids at room temperature, such as flat ginger ale, flat cola, or gelatin.
2. Suck on lollipops or popsicles.
3. Gradually increase amount of liquids. If 4 hours pass without vomiting, progress to Step 2.

STEP 2: DRY DIET

A. The foods in this diet do not meet all daily food requirements and should be used only for a short period before adding foods or advancing to Step 3:
1. Cheerios.
2. Crackers.
3. Cornflakes.
4. Graham crackers.
5. Rice Krispies.
6. Vanilla wafers.
7. Toast.
8. Dinner rolls.

STEP 3: MORE ADVANCED CARBOHYDRATES

A. Oatmeal.
B. Grits, unseasoned.
C. Rice, unseasoned.
D. Mashed potatoes.
E. Baked potato.
F. Noodles.
G. Peanut butter.
H. Pudding.

STEP 4: BLAND FOODS WITH LIMITED ODORS

A. After you are able to eat dry and more complex carbohydrates, a trial of bland foods may be tried. Foods with little or no odors are more easily tolerated after experiencing nausea and vomiting (Table B.9).

BRAT DIET

A. You may be told to use a BRAT diet. This is a combination of foods that make up a bland diet and help with nausea and vomiting: **B**ananas, **R**ice, **A**pplesauce, and **T**oast as tolerated.
B. Side effects of cancer treatments require additional liquid nutrition in order to get enough calories, protein, and nutrition. Commercial liquid nutrition products are available over the counter. Contact your oncologist to discuss the need for other treatments for managing the side effects of those treatments.

TABLE B.9 BLAND FOODS WITH LIMITED ODORS

Apple juice	Canned pears	Ice cream
Applesauce	Chicken noodle soup	Iced tea
Baked chicken	Cottage cheese	Low-fat milk
Baked turkey	Fresh apple	Sherbet
Canned peaches	Fresh banana	Turkey sandwich

NUTRITION FOR WOUND HEALING

A. Food is very important in healing wounds:
- **1.** Foods from all food groups are needed, but foods loaded with protein are the best for healing. See Table B.10 for the minimum amounts for servings of food per day to help heal wounds.
- **2.** Liquids are important too. You need six to eight cups of liquids a day. You will need special instructions on how much you can drink if you have heart failure or kidney problems.
- **3.** If you have diabetes, it is very important to check your blood sugar when you have a wound. Keeping your sugar under control will help you to heal. You may be instructed to check your urine for ketones.
- **4.** You will need special instructions on eating extra protein foods if you have kidney problems.
- **5.** You may be asked to write down all food that you eat in 24 hours to bring to your next visit.
- **6.** You may be asked to weigh at home. Use the same scales and weigh yourself at the same time of the day.
- **7.** You may be told to take vitamin C and vitamin A if you do not eat very much fruit and vegetables.
- **8.** You may be sent to see a dietitian to help plan meals.

DIETARY TIPS FOR HELPING TO HEAL YOUR WOUND

A. Whole eggs are considered the "best" protein food.
B. Eat three meals and snacks.
C. If possible, eat extra portions of protein.
D. You may find it easier to eat five to six smaller meals.
E. Shake-like drinks contain protein, vitamins, and minerals. These drinks are expensive.
F. If you use a protein powder, choose one that is made of 100% whey protein without all of the added sugars and other supplements.
G. Add powdered milk to your food to increase protein. Add powdered milk to scrambled eggs, milk, or cereal.
H. Add tuna to a salad.
I. Add beans to soups or salads.
J. Eat cottage cheese or yogurt with fruits.
K. Nuts make a good snack and may be added to salads.
L. Add eggs to salads and tuna.
M. The following list presents examples of high-protein foods:
- **1.** Whole eggs.
- **2.** Meats:
  - a. Red and white meat.
  - b. Liver.
  - c. Chicken or turkey.
  - d. Fish.
- **3.** Nuts and seeds:
  - a. Almond, cashews, peanuts, or walnuts.
  - b. Pumpkin or sunflower seeds.
  - c. Peanut butter.
- **4.** Beans and peas:
  - a. Black beans, kidney, pinto, white, lima, or soy beans.
  - b. Tofu.
  - c. Falafel patty.
  - d. Hummus.
- **5.** Dairy:
  - a. Milk (full fat).
  - b. Evaporated canned milk.
  - c. Cheese.
  - d. Plain or fruit yogurt.
  - e. Ice cream.
  - f. Cottage cheese.

N. Foods that provide energy, vitamins, and minerals include the following:
- **1.** Any kind of fruit.
- **2.** Any kind of vegetable.
- **3.** Whole grain and fortified cereal and breads including biscuits.
- **4.** Rice and pasta.
- **5.** Fats: margarine/butter, oil, and full-fat dairy foods.

TABLE B.10 SUGGESTED MINIMUM AMOUNTS OF SERVICES OF FOOD PER DAY FOR WOUND HEALING

Food Group	Number of Servings
Grains (using whole grains)	5
Vegetables	2
Fruits	2
Mild	3
Meats and beans	5
Oils, fats, and sugar	5

FOOD SOURCES AND RECOMMENDATIONS FOR COMMON VITAMIN AND MINERAL DEFICIENCIES

TABLE B.11 FOOD SOURCES FOR COMMON VITAMIN AND MINERAL DEFICIENCIES

Common Nutritional Deficiencies	Food Sources
Calcium	Dairy sources of calcium include yogurt and cheese.
	Nondairy sources of calcium are vegetables, including kale, broccoli, and Chinese cabbage.
	Calcium is also found in fortified sources, including breakfast cereals, fruit juices, and tofu.
Folate	Folate is found naturally in vegetables (especially dark green leafy vegetables), fruits, fruit juices, nuts, beans, peas, dairy products, poultry and meat, eggs, seafood, and grains.
	In January 1998, the U.S. Food and Drug Administration began requiring manufacturers to add folic acid to enriched breads, cereals, flours, cornmeal, pasta, rice, and other grain products.
Iron	Heme iron is found in animal foods that originally contained hemoglobin, including red meat, fish, and poultry.
	Nonheme iron is found in plant foods, including lentils and beans.
	Iron is also found in fortified ready-to-eat cereals.
Magnesium	Magnesium is widely distributed in plant and animal foods, including green leafy vegetables, legumes, nuts (almonds, peanuts, and cashews), seeds, and whole grains.
	Magnesium is also found in fortified breakfast cereals.
Vitamin A	Concentrations of preformed vitamin A are highest in liver, fish oils, leafy green vegetables, orange and yellow vegetables, tomato products, fruits, and some vegetable oils.
	Vitamin A is also found in fortified breakfast cereals.
Vitamin B_6	Richest sources include fish; beef, liver, and other organ meats; potatoes and other starchy vegetables such as chickpeas; and fruit (except for citrus).
	Vitamin B_6 is also found in fortified breakfast cereals.
Vitamin B_{12}	Vitamin B_{12} is found naturally in animal products, including fish, meat, poultry, eggs, and milk and milk products.
	It is generally not present in plant foods.
	Vitamin B_{12} is found in fortified breakfast cereals.
Vitamin D	Very few foods in nature contain vitamin D; it is found primarily in fortified foods.
	Almost all of the U.S. milk supply is voluntarily fortified with vitamin D.
	Both the United States and Canada mandate the fortification of infant formula with vitamin D.
Vitamin E	Vitamin E is found in nuts and seeds (sunflower seeds, almonds, hazelnuts, and peanuts), green leafy vegetables, and vegetable oils.
	Vitamin E is found in fortified breakfast cereals.
Zinc	Red meat and poultry provide the majority of zinc in American diets; however, oysters contain more zinc per serving than any other food. Other food sources include beans, nuts, seafood (crab and lobster), and dairy products.
	Zinc is also found in fortified breakfast cereals.

Source: National Institutes of Health. (2022, February 8). Multivitamin/mineral supplements: Fact sheet for health professionals. https://ods.od.nih.gov/factsheets/Mvms-HealthProfessional.

FOOD SOURCES AND RECOMMENDATIONS FOR VITAMIN D AND CALCIUM

CALCIUM-RICH FOODS

A. Sunlight exposure to the skin is recommended for approximately 20 to 30 minutes without sunscreen. Sun exposure provides an adequate source of vitamin D. Care should be taken not to burn skin:

1. Vitamin D assists with calcium absorption into bones.

2. Caffeine interferes with calcium absorption and lowers bone density. Carbonated beverages appear to be worse than coffee.

3. Vitamin D and calcium deficiency contribute to bone loss and osteoporosis.

4. Additional supplemental calcium and vitamin D are needed based on medical diagnoses and malabsorption from gastric bypass procedures (Tables B.12, B.13, and B.14).

TABLE B.12 VITAMIN D-ENRICHED FOODS

Food Source	Serving Size	Food IU
Fish liver oils, cod liver oil	3 oz	1,360
Mushrooms	3 oz	2,700
Fortified milk	8 oz	100
Herring	3 oz	1,383
Catfish	3 oz	425
Mackerel (cooked)	3.5 oz	345
Salmon (cooked)	3.5 oz	360
Sardines (canned in oil, drained)	1.75 oz	250
Fortified orange juice	8 oz	100
Fortified cereal	1 serving	100
Fortified cheese	3 oz	100

IU, international units.

TABLE B.13 THE INSTITUTE OF MEDICINE DIETARY REFERENCE INTAKES FOR CALCIUM AND VITAMIN D

Age Ranges	Calcium (mg/d)	Vitamin D (IU/d)
19–30 years old	800–1,000	600
31–50 years old	800–1,000	600
51- to 70-year-old males	800–1,000	600
51- to 70-year-old females	1,000–1,200	600
>70 years old	1,000–1,200	800
14–18 years old, pregnant/lactating	1,100–1,300	600
19–50 years old, pregnant/lactating	800–1,000	600

IU, international units.

Source: Adapted from National Institutes of Health. (2021, August 17). *Vitamin D Fact Sheet for Health Professionals.* https://ods.od.nih.gov/factsheets/VitaminD-HealthProfessional/.

TABLE B.14 FOODS THAT ARE CALCIUM-RICH

Food Source	Serving Size (mg)	IU
Yogurt	1 cup	448
Orange juice	1 cup	350
Orange	1 medium size	52
Fat-free milk	1 cup	316
Soy milk (original and vanilla with calcium and vitamins A and D added)	1 cup	299
Soy milk (original and vanilla-unfortified)	1 cup	61
Yogurt, plain	8 oz	452
Skim milk	8 oz	452
Reduced fat milk (with vitamins A and D added)	1 cup	293
Ice cream (vanilla)	½ cup	84
Shrimp	3 oz	275
Salmon	3 oz	182
Tuna (canned)	3 oz	12
Instant oatmeal	1 packet	165
Tofu	½ cup	130
Broccoli	1 cup	94
Dried beans, cooked	½ cup	50
Cheddar cheese	1 oz	306
Cottage cheese (low fat)	1 cup	206
Turnip greens	1 cup	197
Spinach (frozen)	1 cup	291
Cereal bars, snack bars (fortified)	1 bar	200

IU, international units.

