COMMUNICABLE DISEASES



Second edition

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A Manual for

Primary Health Workers

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Communicable Diseases

A Manual for Primary Health Workers

First edition
by

Jan Eshuis, MD
Peter Manschot, MD

Second edition
revised by
AMREF staff
and
edited by

John Bennett, MB, ChB, DPH, FFPHM
Geoffrey A.B. Buga, MB, ChB, MMed, PhD

Illustrated by

Sister Jean Lorenz, SCMM
and
Rose Olendi, BA, DipEd

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Please address any correspondence to:
The Publishing Director
African Medical and Research Foundation (AMREF)
P.O. Box 30125
Nairobi
Kenya

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FIRST EDITION 1978

The expansion of training programmes for rural health workers has made obvious to all involved the need for suitable books adapted to the local environment. The work of drafting this manual has been shared by many people. Copies of the drafts were circulated to all medical assistant training centres, and discussed at workshops held at Mwanza in 1975, Moshi in 1976, and Dar es Salaam in 1977, and we are grateful to all the following who have spent time and energy discussing the drafts and making suggestions for improvement: Drs Bhachu, Brooke, Dahoma, Georgiev, Hart, Huenges, Kagimba, Kisanga, Massawe, Mavura, Mbilu, Mkumbwa, Ngoda, Safe, Seda, Shao, Swai, Vaughan, and the members of the National Leprosy Advisory and Co-ordinating Committee.

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Finally, our thanks go to our students, who by their continuous requests for the drafts gave us the stimulus to go on with the book in addition to our normal duties.

Jan Eshuis
Peter Manschot

SECOND EDITION

The original text of the first edition remains the backbone of this manual, so the acknowledgements of 1978 retain their importance.

In this edition, the assistance of past and present AMREF staff is acknowledged, in particular that of Drs Christopher Wood, Philip Rees, Jane Carter and Christopher Nevill. We also acknowledge the contribution of Dr G. J. Steenbergen who kindly revised the chapter on Tuberculosis and Leprosy. We thank Connie Amri and Alice Kivuvani for their wordprocessing skills, and Caroline Agola and Joseph Mbuthia for their editorial skills. Many World Health Organization and other publications have been consulted and the following sources of illustrations are gratefully acknowledged: Figures 4.18, 4.22, 4.24 and 5.16 from *Principles of Medicine in Africa* (2nd edition), Oxford University Press, 1984; Figure 8.9 from Y. Mpairwa and H. L. Malovu, "Cerebrospinal meningitis in East Africa 1911–1962" *Trans. R. Soc. Trop. Med. Hyg.* 65 (1): 70–77 (1971).

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John Bennett
Geoffrey Buga

FOREWORD

'Communicable' and vector-borne diseases are still the most common killing diseases in Africa — and most are preventable. A revised and up-to-date edition of this very valuable practical manual is therefore especially welcome. It combines the necessary information for diagnosing and managing individual patients and the appropriate practical methods for preventing and controlling these diseases.

Since the first edition was published more than ten years ago smallpox has been eradicated from the world. It took more than a century and a half from the discovery of the means to vaccinate against smallpox and the mounting of a worldwide programme to ensure that it was eradicated. There are now prospects for similar worldwide campaigns to bring about the extensive control of poliomyelitis, measles and guinea worm disease, as well as the means available for a considerable reduction in many other diseases.

However, while increasing literacy and the spread of immunization programmes into the rural areas has led to decreasing infant mortality rates, these benefits are now threatened by overpopulation, increasing urbanization and the appearance of a new, so far incurable, disease — AIDS.

When people live close together the threat of communicable disease increases. Overcrowding in poor housing, pollution of water and the surroundings by inadequate disposal of human waste and rubbish contribute to the spread of disease. And, on a larger scale, the degradation of the land by overgrazing and soil erosion and the destruction of forests are leading to even greater problems of inadequate food and water supplies for the future.

As populations grow, therefore, the role of the environment in which we live and die becomes ever more important. The greatest threat to the environment is now seen to stem from the rapidly increasing population.

As we extend our control over the immediate causes of suffering and death from communicable diseases, we must at the same time look ahead and combine these activities with measures to increase public knowledge and responsibility for limiting future population growth and conserving the earth's natural resources.

Christopher H. Wood, MD
formerly Director General, AMREF, Nairobi

May 1992

TO THE READER

This book has been written for you, the primary health workers in health centres and dispensaries of Africa, who are faced with the task of giving primary medical care to farmers and other workers. The majority of the African population expects help and advice from you. Many of you have to bear this heavy responsibility without having access to medical books and periodicals, and without the opportunity to ask advice from other professionals. We hope this book will help you.

The sections of the book on management and treatment are written with the facilities available in health centres in mind, but for the benefit of those whose health centres are far away from a hospital, or who are unable to refer patients because of transport problems, the basic treatment provided in a hospital has also been included.

As well as treating disease, rural health workers must concern themselves with preventing it. This is achieved mainly through health education, immunization programmes, and environmental sanitation. To organize and run such programmes successfully, the interest and co-operation of both community leaders and of staff in Ministries outside the medical field is essential. Suggestions on how this interest can be stimulated are included in the "Action" section for control of each disease.

Another responsibility of the rural health worker is the early detection and reporting of outbreaks of epidemic diseases. By detecting these in time and taking the appropriate immediate action many lives can be saved.