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APPENDIX C

BEERS CRITERIA

TABLE C.1 2019 AMERICAN GERIATRICS SOCIETY BEERS CRITERIA FOR POTENTIALLY INAPPROPRIATE MEDICATION USE IN OLDER ADULTS

Organ System, Therapeutic Category, Drugs	Rationale	Recommendation	Quality of Evidence	Strength of Recommendation
Anticholinergics				
First-generation antihistamines Brompheniramine Carbinoxamine Chlorpheniramine Clemastine Cyproheptadine Dexbrompheniramine Dexchlorpheniramine Dimenhydrinate Diphenhydramine (oral) Doxylamine Hydroxyzine Meclizine Promethazine Pyrilamine Triprolidine	Highly anticholinergic; clearance reduced with advanced age, and tolerance develops when used as hypnotic; risk of confusion, dry mouth, constipation, and other anticholinergic effects or toxicity. Use of diphenhydramine in situations such as acute treatment of severe allergic reaction may be appropriate.	Avoid	Moderate	Strong
Antiparkinsonian Agents				
Benztropine (oral) Trihexyphenidyl	Not recommended for prevention of extrapyramidal symptoms with antipsychotics; more effective agents available for treatment of Parkinson disease.	Avoid	Moderate	Strong
Antispasmodics Atropine (excludes ophthalmic) Belladonna alkaloids Clidinium-chlordiazepoxide Dicyclomine Homatropine (excludes ophthalmic) Hyoscyamine Methscopolamine Propantheline Scopolamine	Highly anticholinergic, uncertain effectiveness.	Avoid	Moderate	Strong
Antithrombotics				
Dipyridamole, oral short-acting (does not apply to the extended-release combination with aspirin)	May cause orthostatic hypotension; more effective alternatives available; intravenous form acceptable for use in cardiac stress testing.	Avoid	Moderate	Strong

(*continued*)

TABLE C.1 2019 AMERICAN GERIATRICS SOCIETY BEERS CRITERIA FOR POTENTIALLY INAPPROPRIATE MEDICATION USE IN OLDER ADULTS (*CONTINUED*)

Organ System, Therapeutic Category, Drugs	Rationale	Recommendation	Quality of Evidence	Strength of Recommendation
Anti-Infective				
Nitrofurantoin	Potential for pulmonary toxicity, hepatotoxicity, and peripheral neuropathy, especially with long-term use; safer alternatives available.	Avoid in individuals with creatinine clearance <30 mL/min or for long-term suppression.	Low	Strong
Cardiovascular				
Peripheral alpha-1 blockers for treatment of hypertension Doxazosin Prazosin Terazosin	High risk of orthostatic hypotension and associated harms, especially in older adults; not recommended as routine treatment for hypertension; alternative agents have superior risk–benefit profile.	Avoid use as an antihypertensive.	Moderate	Strong
Central alpha agonists ***Clonidine for first-line treatment of hypertension*** ***Other CNS alpha-agonists*** Guanabenz Guanfacine Methyldopa Reserpine (>0.1 mg/d)	High risk of adverse CNS effects; may cause bradycardia and orthostatic hypotension; not recommended as routine treatment for hypertension.	Avoid clonidine as first-line antihypertensive. Avoid others as listed.	Low	Strong
Disopyramide	May induce heart failure in older adults due to potent negative inotropic action; strongly anticholinergic; other antiarrhythmic drugs preferred.	Avoid.	Low	Strong
Dronedarone	Worse outcomes have been reported in clients taking dronedarone who have permanent atrial fibrillation or severe or recently decompensated heart failure.	Avoid in individuals with permanent atrial fibrillation or severe or recently decompensated heart failure.	High	Strong
Digoxin	Use in atrial fibrillation: should not be used as a first-line agent in atrial fibrillation because there are safer and more effective alternatives for rate control supported by high-quality evidence.	Avoid as first-line therapy for atrial fibrillation.	Atrial fibrillation: low	Atrial fibrillation: strong
	Use in heart failure: evidence for benefits and harms of digoxin is conflicting and of lower quality; most but not all of the evidence concerns use in HFrEF. There is strong evidence for other agents as first-line therapy to reduce hospitalizations and mortality in adults with HFrEF. In heart failure, higher dosages are not associated with additional benefit and may increase toxicity.	Avoid as first-line therapy for heart failure.	Heart failure: low	Heart failure: strong
	Decreased renal clearance of digoxin may lead to increased risk of toxic effects; further dose reduction may be necessary in clients with stage 4 or 5 chronic kidney disease.	If used for atrial fibrillation or heart failure dosage >0.125 mg/d.	Dosage >0.125 mg/d: moderate	Dosage >0.125 mg/d: strong

(*continued*)

TABLE C.1 2019 AMERICAN GERIATRICS SOCIETY BEERS CRITERIA FOR POTENTIALLY INAPPROPRIATE MEDICATION USE IN OLDER ADULTS (*CONTINUED*)

Organ System, Therapeutic Category, Drugs	Rationale	Recommendation	Quality of Evidence	Strength of Recommendation
Nifedipine, immediate–release	Potential for hypotension; risk of precipitating myocardial ischemia.	Avoid.	High	Strong
Amiodarone	Effective for maintaining sinus rhythm but has greater toxicities than other antiarrhythmics used in atrial fibrillation; may be reasonable first-line therapy in clients with concomitant heart failure or substantial left ventricular hypertrophy if rhythm control is preferred over rate control.	Avoid amiodarone as first-line therapy for atrial fibrillation unless client has heart failure or substantial left ventricular hypertrophy.	High	Strong
Central Nervous System				
Antidepressants, alone or in combination Amitriptyline Amoxapine Clomipramine Desipramine Doxepin >6 mg/d Imipramine Nortriptyline Paroxetine Protriptyline Trimipramine	Highly anticholinergic, sedating, and cause orthostatic hypotension; safety profile of low-dose doxepin (≤6 mg/d) comparable with that of placebo.	Avoid.	High	Strong
Antipsychotics, first (conventional) and second (atypical) generation	Increased risk of cerebrovascular accident (stroke) and greater rate of cognitive decline and mortality in persons with dementia. Avoid antipsychotics for behavioral problems of dementia or delirium unless nonpharmacologic options (e.g., behavioral interventions) have failed or are not possible ***and*** the older adult is threatening substantial harm to self or others.	Avoid, except for schizophrenia, bipolar disorder, or for short-term use as antiemetic during chemotherapy.	Moderate	Strong
Barbiturates Amobarbital Butabarbital Butalbital Mephobarbital Pentobarbital Phenobarbital Secobarbital	High rate of physical dependence, tolerance to sleep benefits, greater risk of overdose at low dosages.	Avoid.	High	Strong
Benzodiazepines *Short- and intermediate-acting* Alprazolam Estazolam Lorazepam Oxazepam Temazepam Triazolam	Older adults have increased sensitivity to benzodiazepines and decreased metabolism of long-acting agents; in general, all benzodiazepines increase risk of cognitive impairment, delirium, falls, fractures, and motor vehicle crashes in older adults.	Avoid.	Moderate	Strong

(*continued*)

TABLE C.1 2019 AMERICAN GERIATRICS SOCIETY BEERS CRITERIA FOR POTENTIALLY INAPPROPRIATE MEDICATION USE IN OLDER ADULTS (*CONTINUED*)

Organ System, Therapeutic Category, Drugs	Rationale	Recommendation	Quality of Evidence	Strength of Recommendation
Long-acting Clorazepate Chlordiazepoxide (alone or in combination with amitriptyline or clidinium) Clonazepam Clorazepate Diazepam Flurazepam Quazepam	May be appropriate for seizure disorders, rapid eye movement sleep disorders, benzodiazepine withdrawal, ethanol withdrawal, severe generalized anxiety disorder, and periprocedural anesthesia.			
Meprobamate	High rate of physical dependence; sedating.	Avoid.	Moderate	Strong
Nonbenzodiazepine, benzodiazepine receptor agonist hypnotics (i.e., "Z-drugs") Eszopiclone Zaleplon Zolpidem	Nonbenzodiazepine benzodiazepine receptor agonists (i.e., "Z drugs") have adverse events similar to those of benzodiazepines in older adults (e.g., delirium, falls, fractures); increased ED visits and hospitalizations; motor vehicle crashes; minimal improvement in sleep latency and duration.	Avoid.	Moderate	Strong
Ergoloid mesylates (dehydrogenated ergot alkaloids) Isoxsuprine	Lack of efficacy.	Avoid.	High	Strong
Endocrine				
Androgens Methyltestosterone Testosterone	Potential for cardiac problems; contraindicated in males with prostate cancer.	Avoid unless indicated for confirmed hypogonadism with clinical symptoms.	Moderate	Weak
Desiccated thyroid	Concerns about cardiac effects; safer alternatives available.	Avoid.	Low	Strong
Estrogens with or without progestins	Evidence of carcinogenic potential (breast and endometrium); lack of cardioprotective effect and cognitive protection in older females. Evidence indicates that vaginal estrogens for the treatment of vaginal dryness are safe and effective; females with a history of breast cancer who do not respond to nonhormonal therapies are advised to discuss the risk and benefits of low-dose vaginal estrogen (dosages of estradiol <25 mcg twice weekly) with their healthcare provider.	Avoid systemic estrogen (e.g., oral and topical patch). Vaginal cream or tablets: acceptable to use low-dose intravaginal estrogen for management of dyspareunia, lower urinary tract infections, and other vaginal symptoms.	Oral and patch: high Vaginal cream or tablets: moderate	Oral and patch: strong Topical vaginal cream or tablets: weak
Growth hormone	Impact on body composition is small and associated with edema, arthralgia, carpal tunnel syndrome, gynecomastia, impaired fasting glucose.	Avoid, except for clients rigorously diagnosed by evidence-based criteria with growth hormone deficiency due to an established etiology.	High	Strong

(*continued*)

TABLE C.1 2019 AMERICAN GERIATRICS SOCIETY BEERS CRITERIA FOR POTENTIALLY INAPPROPRIATE MEDICATION USE IN OLDER ADULTS (*CONTINUED*)

Organ System, Therapeutic Category, Drugs	Rationale	Recommendation	Quality of Evidence	Strength of Recommendation
Insulin, sliding scale (insulin regimens containing only short- or rapid-acting insulin dosed according to current blood glucose levels without concurrent use of basal or long-acting insulin)	Higher risk of hypoglycemia without improvement in hyperglycemia management regardless of care setting; avoid insulin regimens that include only short- or rapid-acting insulin dosed according to current blood glucose levels without concurrent use of basal or long-acting insulin. This recommendation does not apply to regimens that contain basal insulin or long-acting insulin.	Avoid.	Moderate	Strong
Megestrol	Minimal effect on weight; increases risk of thrombotic events and possibly death in older adults.	Avoid.	Moderate	Strong
Sulfonylureas, long-acting Chlorpropamide Glimepiride Glyburide (also known as glibenclamide)	Chlorpropamide: prolonged half-life in older adults; can cause prolonged hypoglycemia; causes syndrome of inappropriate antidiuretic hormone secretion. Glimepiride and glyburide: higher risk of severe prolonged hypoglycemia in older adults.	Avoid.	Moderate	Strong
Gastrointestinal				
Metoclopramide	Can cause extrapyramidal effects, including tardive dyskinesia; risk may be greater in frail older adults and with prolonged exposure.	Avoid, unless for gastroparesis with duration of use not to exceed 12 weeks except in rare cases.	Moderate	Strong
Mineral oil, given orally	Potential for aspiration and adverse effects; safer alternatives available.	Avoid.	Moderate	Strong
Proton pump inhibitors	Risk of *Clostridium difficile* infection and bone loss and fractures.	Avoid scheduled use for >8 weeks unless for high-risk clients (e.g., oral corticosteroids or chronic NSAID use), erosive esophagitis, Barrett esophagitis, pathologic hypersecretory condition, or demonstrated need for maintenance treatment (e.g., due to failure of drug discontinuation trial or H2 receptor blockers).	High	Strong
Pain Medications				
Meperidine	Not effective oral analgesic in dosages commonly used; may have higher risk of neurotoxicity, including delirium, than other opioids; safer alternatives available.	Avoid.	Moderate	Strong

(*continued*)

TABLE C.1 2019 AMERICAN GERIATRICS SOCIETY BEERS CRITERIA FOR POTENTIALLY INAPPROPRIATE MEDICATION USE IN OLDER ADULTS (*CONTINUED*)

Organ System, Therapeutic Category, Drugs	Rationale	Recommendation	Quality of Evidence	Strength of Recommendation
Non-Cyclooxygenase-Selective				
Non-cyclooxygenase-selective NSAIDs, oral: Aspirin >325 mg/d Diclofenac Diflunisal Etodolac Fenoprofen Ibuprofen Ketoprofen Meclofenamate Mefenamic acid Meloxicam Nabumetone Naproxen Oxaprozin Piroxicam Sulindac Tolmetin	Increased risk of gastrointestinal bleeding or peptic ulcer disease in high-risk groups, including those aged >75 or taking oral or parenteral corticosteroids, anticoagulants, or antiplatelet agents; use of proton pump inhibitor reduces but does not eliminate risk. Upper gastrointestinal ulcers, gross bleeding, or perforation caused by NSAIDs occur in approximately 1% of clients treated for 3–6 months and in ~2%–4% of clients treated for 1 year; these trends continue with longer duration of use. Also can increase blood pressure and induce kidney injury. Risks are dose-related.	Avoid chronic use, unless other alternatives are not effective and the client can take gastroprotective agent (proton pump inhibitor or misoprostol).	Moderate	Strong
Indomethacin Ketorolac, includes parenteral	Increased risk of gastrointestinal bleeding, peptic ulcer disease, and acute kidney injury in other adults. Indomethacin is more likely than other NSAIDs to have adverse CNS effects. Of all the NSAIDs, indomethacin has the most adverse effects.	Avoid.	Moderate	Strong
Skeletal muscle relaxants Carisoprodol Chlorzoxazone Cyclobenzaprine Metaxalone Methocarbamol Orphenadrine	Most muscle relaxants poorly tolerated by older adults because some have anticholinergic adverse effects, sedation, increased risk of fractures; effectiveness at dosages tolerated by older adults questionable.	Avoid.	Moderate	Strong
Genitourinary				
Desmopressin	High risk of hyponatremia; safer alternative treatments.	Avoid for treatment of nocturia or nocturnal polyuria.	Moderate	Strong

Note: The primary target audience is practicing clinicians. The intentions of the criteria are to improve the selection of prescription drugs by clinicians and clients; evaluate patterns of drug use within populations; educate clinicians and clients on proper drug usage; and evaluate health outcome, quality-of-care, cost, and utilization data.

CNS, central nervous system; HFrEF, heart failure with reduced ejection fraction; NSAIDs, nonsteroidal anti-inflammatory drugs.

Source: American Geriatrics Society Beers Criteria® Update Expert Panel. (2019). American Geriatrics Society 2019 Updated AGS Beers Criteria® for potentially inappropriate medication use in older adults. *Journal of the American Geriatrics Society*, *67*, 674–694. https://doi.org/10.1111/jgs.15767.

TABLE C.2 2019 AMERICAN GERIATRICS SOCIETY BEERS CRITERIA FOR POTENTIALLY INAPPROPRIATE MEDICATION USE IN OLDER ADULTS DUE TO DRUG–DISEASE OR DRUG–SYNDROME INTERACTIONS THAT MAY EXACERBATE THE DISEASE OR SYNDROME

Disease or Syndrome	Drug(s)	Rationale	Recommendation	Quality of Evidence	Strength of Recommendation
Cardiovascular					
Heart failure	Avoid: cilostazol Avoid in heart failure with reduced ejection fraction: non-dihydropyridine CCBs (diltiazem, verapamil) Use with caution in clients with heart failure who are asymptomatic; avoid in clients with symptomatic heart failure; NSAIDs and COX-2 inhibitors Thiazolidinediones (pioglitazone, rosiglitazone) Dronedarone	Potential to promote fluid retention and/or exacerbate heart failure (NSAIDs and COX-2 inhibitors, non-dihydropyridine CCBs, thiazolidinediones); potential to increase mortality in order adults with heart failure (cilostazol and dronedarone).	Avoid or use with caution.	NSAIDs: moderate Non-dihydropyridine CCBs: moderate Thiazolidinediones: high Cilostazol: low Dronedarone: high	Strong
Syncope	AChEIs Nonselective peripheral alpha-1 blockers (e.g., doxazosin, prazosin, terazosin) Tertiary TCAs Antipsychotics: • Chlorpromazine • Thioridazine • Olanzapine	AChEIs cause bradycardia and should be avoided in older adults whose syncope may be due to bradycardia. Nonselective peripheral alpha-1 blockers cause orthostatic blood pressure changes and should be avoided in older adults whose syncope may be due to orthostatic hypotension. Tertiary TCAs and the antipsychotics listed increase the risk of orthostatic hypotension or bradycardia.	Avoid.	AChEIs, TCAs, and antipsychotics: high Nonselective peripheral alpha-1 blockers: high	AChEIs, TCAs: strong Nonselective peripheral alpha-1 blockers, antipsychotics: weak
Central Nervous System					
Delirium	Anticholinergics[a] Antipsychotics[b] Benzodiazepines Corticosteroids (oral and parenteral)[c] H2 receptor antagonists: • Cimetidine • Famotidine • Nizatidine • Ranitidine • Meperidine Nonbenzodiazepine, benzodiazepine receptor agonist hypnotics: eszopiclone, zaleplon, zolpidem	Avoid in older adults with or at high risk of delirium because of the potential of inducing or worsening delirium. Avoid antipsychotics for behavioral problems of dementia and/or delirium unless nonpharmacologic options (e.g., behavioral interventions) have failed or are not possible ***and*** the older adult is threatening substantial harm to self or others. Antipsychotics are associated with greater risk of cerebrovascular accident (stroke) and mortality in persons with dementia.	Avoid.	H2 receptor antagonists: low All others: moderate	Strong

(*continued*)

TABLE C.2 2019 AMERICAN GERIATRICS SOCIETY BEERS CRITERIA FOR POTENTIALLY INAPPROPRIATE MEDICATION USE IN OLDER ADULTS DUE TO DRUG–DISEASE OR DRUG–SYNDROME INTERACTIONS THAT MAY EXACERBATE THE DISEASE OR SYNDROME (*CONTINUED*)

Disease or Syndrome	Drug(s)	Rationale	Recommendation	Quality of Evidence	Strength of Recommendation
Dementia or cognitive impairment	Anticholinergics[a] Benzodiazepines Nonbenzodiazepine, benzodiazepine receptor agonist hypnotics: • Eszopiclone • Zolpidem • Zaleplon Antipsychotics, chronic and as-needed use[b]	Avoid because of adverse CNS effects. Avoid antipsychotics for behavioral problems of dementia or delirium unless nonpharmacologic options (e.g., behavioral interventions) have failed or are not possible ***and*** the older adult is threatening substantial harm to self or others. Antipsychotics are associated with greater risk of cerebrovascular accident (stroke) and mortality in persons with dementia.	Avoid.	Moderate	Strong
History of falls or fractures	Antiepileptics Antipsychotics[b] Benzodiazepines Nonbenzodiazepine, benzodiazepine receptor agonist hypnotics: • Eszopiclone • Zaleplon • Zolpidem Antidepressants: • TCAs • SSRIs • SNRIs • Opioids	May cause ataxia, impaired psychomotor function, syncope, additional falls; shorter-acting benzodiazepines are not safer than long-acting ones. If one of the drugs must be used, consider reducing use of other CNS-active medications that increase risk of falls and fractures (i.e., antiepileptics, opioid receptor agonists, antipsychotics, antidepressants, benzodiazepine receptor agonists, other sedatives and hypnotics) and implement other strategies to reduce fall risk. Data for antidepressants are mixed but no compelling evidence that certain antidepressants confer less fall risk than others.	Avoid unless safer alternatives are not available; avoid antiepileptics except for seizure and mood disorders Opioids: Avoid except for pain management in the setting of severe acute pain (e.g., recent fractures or joint replacement).	Opioids: moderate All others: high	Strong
Parkinson disease	Antiemetics: • Metoclopramide • Prochlorperazine • Promethazine All antipsychotics (except aripiprazole, quetiapine, clozapine)	Dopamine receptor antagonists with potential to worsen parkinsonian symptoms. Exceptions: Pimavanserin and clozapine appear to be less likely to precipitate worsening of Parkinson disease. Quetiapine has only been studied in low-quality clinical trials with efficacy comparable with that of placebo in five trials and to that of clozapine in two others.	Avoid.	Moderate	Strong

(*continued*)

TABLE C.2 2019 AMERICAN GERIATRICS SOCIETY BEERS CRITERIA FOR POTENTIALLY INAPPROPRIATE MEDICATION USE IN OLDER ADULTS DUE TO DRUG–DISEASE OR DRUG–SYNDROME INTERACTIONS THAT MAY EXACERBATE THE DISEASE OR SYNDROME (*CONTINUED*)