We have tried to base the contents of this book on currently prevailing concepts. Medicine is a dynamic science, however, and what is advanced today may be old-fashioned tomorrow. So journals and periodicals must be read to keep in touch with new developments.

If this book stimulates you to keep in mind the health and medical needs of the whole community rather than just satisfying the demands of a few, then it will have served the purpose for which it was intended.

Jan Eshuis
Peter Manschot
John Bennett
Geoffrey Buga

September 1978
April 1992

Chapter one

Introduction

Diseases can be divided into a few large groups according to their main causes — see Table 1.1.

Most of the common diseases in Africa are environmental diseases and are due to infection by living organisms — viruses, chlamydiae, rickettsiae, bacteria, fungi, protozoa, or metazoa. These are called communicable diseases because they spread from person to person, or sometimes from animals to people. Together with malnutrition, they are today the major cause of illness in Africa. The communicable diseases occur at all ages but are most serious in childhood. These diseases are, to a great extent, preventable. In countries where they have been largely prevented, other conditions such as accidents and the degenerative and malignant diseases, which occur mainly at an older age, have taken their place and become the commonest.

Communicable diseases are very important in Africa because:

1. Many of them are very common;
2. Some of them are serious and cause death and disability;
3. Some come as widespread outbreaks, that is, epidemics;
4. Most of them are preventable by fairly simple means;
5. Many are particularly serious (and more common) in infants and children.

Table 1.1 Main groups of diseases, and their causes

Disease group	Cause	Example
Genetic	Inherited	Sickle cell anaemia
Deficiency	Shortage of intake of essential nutrients	Protein-energy malnutrition Iron deficiency anaemia Iodine deficiency disorders
Environmental	Most commonly due to organisms	TB, tetanus, malaria, worms
	Sometimes due to physical factors	Heat stroke
	Sometimes due to chemical factors	Insecticide poisoning
	Sometimes due to social factors, e.g., stress	Anxiety, depression
Degenerative	Due to wear and tear of the body	Heart failure of old age Osteo-arthrosis
Metabolic	Due to disordered biochemistry in the body	Diabetes
Malignant	Change in cell growth due to known or unknown causes	Cancer of the liver Cancer of the cervix

ORGANISMS AND AGENTS OF DISEASE

The living organisms that cause communicable diseases are of different sizes and sorts. The largest, like tapeworms or the filariae, are visible to the naked eye. They are made up of many cells and are called metazoa. Complicated but single-cell organisms, like malaria parasites and amoebae, are called protozoa. They are smaller and can only be seen when magnified by a microscope.

Smaller still are bacteria which are simple, single cells best seen under a microscope after they have been stained with dyes. Rickettsiae and chlamydiae are smaller and can only multiply within cells. Smallest of all are viruses which cannot even be seen with an ordinary microscope.

PATTERNS OF COMMUNICABLE DISEASE

Different diseases are common in different places and at different times. To understand why this is so, we need to consider the living organisms of disease, the *agents*; the people they infect, the *hosts*; and the surroundings in which they live, the *environment.*

The agents need a suitable environment in which to grow and multiply and thus be able to spread and infect another host. If they are not successful in doing this they die out. There is therefore a balance between the agent, the host and the environment which can be shown diagrammatically, as in Figure 1.1.

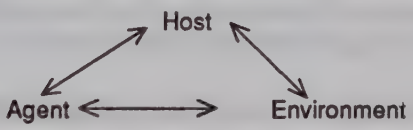

Figure 1.1 **The host, agent, environment triad**

Hosts (people) are affected by their environment. For example, they may live in a hot and wet climate in which there are many mosquitoes. But people can also change this environment by draining swamps. Similarly, the environment can affect the agent, for example the altitude and temperature for malaria.

When the balance between these three is constant, there will be a fairly steady number of people getting sick all the time. When this happens, a disease is said to be *endemic*.

Where the balance is shifted in favour of the organism, for example when many non-immune children have been born in an area since the last measles epidemic, a large number of cases of measles will occur in a short time. This is called an *epidemic*. When all the non-immune have had the disease it will die down again.

If the balance can be shifted against the agent, the disease will be controlled and the number of cases will go down.

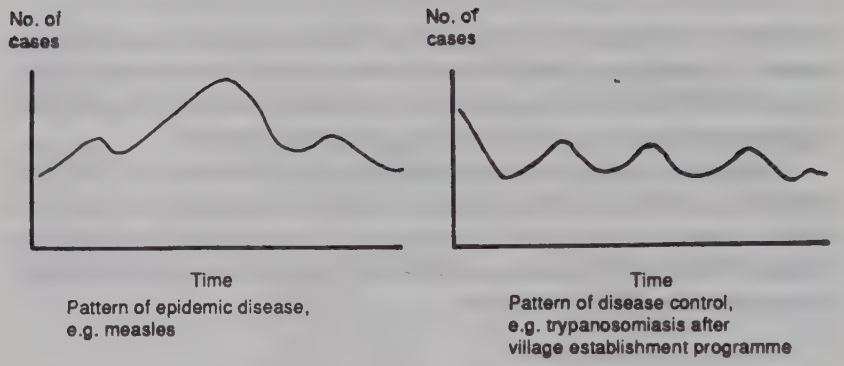

Figure 1.2 **Example of graphs showing the number of recorded patients in an epidemic and after a control programme**

THE MANAGEMENT OF COMMUNICABLE DISEASES

The proper management of communicable diseases involves both trying to stop people getting diseases (*prevention*), and looking after those who have them (*treatment* and *cure*). The two are closely related and together amount to control. Doing one without the other is doing only half the job.

To be able to prevent communicable disease it is necessary to understand the factors which affect the balance between the host, the agent, and the environment, and to know what can be done to tip the balance in favour of the host.

THE HOST AND INFECTION

Most of the communicable diseases in Africa have people as their main host. There are, however, a few important animal infections which may sometimes spread to humans, such as rabies and plague. These diseases are called *zoonoses*.

When an organism infects a person there are several stages to consider. The time between infection and the appearance of symptoms and signs of illness is called the *incubation period*. The shorter the incubation period, the more rapidly the disease can spread or die out in the community. Infection with certain organisms nearly always

leads to detectable symptoms and signs — *a clinical infection*. Others are able to infect people without always producing obvious symptoms or signs; these are called *subclinical infections*. This is important because people with symptoms and signs are ill, and therefore, they come for help from the medical services, or medical workers can find them. But people with subclinical infections do not know they are infected and that they are a danger to other people. It is also very difficult to detect them in the general population without special tests. These people are called *carriers* because they have a subclinical infection and are carrying or excreting organisms which are dangerous for other people. This can occur during the incubation period (e.g., measles with a short incubation period, or HIV infection leading to AIDS after a long period), or following recovery (e.g., typhoid).

Table 1.2 **Possible results of invasion by a micro-organism**

Invasion by a micro-organism	Clinical infection — with symptoms and signs. Subclinical infection — without symptoms or signs, but spread may occur by transmission of the organism by the infected person. No infection. The organism is killed by the person invaded. No danger.

People are susceptible to many diseases. They may develop resistance to some diseases. The *resistance* of the human body is provided by its various defence mechanisms: the defensive properties of the skin structure and special antibodies, antibacterial action of secretions (both chemical and antibodies), white blood cells and immunity. Immunity is due to special body cells and to antibodies circulating in the blood (see immunization chapters in AMREF's *Community Health* and *Child Health* manuals). Immunization procedures are an artificial way to raise people's resistance to certain infections by giving them vaccines.

The *reservoir* of the infection is the animal or place in which a particular organism usually lives and multiplies. For most of the

important communicable diseases, man himself is the main reservoir. For brucellosis, it is cattle; for rabies, wild animals; and for a few, e.g., tetanus, it is the soil. The *source* of the infection is the animal or place from which the particular organism spreads to its new host.

The way in which an organism leaves the infected host or source and travels to a new susceptible person is called the *route of transmission*. Each disease organism has particular routes which play a large part in how these organisms spread in the community. For example, some organisms are spread through water and food, while others are spread by vectors like mosquitoes and snails.

THE TRANSMISSION CYCLE

The *transmission cycle* describes how an organism grows, multiplies, and spreads. In some cases, man may be the only host, in which case the infection spreads directly from man to man, e.g., measles.



Figure 1.3 **Transmission cycle of measles**

In other cases, man may be the final host from whom the organism has no chance to pass further, e.g., tetanus.

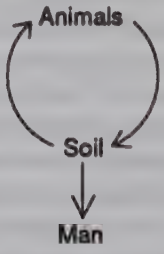

Figure 1.4 **Transmission cycle of tetanus**

But usually, man is an integral part of the transmission cycle as well as being the main reservoir, e.g., malaria or schistosomiasis.

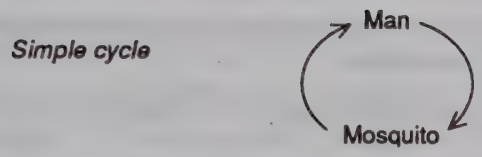

Figure 1.5 **Transmission cycle of malaria**

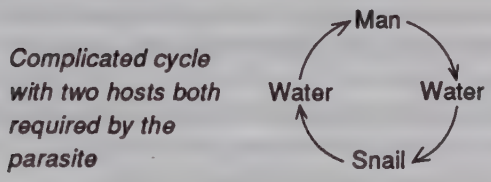

Figure 1.6 **Transmission cycle of schistosomiasis**

The three parts of the transmission cycle for an agent or organism can be illustrated thus:

Source —> Transmission —> Susceptible host

Source

The source of an infection can be an infected person or animal or soil. People and animals may have clinical diseases, subclinical infection or be carriers. If there is a reservoir, it should be considered with the source.

Transmission

The main routes of transmission are:

- Direct contact (skin contact, mucous membrane, sexual contact);
- Vector;
- Faecal contamination of food, and water, which are ingested;
- Contact with animals and their products (e.g., biting);
- Airborne (inhalation);
- Transplacental (during pregnancy);
- Blood contact (injection, surgery, blood transfusion).

Susceptible host

A susceptible host is one with low resistance to the particular infection. Low resistance may be due to:

- Not having met the organism before and therefore not having any immunity to it. For example, at the age of 6 –12 months a child loses the passive immunity against measles that he acquired from his mother during pregnancy. When he comes in contact with another child who has measles, he will develop the disease because he no longer has immunity against measles, unless he has had active immunization;
- Having another serious illness like AIDS at the same time; such people have a high risk of developing tuberculosis;
- Malnutrition, which can make infections worse.

PRINCIPLES OF COMMUNICABLE DISEASE CONTROL

The aim of control is to tip the balance against the agent. This may be done by:

- Attacking the source;
- Interrupting the route of transmission;
- Protecting the host.