Disease or Syndrome	Drug(s)	Rationale	Recommendation	Quality of Evidence	Strength of Recommendation
Gastrointestinal					
History of gastric or duodenal ulcers	Aspirin (>325 mg/d) Non-COX-2 selective NSAIDs	May exacerbate existing ulcers or cause new/additional ulcers.	Avoid unless other alternatives are not effective and the client can take gastroprotective agent (e.g., proton pump inhibitor or misoprostol).	Moderate	Strong
Kidney/Urinary Tract					
Chronic kidney disease stage IV or higher (creatinine clearance <30 mL/min)	NSAIDs (non-COX and COX-selective, oral and parenteral, nonacetylated salicylates)	May increase risk of acute kidney injury and further decline of renal function.	Avoid.	Moderate	Strong
Urinary incontinence (all types) in females	Estrogen oral and transdermal (excludes intravaginal estrogen) Peripheral alpha-1 blockers: doxazosin, prazosin, terazosin	Lack of efficacy (oral estrogen) and aggravation of incontinence (alpha-1 blockers).	Avoid in females.	Estrogen: high Peripheral alpha-1 blockers: moderate	Estrogen: strong Peripheral alpha-1 blockers: strong
Lower urinary tract symptoms, benign prostatic hyperplasia	Strongly anticholinergic drugs, except antimuscarinics for urinary incontinence[a]	May decrease urinary flow and cause urinary retention.	Avoid in males.	Moderate	Strong

Note: The primary target audience is the practicing clinician. The intentions of the criteria are to improve selection of prescription drugs by clinicians and clients; evaluate patterns of drug use within populations; educate clinicians and clients on proper drug usage; and evaluate health outcome, quality-of-care, cost, and utilization data.

[a]See Table 7 in full criteria available on geriatricscareonline.org.

[b]May be required to treat concurrent schizophrenia, bipolar disorder, and other selected mental health conditions but should be prescribed in the lowest effective dose and shortest possible duration.

[c]Excludes inhaled and topical forms. Oral and parenteral corticosteroids may be required for conditions such as exacerbations of chronic obstructive pulmonary disease but should be prescribed in the lowest effective dose and for the shortest possible duration.

AChEI, acetylcholinesterase inhibitor; CCB, calcium channel blocker; CNS, central nervous system; COX, cyclooxygenase; NSAID, nonsteroidal anti-inflammatory drug; SNRIs, serotonin-norepinephrine reuptake inhibitor; SSRIs, selective serotonin reuptake inhibitors; TCA, tricyclic antidepressant.

Source: American Geriatrics Society Beers Criteria® Update Expert Panel. (2019). American Geriatrics Society 2019 Updated AGS Beers Criteria® for potentially inappropriate medication use in older adults. Journal of the American Geriatrics Society, 67, 674–694. https://doi.org/10.1111/jgs.15767.

TABLE C.3 2019 AMERICAN GERIATRICS SOCIETY BEERS CRITERIA FOR POTENTIALLY INAPPROPRIATE MEDICATIONS TO BE USED WITH CAUTION IN OLDER ADULTS

Drug(s)	Rationale	Recommendation	Quality of Evidence	Strength of Recommendation
Aspirin for primary prevention of cardiovascular disease and colorectal cancer	Risk of major bleeding from aspirin increases markedly in older age. Several studies suggest lack of net benefit when used for primary prevention in older adults with cardiovascular risk factors, but evidence is not conclusive. Aspirin is generally indicated for secondary prevention in older adults with established cardiovascular disease.	Use with caution in adults aged ≥70 years old.	Moderate	Strong
Dabigatran Rivaroxaban	Increased risk of gastrointestinal bleeding compared with warfarin and reported rates with other direct oral anticoagulants when used for long-term treatment of VTE or atrial fibrillation in adults aged ≥75 years.	Use with caution for treatment of VTE or atrial fibrillation in adults aged ≥75 years old.	Moderate	Strong
Prasugrel	Increased risk of bleeding in older adults; benefit in highest risk older adults (e.g., those with prior myocardial infarction or diabetes mellitus) may offset risk when used for its approved indication of acute coronary syndrome to be managed with percutaneous coronary intervention.	Use with caution in adults aged ≥75 years old.	Moderate	Weak
Antipsychotics Carbamazepine Diuretics Mirtazapine Oxcarbazepine SNRIs SSRIs TCAs Tramadol	May exacerbate or cause syndrome of inappropriate antidiuretic hormone secretion or hyponatremia; monitor sodium level closely when starting or changing dosages in older adults.	Use with caution.	Moderate	Strong
Dextromethorphan/ quinidine	Limited efficacy in clients with behavioral symptoms of dementia (does not apply to treatment of PBA). May increase risk of falls and concerns with clinically significant drug interactions.	Use with caution.	Moderate	Strong
Trimethoprim-sulfamethoxazole	Increased risk of hyperkalemia when used concurrently with an ACEI or ARB in the presence of decreased creatinine clearance.	Use with caution in clients on ACEI or ARB and decreased creatinine clearance.	Low	Strong

Note: The primary target audience is the practicing clinician. The intentions of the criteria are to improve selection of prescription drugs by clinicians and clients; evaluate patterns of drug use within populations; educate clinicians and clients on proper drug usage; and evaluate health outcome, quality-of-care, cost, and utilization data.

ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; PBA, pseudobulbar affect; SNRIs, serotonin-norepinephrine reuptake inhibitors; SSRIs, selective serotonin reuptake inhibitors; TCAs, tricyclic antidepressants; VTE, venous thromboembolism.

Source: American Geriatrics Society Beers Criteria® Update Expert Panel. (2019). American Geriatrics Society 2019 Updated AGS Beers Criteria® for potentially inappropriate medication use in older adults. *Journal of the American Geriatrics Society*, *67*, 674–694. https://doi.org/10.1111/jgs.15767.

TABLE C.4 2019 AMERICAN GERIATRICS SOCIETY BEERS CRITERIA FOR POTENTIALLY CLINICALLY DRUG–DRUG INTERACTIONS THAT SHOULD BE AVOIDED IN OLDER ADULTS

Object Drug and Class	Interacting Drug and Class	Risk Rationale	Recommendation	Quality of Evidence	Strength of Recommendation
RAS inhibitor (ACEIs, ARBs, aliskiren) or potassium-sparing diuretics (amiloride, triamterene)	Another RAS inhibitor (ACEIs, ARBs, aliskiren)	Increased risk of hyperkalemia	Avoid routine use in those with chronic kidney disease stage 3a or higher.	Moderate	Strong
Opioids	Benzodiazepines	Increased risk of overdose	Avoid.	Moderate	Strong
Opioids	Gabapentin, pregabalin	Increased risk of severe sedation-related adverse events, including respiratory depression and death	Avoid; exceptions are when transitioning from opioid therapy to gabapentin or pregabalin, or when using gabapentinoids to reduce opioid dose, although caution should be used in all circumstances.	Moderate	Strong
Anticholinergic	Anticholinergic	Increased risk of cognitive decline	Avoid, minimize number of anticholinergic drugs.	Moderate	Strong
Antidepressants (i.e., TCAs and SSRIs) Antipsychotics Antiepileptics Benzodiazepines and nonbenzodiazepines, benzodiazepine receptor agonist hypnotics (i.e., "Z-drugs") Opioids	Any combination of ≥3 of these CNS-active drugs[a]	Increased risk of falls (all) and of fracture (benzodiazepines and nonbenzodiazepine, benzodiazepine receptor agonist hypnotics)	Avoid total of ≥3 CNS-active drugs[a]; minimize number of CNS-active drugs.	Combinations including benzodiazepines and nonbenzodiazepine, benzodiazepine receptor agonist hypnotics or opioids: high All other combinations: moderate	Strong
Corticosteroids, oral or parenteral	NSAIDs	Increased risk of peptic ulcer disease or gastrointestinal bleeding	Avoid; if not possible, provide gastrointestinal protection.	Moderate	Strong
Lithium	ACEIs	Increased risk of lithium toxicity	Avoid, monitor lithium concentrations.	Moderate	Strong
Lithium	Loop diuretics	Increased risk of lithium toxicity	Avoid, monitor lithium concentrations.	Moderate	Strong
Peripheral alpha-1 blockers	Loop diuretics	Increased risk of urinary incontinence in older females	Avoid in older females, unless conditions warrant both drugs.	Moderate	Strong
Phenytoin	Trimethoprim-sulfamethoxazole	Increased risk of urinary incontinence in older females	Avoid.	Moderate	Strong
Theophylline	Cimetidine Ciprofloxacin	Increased risk of theophylline toxicity	Avoid.	Moderate	Strong
Warfarin	Amiodarone	Increased risk of bleeding	Avoid when possible; if used together, monitor international normalized ratio closely.	Moderate	Strong

(*continued*)

TABLE C.4 2019 AMERICAN GERIATRICS SOCIETY BEERS CRITERIA FOR POTENTIALLY CLINICALLY DRUG–DRUG INTERACTIONS THAT SHOULD BE AVOIDED IN OLDER ADULTS (*CONTINUED*)

Object Drug and Class	Interacting Drug and Class	Risk Rationale	Recommendation	Quality of Evidence	Strength of Recommendation
Warfarin	Ciprofloxacin	Increased risk of bleeding	Avoid when possible; if used together, monitor international normalized ratio closely.	Moderate	Strong
Warfarin	Macrolides (excluding azithromycin)	Increased risk of bleeding	Avoid when possible; if used together, monitor international normalized ratio closely.	Moderate	Strong
Warfarin	Trimethoprim-sulfamethoxazole	Increased risk of bleeding	Avoid when possible; if used together, monitor INR closely.	Moderate	Strong
Warfarin	NSAIDs	Increased risk of bleeding	Avoid when possible; if used together, monitor for bleeding closely.	High	Strong

[a]CNS-active drugs: antipsychotics; benzodiazepines; nonbenzodiazepines, benzodiazepine receptor agonist hypnotics; TCAs; SSRI; and opioids.

ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; CNS, central nervous system; INR, international normalized ratio; NSAID, nonsteroidal anti-inflammatory drug; RAS, recurrent aphthous stomatitis; SSRI, selective serotonin reuptake inhibitor; TCA, tricyclic antidepressant.

Source: American Geriatrics Society Beers Criteria® Update Expert Panel. (2019). American Geriatrics Society 2019 Updated AGS Beers Criteria® for potentially inappropriate medication use in older adults. *Journal of the American Geriatrics Society*, *67*, 674–694. https://doi.org/10.1111/jgs.15767.

TABLE C.5 2019 AMERICAN GERIATRICS SOCIETY BEERS CRITERIA FOR MEDICATIONS THAT SHOULD BE AVOIDED OR HAVE THEIR DOSAGE REDUCED WITH VARYING LEVELS OF KIDNEY FUNCTION IN OLDER ADULTS

Medication Class and Medication	Creatinine Clearance, mL/min, at Which Action Required	Rationale	Recommendation	Quality of Evidence	Strength of Recommendation
Anti-Infective					
Ciprofloxacin	<30	Increased risk of CNS effects (e.g., seizures, confusion) and tendon rupture	Doses used to treat common infections typically require reduction when CrCl <30 mL/min.	Moderate	Strong
Trimethoprim-sulfamethoxazole	<30	Increased risk of worsening renal function and hyperkalemia	CrCl 15–29 mL/min: Reduce dose. <15 mL/min: Avoid.	Moderate	Strong
Cardiovascular or Hemostasis					
Amiloride	<30	Increased potassium, and decreased sodium	Avoid.	Moderate	Strong
Apixaban	<25	Lack of evidence for efficacy and safety in clients with a CrCl <25 mL/min	Avoid.	Moderate	Strong
Dabigatran	<30	Lack of evidence for efficacy and safety in individuals with a CrCl <30 mL/min Label dose for clients with a CrCl 15–30 mL/min based on pharmacokinetic data	Avoid; dose adjustment is advised when CrCl >30 mL/min in the presence of drug–drug interactions.	Moderate	Strong
Dofetilide	<60	QTc prolongation and torsades de pointes	CrCl 20–59 mL/min: Reduce dose. CrCl <20 mL/min: Avoid.	Moderate	Strong
Edoxaban	15–50 <15 or >95	Lack of evidence of efficacy or safety in clients with a CrCl <30 mL/min	CrCl 15–50 mL/min: Reduce dose. CrCl <15 or >95 mL/min: Avoid.	Moderate	Strong
Enoxaparin	<30	Increased risk of bleeding	Reduce dose.	Moderate	Strong
Fondaparinux	<30	Increased risk of bleeding	Avoid.	Moderate	Strong

(*continued*)

TABLE C.5 2019 AMERICAN GERIATRICS SOCIETY BEERS CRITERIA FOR MEDICATIONS THAT SHOULD BE AVOIDED OR HAVE THEIR DOSAGE REDUCED WITH VARYING LEVELS OF KIDNEY FUNCTION IN OLDER ADULTS (*CONTINUED*)

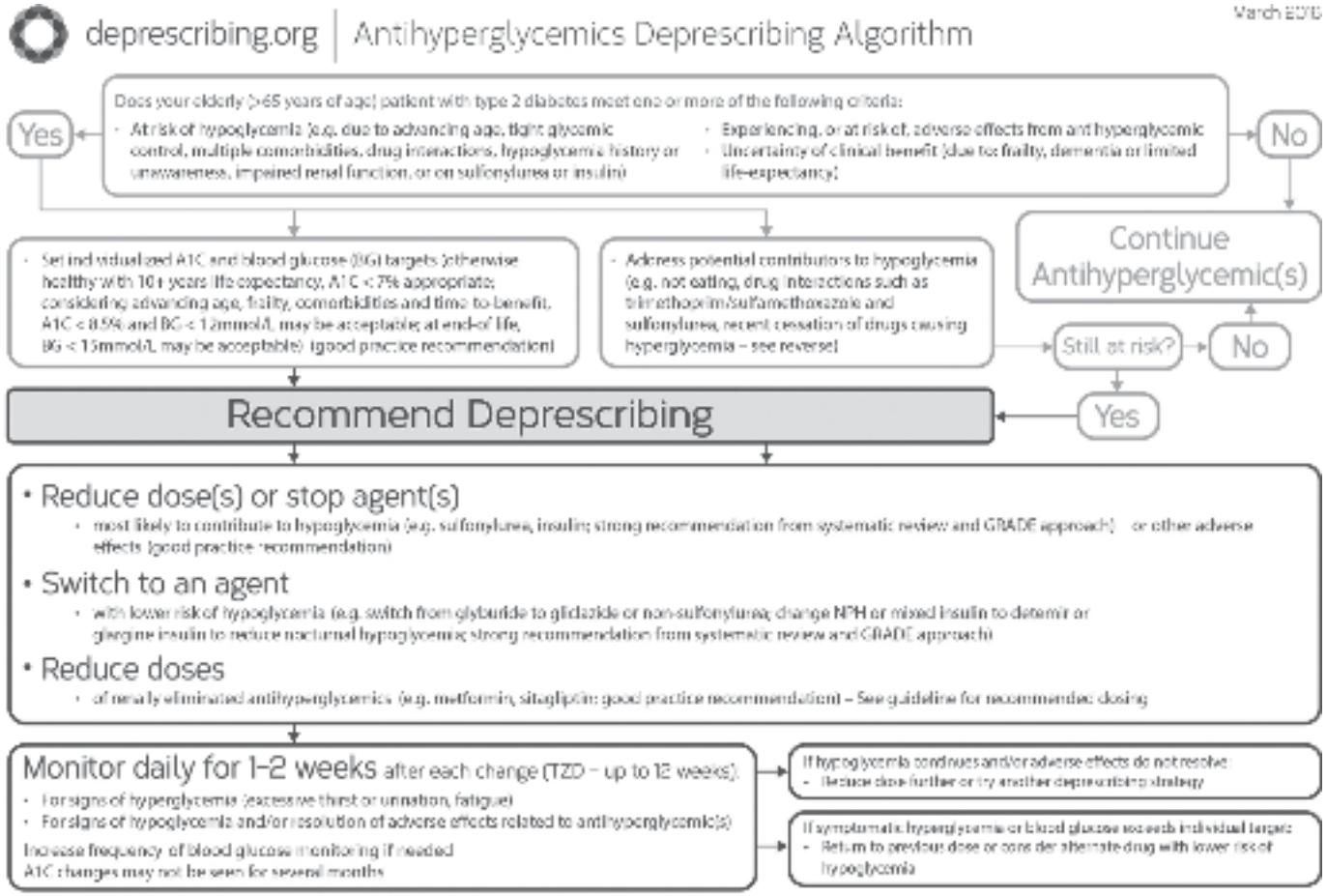
Medication Class and Medication	Creatinine Clearance, mL/min, at Which Action Required	Rationale	Recommendation	Quality of Evidence	Strength of Recommendation
Rivaroxaban	<50	Lack of efficacy or safety evidence in clients with a CrCl <30 mL/min	Nonvalvular atrial fibrillation: Reduce dose if CrCl 15–50 mL/min; avoid if CrCl <15 mL/min. Venous thromboembolism treatment for VTE prophylaxis with hip or knee replacement: Avoid if CrCl <30 mL/min.	Moderate	Strong
Spironolactone	<30	Increased potassium	Avoid.	Moderate	Strong
Triamterene	<30	Increased potassium and decreased sodium	Avoid.	Moderate	Strong
Central Nervous System and Analgesics					
Duloxetine	<30	Increased gastrointestinal adverse effects (nausea, diarrhea)	Avoid.	Moderate	Weak
Gabapentin	<60	CNS adverse effects	Reduce dose.	Moderate	Strong
Levetiracetam	≤80	CNS adverse effects	Reduce dose.	Moderate	Strong
Pregabalin	<60	CNS adverse effects	Reduce dose.	Moderate	Strong
Tramadol	<30	CNS adverse effects	Immediate–release: Reduce dose. Extended–release: Avoid.	Low	Weak
Gastrointestinal					
Cimetidine	<50	Mental status changes	Reduce dose.	Moderate	Strong
Famotidine	<50	Mental status changes	Reduce dose.	Moderate	Strong
Nizatidine	<50	Mental status changes	Reduce dose.	Moderate	Strong
Ranitidine	<50	Mental status changes	Reduce dose.	Moderate	Strong
Hyperuricemia					
Colchicine	<30	Gastrointestinal, neuromuscular, bone marrow toxicity	Reduce dose; monitor for adverse effects.	Moderate	Strong
Probenecid	<30	Loss of effectiveness	Avoid.	Moderate	Strong

CNS, central nervous system; CrCl, creatinine clearance; VTE, venous thromboembolism.

Source: American Geriatrics Society Beers Criteria® Update Expert Panel. (2019). American Geriatrics Society 2019 Updated AGS Beers Criteria® for potentially inappropriate medication use in older adults. *Journal of the American Geriatrics Society*, *67*, 674–694. https://doi.org/10.1111/jgs.15767.