Table 1.3 **Main methods of communicable disease control**

1. *Attacking the source*	2. *Interrupting transmission*	3. *Protecting the host*
Treatment of cases and carriers	Environmental sanitation	Immunization
Isolation	Personal hygiene and behaviour change	Chemoprophylaxis
Reservoir control	Vector control	Personal protection
Notification	Disinfection and sterilization	Better nutrition

Attacking the source or reservoir

Treatment of cases

If cases can be treated with drugs that destroy the organism, then fewer (or none) are available to spread to new hosts. The effectiveness

of treatment as a control measure depends on how many of the cases in the country can be reached and whether the treatment affects the agent's capacity to reproduce (for example, chloroquine has no gametocidal action). Treatment is an important method in the control of tuberculosis and leprosy, and most sexually transmitted diseases.

Subclinical cases and carriers

For treatment to be effective, subclinical *cases* and *carriers* must also be treated. However, special efforts have to be made to find them first as they do not usually present with any apparent illness, e.g., subclinical infections of cholera, or ankylostomiasis, or asymptomatic sexually transmitted diseases.

Mass treatment

Where a high percentage of the population are known to have a disease, it is sometimes advisable to treat everybody, without checking whether individuals have the disease or not. This is called *mass treatment* and has been used, for example, in the treatment of schistosomiasis in school children.

Isolation of cases

Isolation means that the person with the disease is not allowed to come into close contact with other people, except those who are looking after him. Therefore the organisms cannot spread. Isolation is used to control highly infectious and serious conditions such as haemorrhagic viral fevers (e.g., Marburg and Lassa fever).

Isolation is very difficult to enforce, however, and has a number of disadvantages. In particular, people are frightened of being isolated and this stops them coming for treatment, and so increases the spread of the disease.

Reservoir control

In those diseases that have their main reservoir in animals, mass treatment or chemoprophylaxis or immunization of the animals can be carried out, e.g., in brucellosis. Other ways include separating man from animals, or killing the animals and so destroying the reservoir, e.g., plague and rabies.

Notifications and reports

Although these do not directly affect the source, notifications are an essential means of keeping a watch (*surveillance*) on the number of new cases and thereby monitoring the effectiveness of the control programme.

Notification means that you immediately inform the local health authorities (e.g., the DMO) that you suspect a patient is suffering from an infectious disease. The authorities can then take measures to have your suspicion confirmed and to prevent the disease from spreading.

Notification must be done immediately and by the most rapid means. When you do not have a telephone, it is best to send a messenger to the DMO's office.

Some diseases spread so quickly that they need international control measures. These diseases will be reported by the authorities to the World Health Organization (WHO).

Internationally notifiable diseases are:

- Cholera
- Plague
- Yellow fever.

In addition to these three diseases on the international list, national ministries of health also require notification of certain diseases in their own countries, e.g., meningococcal meningitis and acute polio.

Interrupting transmission

Environmental sanitation

Many organisms spread through contaminated food and water, particularly those that are dependent on the faeco-oral route. Other diseases are spread through refuse and dirty living conditions. The airborne diseases usually spread when housing is inadequate and people live in crowded rooms. The main methods of improving environmental hygiene are described in AMREF'S *Community Health* manual.

Personal hygiene and behaviour change

Many careless or unhygienic personal habits help to spread some diseases, particularly the contact diseases and those that may be

spread by faecal contamination of hands, food, soil and water. Changes in personal behaviour are often difficult to initiate (especially sexual behaviour) and require an educational process, as described in AMREF'S *Health Education* manual.

Vector control

Any organism that requires a vector, like a mosquito or snail, for its transmission cycle may be affected if the vectors are killed off or reduced. Methods of vector control include altering the environment so that it is unfavourable to the vector (e.g., draining swamps), using toxic substances (e.g., larvicides), and using other living organisms that attack the vector — biological methods (e.g., introducing larvae-eating fish into water where the mosquito vectors of malaria are breeding).

Disinfection and sterilization

These methods aim at destroying the organism when it is in the environment, e.g., the use of chlorine in wells, and sterilization of surgical instruments.

Protecting the host

Immunization

Immunization increases host resistance by strengthening the internal defences — antibodies, killer cells, etc. It is one of the most effective methods of control for some communicable diseases. Immunization plays a critical role in the control of many communicable diseases in Africa. For example, it was responsible for the worldwide eradication of smallpox. Communicable diseases in Africa which are now being controlled through immunization include measles, poliomyelitis, whooping cough, diphtheria, tuberculosis, hepatitis B and tetanus.

An Expanded Programme on Immunization has been assisted throughout the world by WHO and UNICEF. This programme has moved towards universal coverage of immunization in infants and children.

To be effective in control of a disease in a community, immunizations have to be given to a high proportion of the people — 80% or more (80% coverage). The protective effect obtained when a

high proportion of the population has been immunized is called *herd immunity*.

Herd immunity may be produced by:

- Many people having a disease (active natural immunization);
- Giving immunizations to most of a population (active artificial immunization).

Chemoprophylaxis

Drugs that protect the host may be used for suppressing malaria, and for preventing infection with such diseases as plague and meningitis.

Personal protection

The spread of some diseases may be limited by the use of barriers against infection, e.g., shoes to prevent entry of hookworm from the soil, nets and insect repellents to prevent mosquito bites. These measures require health education for the community and individuals.

Better nutrition

Malnourished children get infections more easily and suffer more severe complications (e.g., diarrhoea). Also, infections occur during famines where people crowd together for assistance making it easy for the disease to spread and result in epidemics.

Effective control of a disease is most likely when a combination of methods — attacking the source, interrupting transmission, and protecting the host — is used

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STARTING A DISEASE CONTROL PROGRAMME

In almost all areas there are diseases which could be reduced by relatively simple measures. The difficulty is to know how and where to begin.

One of the most important starting points is the understanding that something can be done. Too often, people are unaware that high

morbidity and mortality rates can be reduced. Much can be achieved both by preventive measures and by prompt simple treatment of, for instance, malaria, diarrhoea, pneumonia and other diseases. However, without the understanding and active co-operation of individuals and communities, few disease-control programmes will succeed.

Before a communicable-disease control programme starts, it is best if the following can be done:

1. Study the problem in your area or community and find out all you can about the disease

Start with a survey to find out the extent and distribution of the disease and what people think and do about it — this is called *community diagnosis*.

2. Prepare yourself for a programme

Study the information collected. Work out with the health team what needs to be done and discuss this with community leaders. Make sure that the necessary equipment, drugs, and transport are available, and that the staff have the necessary training.

3. Make a plan with your community

Make sure that the necessary understanding and motivation are there so that there will be community participation. Ensure that you have the support of other health workers, e.g., extension officers in community development, information, education and water development.

4. Follow-up

Make a plan to record information. Measure what progress is made and then follow and compare the situation with what it was before. This helps you to see what is being achieved — that is, *monitoring and evaluation*.

APPLICATION OF COMMUNITY CONTROL MEASURES

The actual application of the control methods can be undertaken by different groups of people and the responsibility for them is best thought of at three levels: individuals and villages; dispensaries and health centres; and district and other higher levels.

Individuals and villages

Individuals and groups of people at the village level are responsible for:

- Completing immunization;
- Personal, household and food hygiene;
- Protective barriers — shoes, mosquito nets;
- Chemoprophylaxis — malaria;
- Avoiding infected sexual contacts, using condoms;
- Protection of water supplies and boiling or filtering water;
- Building and use of latrines;
- Rubbish collection and disposal;
- Vector control — clearing the surroundings, drainage;
- Avoiding bilharzial water;
- Eliminating rats and fleas.

Health centres

Health centre and dispensary staff should support and encourage community-based disease control programmes, and with help from the District Health Management Team (DHMT) should also ensure:

- Increasing immunization coverage;
- Reservoir control;
- Larviciding, molluscicide;
- Water protection and purification;
- Inspection of food supplies, markets and shops;
- Sanitary control of public toilets;
- Information, education and communication on health promotion and disease prevention;
- Notifying disease.

District, regional, and central ministry services

Staff at these higher levels are responsible for:

- Mass immunization campaigns;
- Mass chemotherapy;
- Vector-control schemes;
- Major water schemes;
- Radio and mass media information programmes;
- Health legislation;
- Research into control methods;
- Famine relief/refugee camps;
- Emergency epidemiology and control teams;
- Continuing education of staff.

All these activities need supporting health education

The best communicable-disease control programmes are those which involve the community. Thus, the nearby dispensaries and health centres have a vital role to play in stimulating community activities in their catchment areas.

First, arouse awareness, and then, supply the necessary technical support

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Chapter two

Contact Diseases

INTRODUCTION

Contact diseases are diseases that are transmitted by direct or indirect contact.

Direct contact is by skin-to-skin contact, for example by touching an infected person.

Indirect contact is through the handling of contaminated objects such as clothing, bedding, dressings, and eating utensils.

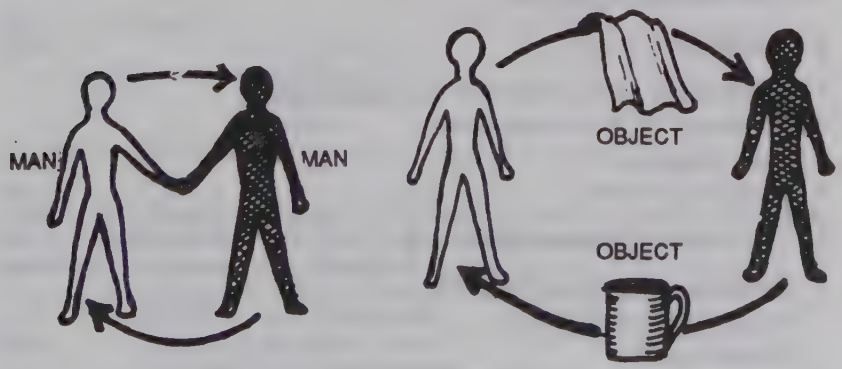


Figure 2.1 Transmission of contagious diseases



Figure 2.2 **Scarcity of water contributes to disease transmission**

Contagious diseases are diseases which are transmitted through direct contact. These diseases tend to occur in *clusters* within households, children's play-groups, schools, and factories. Such groups of infected people are called "clusters".

Transmission of contagious diseases is encouraged by:

- High population density (urban areas);
- Overcrowding (poor housing);
- Poor personal hygiene; encouraged by poor water supply or having to fetch water from long distances, as often happens in dry rural areas;
- Close personal contact, e.g., during sexual intercourse.

Control of contagious diseases can be directed at:

- The source
- The route of transmission
- The susceptible person.

1. The source, e.g.,

- Elimination of the reservoir by case-finding;
- Treatment of individual cases;
- Mass treatment;
- Isolation of cases.