APPENDIX D

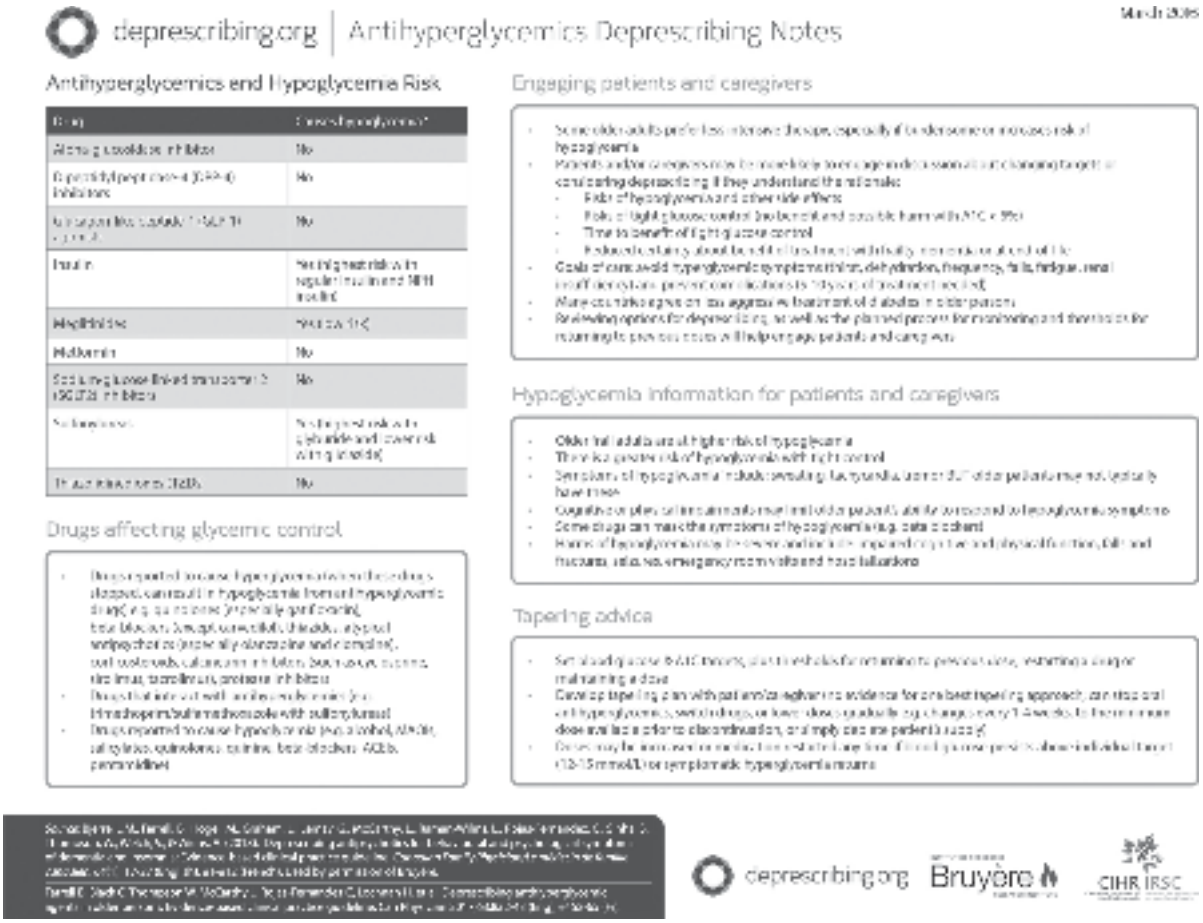
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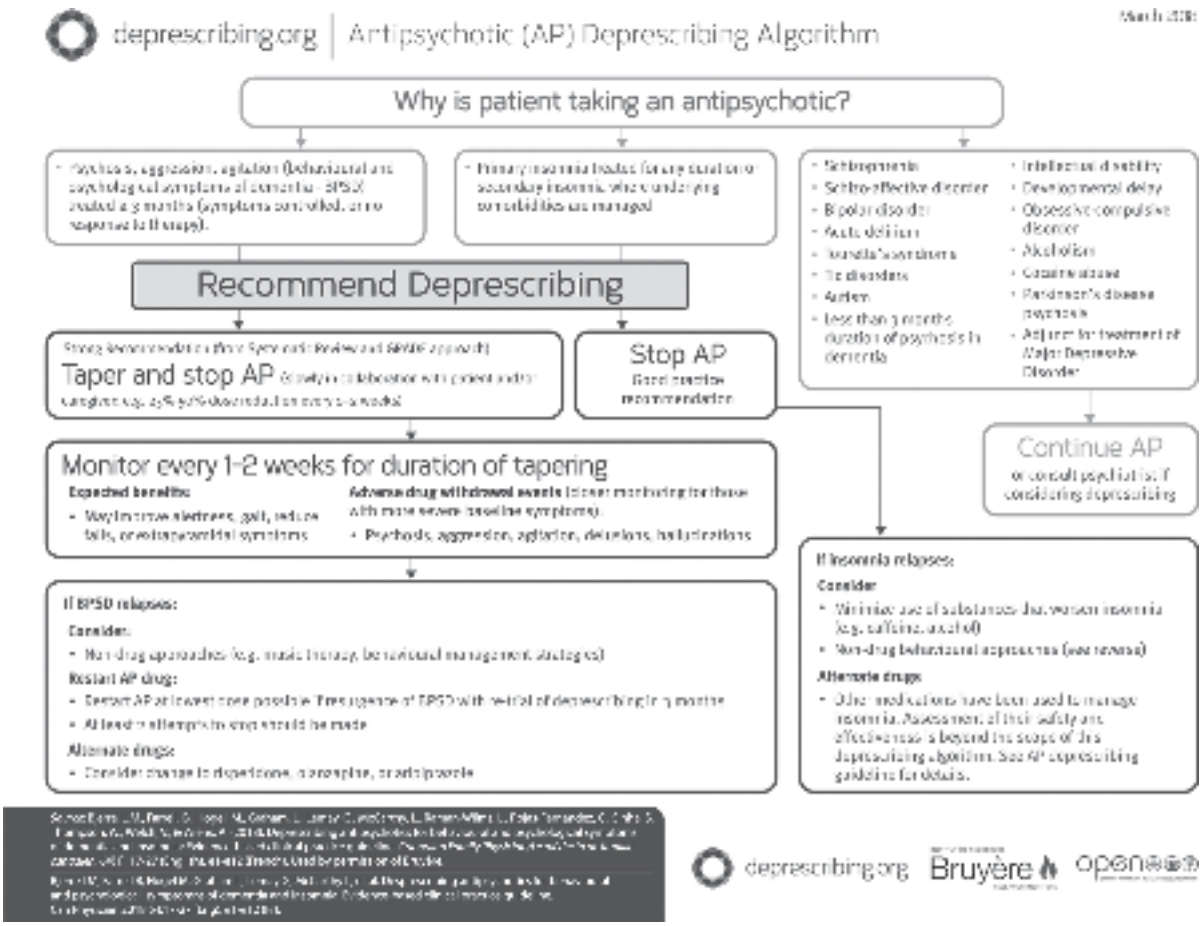


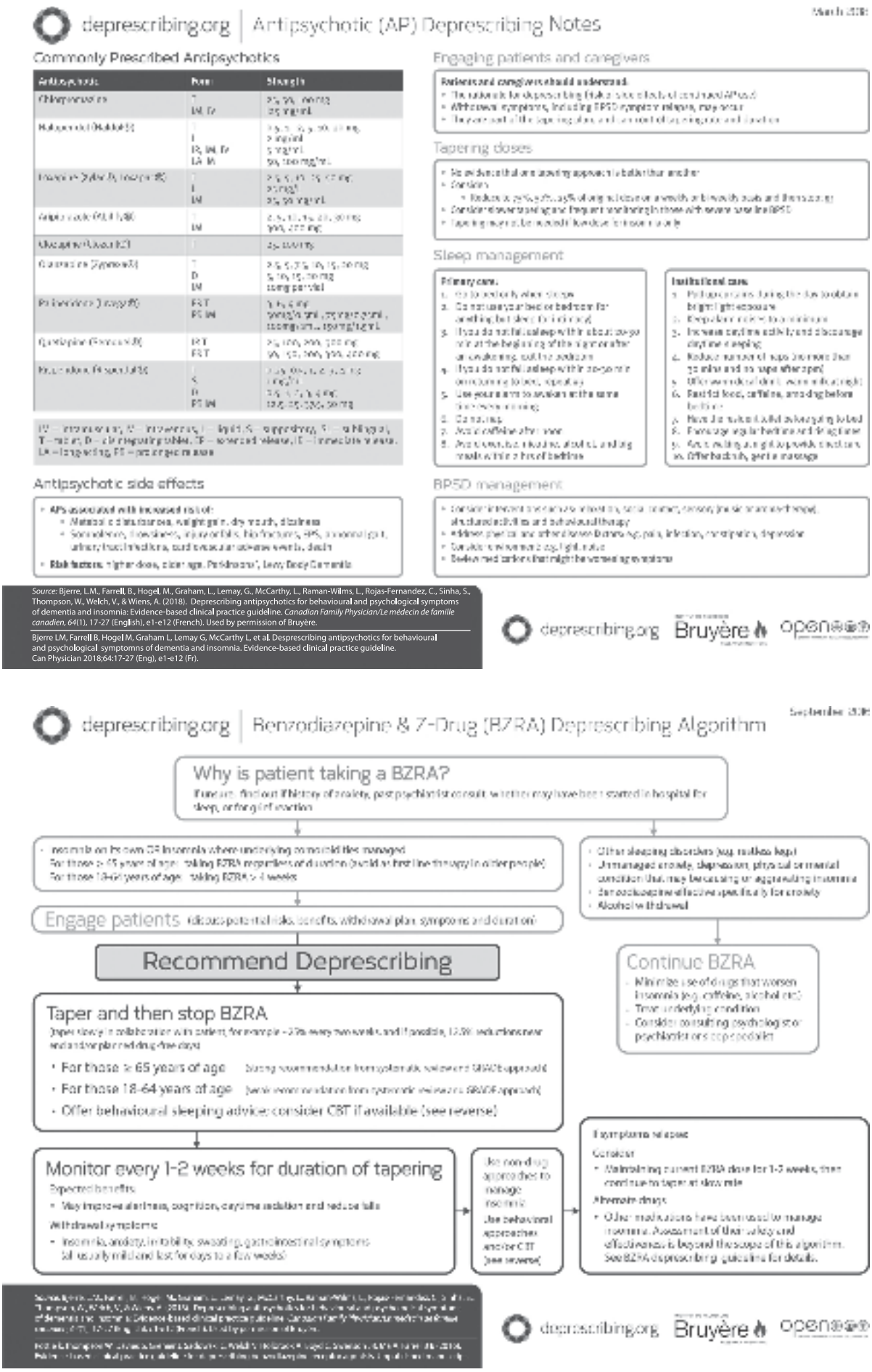
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Farrell B, Black C, Thompson W, McCarthy L, Rojas-Fernandez C, Lochnan H, et al. Deprescribing anthiyperglycemic agents in older persons. Evidence-based clinical practice guideline. Can Physician 2017;63:832-43 (Eng), e452-65 (Fr).