2. The route of transmission, e.g.,

- Environmental improvements;
- Proper refuse disposal;
- Water supply;
- Improved living conditions;

- Better housing; correction of overcrowding;
- Personal hygiene and education.

3. **The susceptible person**, e.g., personal protection, and education with behaviour change.

Table 2.1 Diseases transmitted by contact

Organism	Diseases	Remarks
Arthropods	Scabies Pediculosis — lice Other infestations, e.g., fleas, bedbugs	See *Note* below (Some of these are not contagious but are considered here for convenience.)
Fungi	Ringworm • tinea capitis • tinea corporis • tinea pedis • tinea unguium Candidiasis	 Vaginal and mouth
Bacteria	Impetigo	See diseases caused by streptococci and staphylococci in Chapter 8 on Airborne Diseases
Chlamydiae	Trachoma	
Spirochaetes	Yaws	Eradicated from eastern Africa
Other	All diseases which can be transmitted through sexual intercourse	See Chapter 3 on STDs

Note
Infestation is the lodging, development, and reproduction of arthropods on the surface of the body or in the clothing. Insects which may do this on the human body are:

Human lice	1. The body louse (*Pediculus corporis*) 2. The head louse (*Pediculus capitis*) 3. The pubic louse (*Phthirus pubis*)
Fleas	1. The human flea (*Pulex irritans*) 2. The jigger flea (*Tunga penetrans*) 3. Rat fleas (*Xenopsylla* spp.)
Flies	The larvae of the tumbu fly and, under special conditions, larvae of other flies develop in the tissues of warm-blooded living animals, including man.
Mites	Scabies (*Sarcoptes scabiei*).

SCABIES

1. Scabies is a parasitic infestation of the skin characterized by severe itching with a typical distribution.

2. Occurrence and importance

Scabies is common in rural Africa. The prevalence in some villages is very high, especially if there is shortage of water. Infection and symptoms are more severe in children than in adults.

3. Epidemiology

Scabies is caused by a small arthropod, the itch mite, *Sarcoptes scabiei*. The female mite enters the skin and makes a small tunnel or burrow. The burrow is always superficial, never below the horny layer of the skin. The skin selected for burrows is always thin and wrinkled, giving the scabies rash its typical distribution. In the burrows, eggs and faeces are produced. The eggs hatch in 4–5 days. The larvae leave the parent tunnel and bury themselves in the skin in other places, but they do not make tunnels.

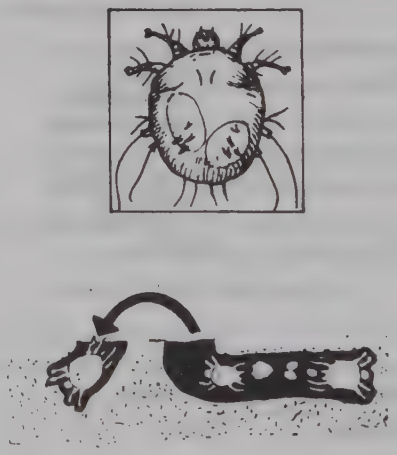


Figure 2.3 **The itch mite (*Sarcoptes scabiei*) and female mites in burrows in skin**

The infection is spread by direct close body contact, as in bed, or in the contact between children and their parents, and between children playing or in school. Transmission is also possible indirectly through bedclothes and clothing. Low socio-economic conditions favour the spread of this disease.

Scabies is a disease of whole families

Scabies is not a very contagious disease, and is uncommon in people who bathe regularly.

4. Clinical picture

Very often patients with scabies do not seek medical attention. The skin lesions may be so common that they are not considered to be a disease.

The skin lesions itch severely, especially at night; this itching leads to scratching. So, secondary infection with bacteria is almost inevitable. The diagnosis is made by finding an itchy rash with the typical distribution on the body (see below).

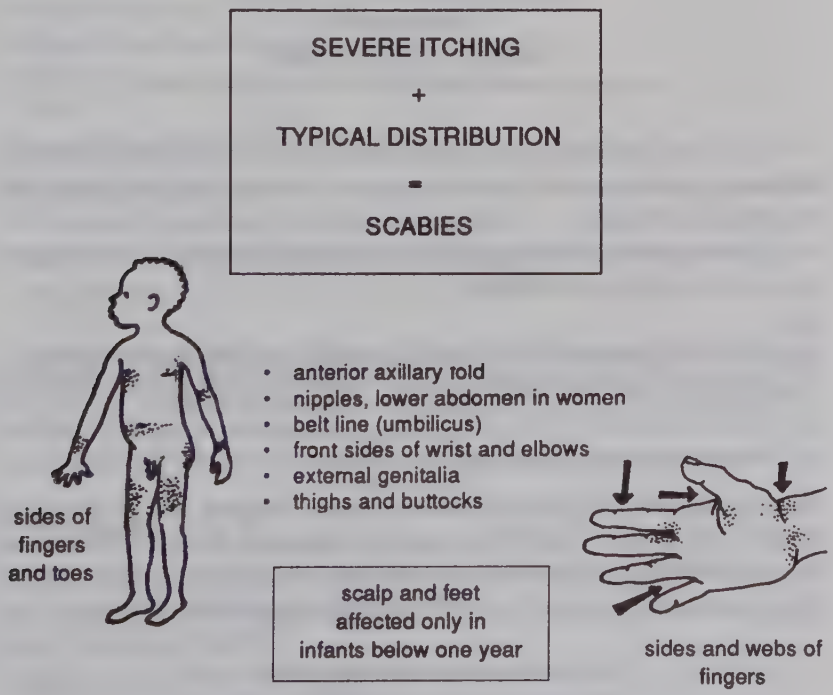


Figure 2.4 **Commonest places for infestation**

Note

Patients who are suffering from leprosy or other diseases which interfere with normal sensation may not feel the itching caused by scabies. In these cases, scabies can be very extensive and thick crusts can form.