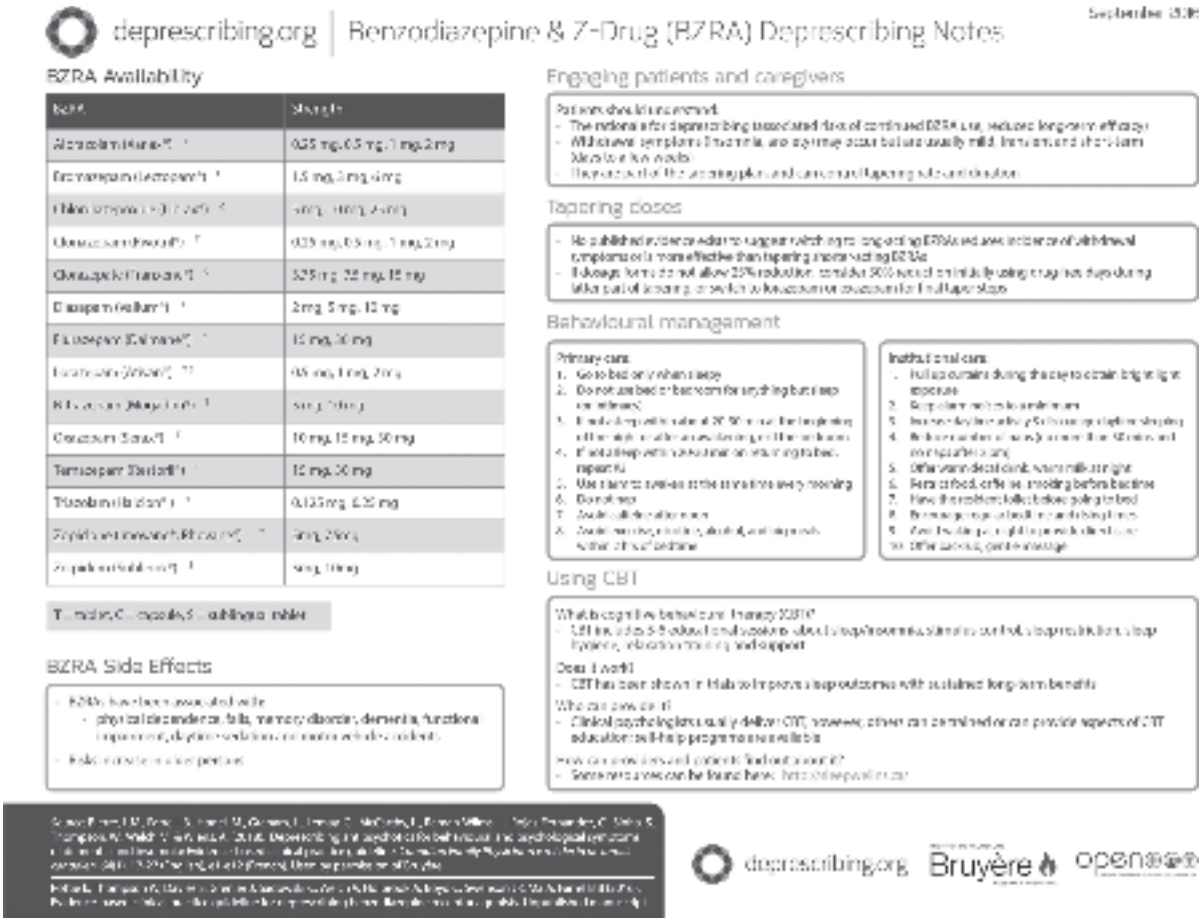
deprescribing.org Bruyère CIHR IRSC

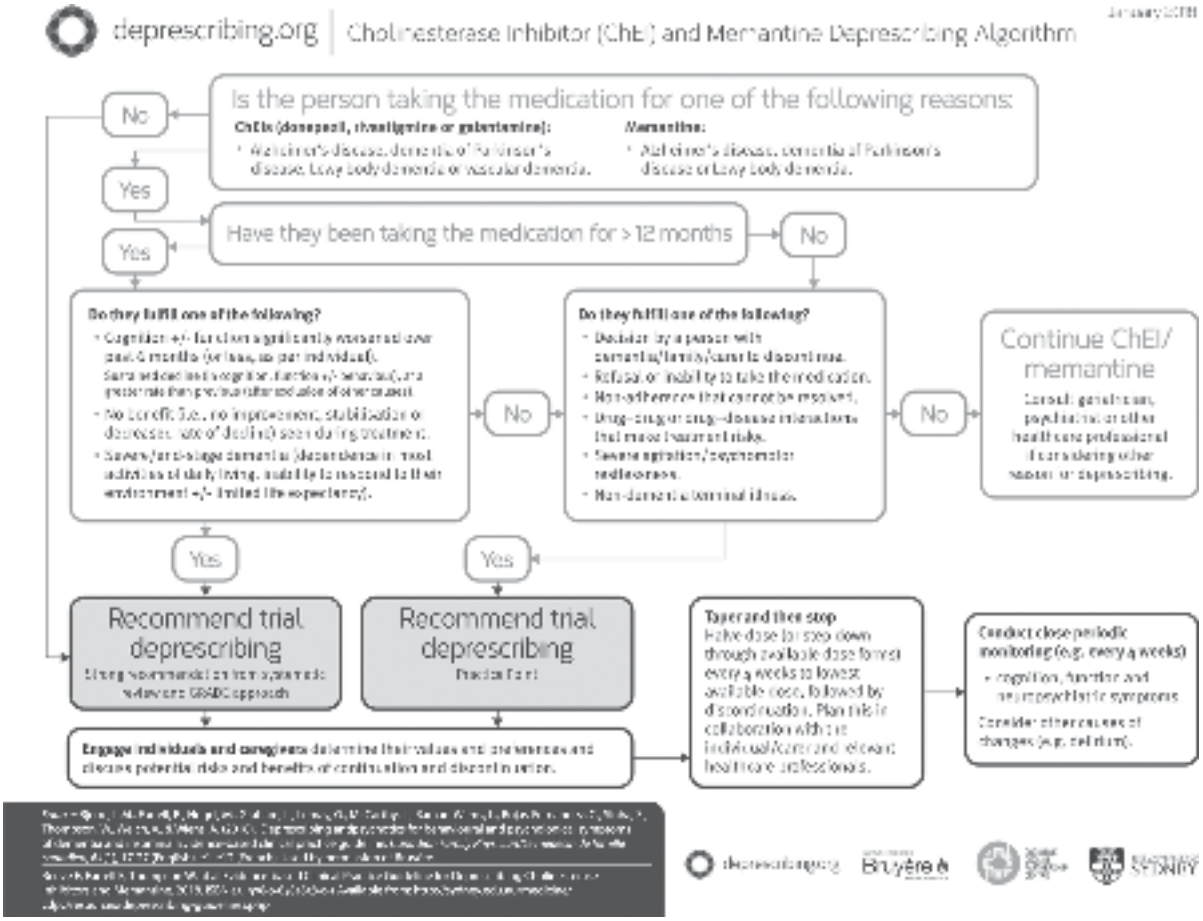

deprescribing.org | Antihyperglycemics Deprescribing Notes
March 2018
Antihyperglycemics and Hypoglycemia Risk
Drug
Causes hypoglycemia?
Alpha-glucosidase inhibitor
No
Dipeptidyl peptidase-4 (DPP-4) inhibitor
No
Glucagon-like peptide-1 (GLP-1) agonist
No
Insulin
Yes (highest risk in regular treatment NPH insulin)
Meglitinide
Yes (rare risk)
Metformin
No
Sodium-glucose cotransporter 2 (SGLT2) inhibitor
No
Sulfonylurea
Yes (highest risk in glyburide and chlorpropamide)
Thiazolidinedione (TZD)
No
Drugs affecting glycemic control
Engaging patients and caregivers
Hypoglycemia information for patients and caregivers
Tapering advice

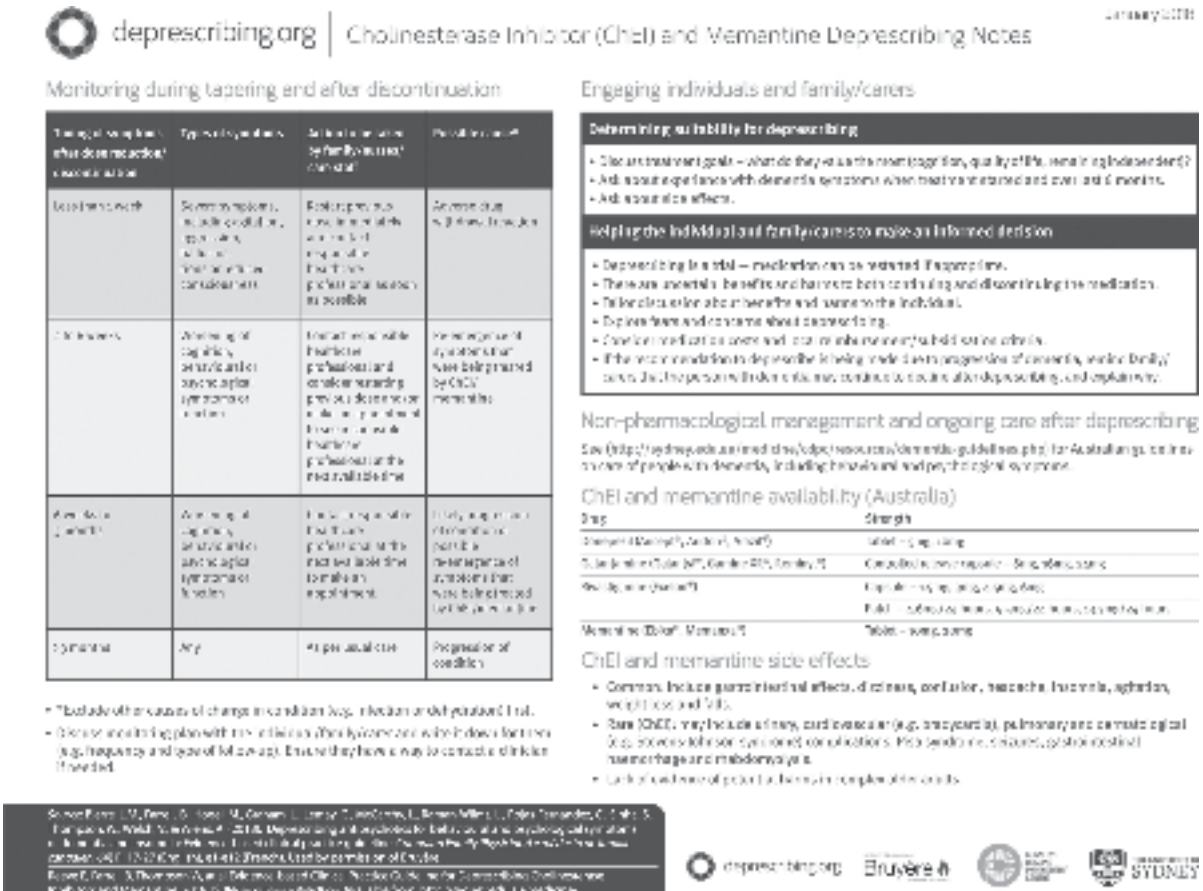

deprescribing.org | Antipsychotic (AP) Deprescribing Algorithm
March 2018
Why is patient taking an antipsychotic?
Recommend Deprescribing
Taper and stop AP
Stop AP
Good practice recommendation
Monitor every 1-2 weeks for duration of tapering
Expected benefits:
Adverse drug withdrawal events (closer monitoring for those with more severe baseline symptoms):
If BPSD relapses:
Consider:
Restart AP drug:
Alternate drugs:
If insomnia relapses:
Consider
Alternate drugs
Continue AP
or consult psychiatrist if considering deprescribing

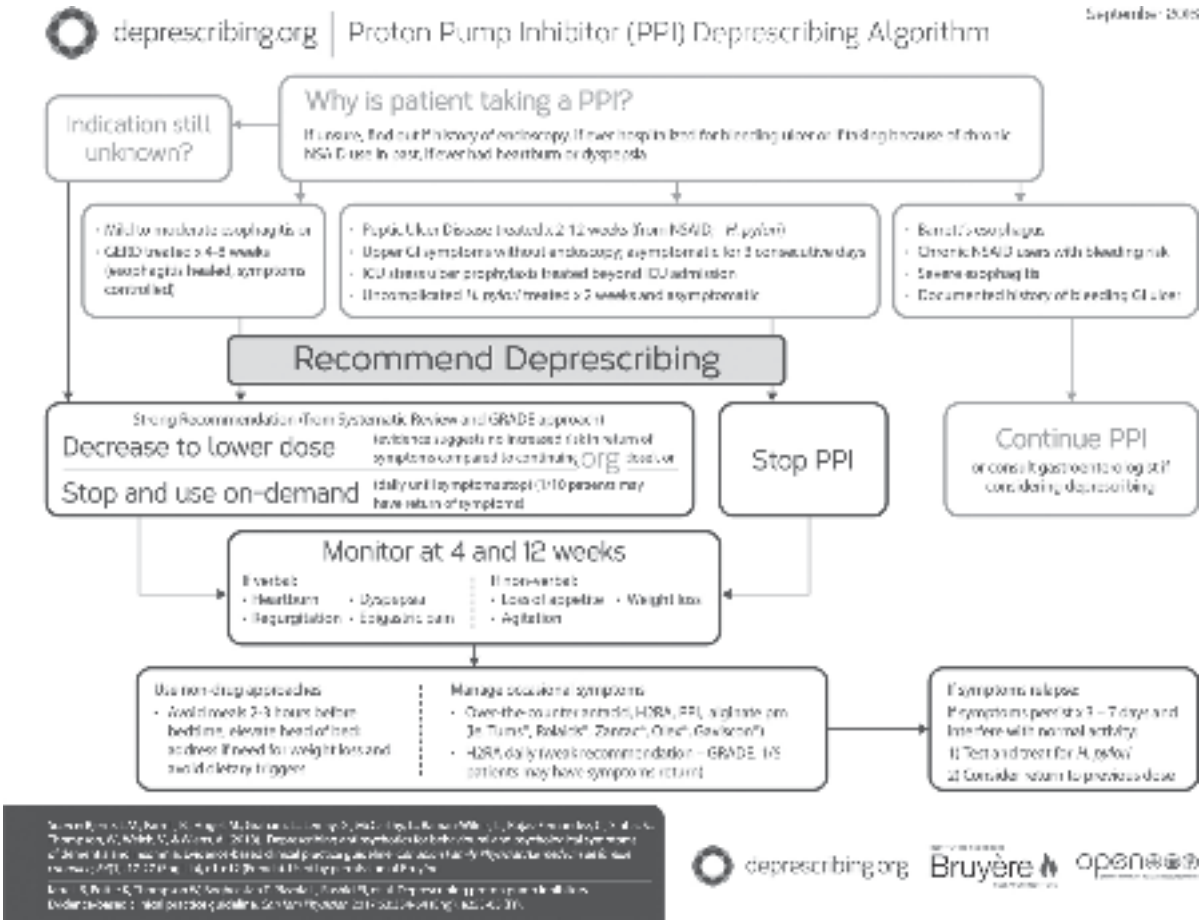

deprescribing.org | Antipsychotic (AP) Deprescribing Notes
Commonly Prescribed Antipsychotics
Engaging patients and caregivers
Tapering doses
Sleep management
Primary care:
Institutional care:
Antipsychotic side effects
BPSD management
Source: Bjerre, L.M., Farrell, B., Hogel, M., Graham, L., Lemay, G., McCarthy, L., Raman-Wilms, L., Rojas-Fernandez, C., Sinha, S., Thompson, W., Welch, V., & Wiens, A. (2018). Deprescribing antipsychotics for behavioural and psychological symptoms of dementia and insomnia: Evidence-based clinical practice guideline. Canadian Family Physician/Le médecin de famille canadien, 64(1), 17-27 (English), e1-e12 (French). Used by permission of Bruyère.
Bjerre LM, Farrell B, Hogel M, Graham L, Lemay G, McCarthy L, et al. Deprescribing antipsychotics for behavioural and psychological symptomns of dementia and insomnia. Evidence-based clinical practice guideline. Can Physician 2018;64:17-27 (Eng), e1-e12 (Fr).
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deprescribing.org | Benzodiazepine & Z-Drug (BZRA) Deprescribing Algorithm
Why is patient taking a BZRA?
Engage patients
Recommend Deprescribing
Taper and then stop BZRA
For those ≥ 65 years of age
For those 18-64 years of age
Offer behavioural sleeping advice; consider CBT if available (see reverse)
Monitor every 1-2 weeks for duration of tapering
Continue BZRA
Symptoms relapse
Alternate drugs
deprescribing.org
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deprescribing.org | Benzodiazepine & Z-Drug (BZRA) Deprescribing Notes
BZRA Availability
BZRA
Strength
Alprazolam (Xanax®, ...)
0.25 mg, 0.5 mg, 1 mg, 2 mg
Bromazepam (Lectopam®, ...)
1.5 mg, 3 mg, 6 mg
Chlordiazepoxide (Librium®, ...)
5 mg, 10 mg, 25 mg
Clonazepam (Rivotril®, ...)
0.25 mg, 0.5 mg, 1 mg, 2 mg
Clorazepate (Tranxene®, ...)
3.75 mg, 7.5 mg, 15 mg
Diazepam (Valium®, ...)
2 mg, 5 mg, 10 mg
Flurazepam (Dalmane®, ...)
15 mg, 30 mg
Lorazepam (Ativan®, ...)
0.5 mg, 1 mg, 2 mg
Oxazepam (Serax®, ...)
10 mg, 15 mg, 30 mg
Temazepam (Restoril®, ...)
15 mg, 30 mg
Triazolam (Halcion®, ...)
0.125 mg, 0.25 mg
Zopiclone (Imovane®, Rhovane®, ...)
3.75 mg, 7.5 mg
Zolpidem (Ambien®, ...)
5 mg, 10 mg
BZRA Side Effects
Engaging patients and caregivers
Tapering doses
Behavioural management
Primary care
Institutional care
Using CBT
What is cognitive behavioural therapy (CBT)?
Does it work?
Who can provide it?
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deprescribing.org | Cholinesterase Inhibitor (ChEI) and Memantine Deprescribing Algorithm
Is the person taking the medication for one of the following reasons:
ChEIs (donepezil, rivastigmine or galantamine):
Alzheimer's disease, dementia of Parkinson's disease, Lewy body dementia or vascular dementia.
Memantine:
Alzheimer's disease, dementia of Parkinson's disease or Lewy body dementia.
No
Yes
Have they been taking the medication for > 12 months
No
Yes
Do they fulfill one of the following?
Do they fulfill one of the following?
Continue ChEI/ memantine
No
No
Yes
Yes
Recommend trial deprescribing
Strong recommendation, moderate evidence GRADE approach
Recommend trial deprescribing
Practice Point
Taper and then stop
Engage individuals and caregivers to determine their values and preferences and discuss potential risks and benefits of continuation and discontinuation.
Conduct close periodic monitoring (e.g. every 4 weeks)
deprescribing.org
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SYDNEY


deprescribing.org | Cholinesterase Inhibitor (ChEI) and Memantine Deprescribing Notes
Monitoring during tapering and after discontinuation
Engaging individuals and family/carers
Determining suitability for deprescribing
Helping the individual and family/carers to make an informed decision
Non-pharmacological management and ongoing care after deprescribing
ChEI and memantine availability (Australia)
Drug
Strength
ChEI and memantine side effects
deprescribing.org
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SYDNEY


deprescribing.org | Proton Pump Inhibitor (PPI) Deprescribing Algorithm
September 2015
Why is patient taking a PPI?
Indication still unknown?
Recommend Deprescribing
Strong Recommendation (from Systematic Review and GRADE approach)
Decrease to lower dose
Stop and use on-demand
Stop PPI
Continue PPI
Monitor at 4 and 12 weeks
Use non-drug approaches
Manage withdrawal symptoms
If symptoms relapse
deprescribing.org
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deprescribing.org | Proton Pump Inhibitor (PPI) Deprescribing Notes

September 2018

PPI Availability

PPI	Standard dose (Usual regimen: daily)*	Low dose (maintenance) (once daily)
Omeprazole (Losec®) - Capsule	20 mg[a]	10 mg[a]
Esomeprazole (Nexium®) - Tablet	20[a] or 40[b] mg	20 mg
Lansoprazole (Prevacid®) - Capsule	30 mg[a]	15 mg[a]
Dexlansoprazole (Dexilant®) - Tablet	30[a] or 60[b] mg	30 mg
Pantoprazole (Tecta®, Pantoloc®) - Tablet	40 mg	20 mg
Rabeprazole (Pariet®) - Tablet	20 mg	10 mg

Legend

a Non-erosive reflux disease

b Reflux esophagitis

c Symptomatic non-erosive gastroesophageal reflux disease

d Healing of erosive esophagitis

+ Can be sprinkled on food

* Standard dose PPI taken BID only indicated in treatment of peptic ulcer caused by *H. pylori*; PPI should generally be stopped once eradication therapy is complete unless risk factors warrant continuing PPI (see guideline for details)

Key

GERD = gastroesophageal reflux disease

NSAID = nonsteroidal anti-inflammatory drugs

H2RA = H2 receptor antagonist

SR = systematic review

GRADE = Grading of Recommendations Assessment, Development and Evaluation

Engaging patients and caregivers

Patients and/or caregivers may be more likely to engage if they understand the rationale for deprescribing (risks of continued PPI use, long-term therapy may not be necessary), and the deprescribing process

PPI side effects

- When an ongoing indication is unclear, the risk of side effects may outweigh the chance of benefit
- PPIs are associated with higher risk of fractures, *C. difficile* infections and diarrhea, community-acquired pneumonia, vitamin B12 deficiency and hypomagnesemia
- Common side effects include headache, nausea, diarrhea and rash

Tapering doses

- No evidence that one tapering approach is better than another
- Lowering the PPI dose (for example, from twice daily to once daily, or halving the dose, or taking every second day) OR stopping the PPI and using it on-demand are equally recommended strong options
- Choose what is most convenient and acceptable to the patient

On-demand definition

Daily intake of a PPI for a period sufficient to achieve resolution of the individual's reflux related symptoms; following symptom resolution, the medication is discontinued until the individual's symptoms recur, at which point, medication is again taken daily until the symptoms resolve

Source: Bjerre, L.M., Farrell, B., Hogel, M., Graham, L., Lemay, G., McCarthy, L., Raman-Wilms, L., Rojas-Fernandez, C., Sinha, S., Thompson, W., Welch, V., & Wiens, A. (2018). Deprescribing antipsychotics for behavioural and psychological symptoms of dementia and insomnia: Evidence-based clinical practice guideline. *Canadian Family Physician/Le médecin de famille canadien, 64*(1), 17-27 (English), e1-e12 (French). Used by permission of Bruyère.

farrell B, Pottie K, Thompson W, Boghossian T, Pizzola L, Rashid FJ, et al. Deprescribing proton pump inhibitors. Evidence-based clinical practice guideline. *Can fam Physician* 2017;63:354-64 (Eng), e253-65 (Fr).

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