5. Management of the individual patient

The drug most often available is 10% benzyl benzoate emulsion (BBE). After the patient has taken a warm bath, a handful of BBE should be rubbed over the whole body by the mother or a nurse, preferably using her bare hands. After 24 hours the patient should bathe again and put on clean clothes. BBE has little effect on the eggs; therefore, treatment should be repeated after 4–7 days to kill those larvae which have hatched since the first treatment.

Bathe and rub BBE over whole body
Bathe again after 24 hours
Clean clothes
Repeat after one week

The itching will not disappear immediately. If necessary, treat the itching symptomatically with calamine lotion. Do not repeat the treatment too soon.

Tetmosol solution or soap is a more convenient alternative treatment but it is not always available.

Note

1. In infants, the scalp should also be treated for scabies, but protect the eyes carefully.
2. In some patients, secondary infection is so severe that a course of PPF is advisable. The penicillin has no action, however, against the scabies mites themselves.

6. Prevention and control

Regular bathing, washing of clothes, and frequent use of soap will control the disease. Health education, stressing the use of soap and regular bathing of children, is essential in combating this disease. Piped water supplies will greatly help to reduce the incidence.

Soap and water control scabies

Treatment of a single child infected with scabies is useless. As soon as he returns home, he will be re-infected by the other members of the family.

Treat the whole family

Scabies lesions in an adult may not be visible clinically. Therefore, treat the whole household, even the apparently healthy. If Tetmosol soap is available, the simplest way to treat a family is to encourage every member to use it for bathing.

7. Action

If you treat scabies:

- Treat the whole body;
- Treat the whole family;
- Give health education at the same time.

If scabies is a problem in a village:

- Treat as many families as possible by making house-to-house visits and discussing personal hygiene;
- Do a survey of the children of the primary school. You can do this by just looking at the hands and wrists.

Stress the importance of improvement of water supply to the local development committee. When water supply improves, there should be less of other diseases caused by water shortage (e.g., trachoma, louse infestations, gastro-enteritis, dysentery).

8. Summary

Scabies is characterized by severe itching of typical distribution.
It is treated with BBE or Tetmosol.
Treat the whole family.
Regular use of water and soap will control scabies.

PEDICULOSIS

1. Pediculosis is the infestation of the scalp, hairy parts of the body, or clothing with adult lice (blood-sucking parasites), and their larvae or nits (eggs).

2. Occurrence and importance

The head louse is common in schoolchildren and people with long hair which is seldom washed.

Lice are especially common when people are crowded in bad hygienic conditions such as occur after natural disasters, in wars, and in refugee camps.

Pediculosis is more common in cold areas where people seldom wash or change clothes. The body louse is the vector of some important diseases like epidemic (louse-borne) typhus and relapsing fever. These two diseases have been reported in some countries of Africa, e.g., Uganda, Rwanda, Ethiopia.

Pubic (crab) lice do not transmit disease, but are spread by sexual or other close contact. In adults, they are an indicator of the possibility of other sexually transmitted diseases.

3. Epidemiology

Three different kinds of lice can cause pediculosis. The head louse (*Pediculus humanus capitis*) and body louse (*P. humanus corporis*) do not differ very much and distinction is of no practical value. Both are transmitted by direct contact with an infested person or his personal belongings, especially clothing.

Pubic lice (*Phthirus pubis*) are usually acquired during sexual intercourse.

Life cycle: The female parasite produces several hundred eggs daily, each of which is attached to a hair with a special glue which makes it very difficult to remove the eggs (called nits).

A larva hatches in 6–9 days and develops into a mature louse in 1–2 weeks.

4. Clinical picture

Lice cause irritating bites and itching, and this results in scratching which may be followed by chronic secondary infection.

Pubic lice live on pubic hair and sometimes other places; head lice on scalp hairs; and body lice on clothing, from which they bite the covered areas of the body. Impetigo of the head and neck and enlarged occipital lymph glands may be a manifestation of head lice; impetigo and enlarged lymph glands elsewhere may similarly be due to body or pubic lice.

5. Management of the individual patient

Head lice: Use any of the following preparations:

- Apply carbaryl to dry hair and rub into scalp and allow to dry; comb and remove by washing 12 hours later; repeat after one week; or
- Rub 0.1–1% lindane into hair and scalp; allow to dry and remove by washing after 24 hours; repeat after 7 days; or
- Rub 0.5% malathion lotion into dry hair and scalp; comb and allow to dry; remove by washing after 12 hours; repeat after one week.

Body lice: Take a hot bath and put on clean clothes; wash and iron clothing and bedding. Use any of the following preparations:

- Apply 25% benzyl benzoate to the affected area for 24 hours; remove by washing; repeat 2–3 times; or
- Rub 0.1–1% lindane on the affected area; allow to dry; remove by washing after 24 hours; repeat once per week; or
- Rub 0.5% malathion on the affected area; allow to dry; remove by washing after 12 hours; repeat after 7–9 days.

Crab lice: Use benzyl benzoate or carbaryl or lindane or malathion, as indicated for head lice. Shaving of hair is a common practice, but it is not strictly necessary. It is usually done in prisons.

6. Prevention and control

Health education on the importance of water and soap to maintain cleanliness, and the laundering of clothes to destroy nits and lice.

Case finding by direct inspection of bodies and clothing of schoolchildren, and adults living in camps.

Examination of household contacts.

7. Action

- Organize a school survey in co-operation with the headmaster;
- Examine household contacts of children with lice;
- Check all newcomers in refugee camps for lice as a routine.

8. Summary

Pediculosis is infestation with lice. The main medical importance of lice is their ability, in countries where these diseases are present, to transmit epidemic typhus and relapsing fever. The usual practical importance is the irritation they cause and the lack of cleanliness they indicate. Young adults with pubic lice should be suspected of also having a sexually transmitted disease.

OTHER ARTHROPOD INFESTATIONS

Fleas

Pulex irritans

Pulex irritans is the human flea; it breeds in dwellings and jumps on human beings to take a blood meal; it will also attack animals if no human is available. The bite causes an erythematous spot in the centre of which a petechia can be seen. Treatment is not necessary.

Control

Eradication of fleas of man and of domestic animals with pyrethrum. Periodic bathing of dogs.

Flea dermatitis (not contagious)

Allergic reactions to fleas and bugs can cause severe dermatitis, especially in children. It is characterized by small firm papules with an erythematous and sometimes swollen area around them. The condition may at first sight resemble chickenpox but the papules are firm and do not develop into vesicles and pustules.

Very often there is secondary infection and the differentiation from scabies may be difficult (but remember the distribution of scabies).

Treatment

Calamine to relieve the itching, and spraying or dusting with pyrethrum.

Tunga penetrans*

Synonym: Jiggers.





Position of flea in tissue

Figure 2.5 **Female jigger flea with swollen abdomen full of eggs**

Both male and female jigger fleas are blood-suckers of all warm-blooded animals. The male leaves its host after obtaining its meal but the females, when pregnant, burrow into soft skin, commonly between the toes. There they are in an environment rich in food, so they lay many eggs.

The hind part of the flea protrudes through the skin so the eggs can be released.

The result is a small, round, itching, painful tumour with a point (the hind part of the flea). Secondary bacterial infection is very common; even at times tetanus may result.

Treatment

This requires the mechanical removal of the flea with a sterile pin, followed by an antiseptic dressing. Application of kerosene will kill the flea but results in ulceration of skin until the dead flea is expelled.

Jiggers are common in some rural locations.

Prevention

This flea cannot jump very well. Therefore, only the feet are affected, and shoes provide protection. In children, other parts of the body may

*Jiggers and myiasis are not contagious diseases. Transmission is not possible direct from man to man as in other infestations, but is always through the external environment.

be infected (buttocks). Keeping a house clean will reduce contact between man and fleas. Pyrethrum as a spray or as a powder will kill the fleas.

Xenopsylla are tropical rat fleas. Sometimes these fleas may also feed on human beings and become the vectors of plague.

Bedbugs

Bedbugs are blood-sucking insects that are notoriously common in many poor communities; they are not known to cause any disease. After the bite an itching papule may develop, which disappears within several hours. Treatment is not necessary. Powdering beds and floors with insecticide is very effective. Bedbugs hide in the day time in cracks in walls, beds and furniture. They can also hide in clothing or personal belongings and can be brought into hospitals, clinics and dormitories. Therefore, couches and beds should be inspected periodically.

Control

Systematic dusting of the infected and neighbouring houses with insecticides.

Flies

Tumbu fly or mango fly (*Cordylobia anthropophaga*)



Figure 2.6 **Mature larva of tumbu fly**

The tumbu fly is an obligatory parasite causing myiasis. Myiasis is due to fly larvae invading living tissues. Three types of larvae can be distinguished producing:

1. **Obligatory myiasis**: the larvae can only develop in living tissues;
2. **Facultative myiasis**: the larvae usually develop in decaying tissues but may also invade necrotic wounds;
3. **Accidental or intestinal myiasis**: the eggs or larvae are ingested. They are not killed in the intestine and may even develop further.

Life cycle

The eggs of the tumbu fly are laid on dry soil, sand, or clothing which has been contaminated by the sweat or urine of man or animals, particularly rats, the normal hosts. The larvae hatch in 2 days. If a host comes into contact with the larvae, they are activated by the body heat of the host and penetrate the skin, producing a furuncle-like swelling. In this subdermal cavity the larvae will mature in 12 days. Contact with the host often happens through the eggs being laid on clothes, towels or napkins which have been left to dry in the sun and not properly ironed afterwards.

Treatment

Put Vaseline on the furuncle. The larva will try to come out to get air and, in so doing, will lubricate its hole. Now it can be gently squeezed out or pulled out with a pin.

Prevention and control

Ironing of clothes.

FUNGAL SKIN INFECTIONS (DERMATOMYCOSIS)

1. Dermatomycosis is a term applied to fungal infections of the skin and mucous membranes. Several types are identified according to causative organisms, site, and clinical appearance.

2. Occurrence and importance

Fungal skin infections are usually problems of appearance rather than illness, but it is important to distinguish them from leprosy and syphilis. They are sometimes indicators of immunosuppression as occurs in AIDS, cancer and tuberculosis.

3. Epidemiology

All fungi may be spread by direct or indirect contact. Genital infection (balanitis and vulvo-vaginitis) may be spread during sexual intercourse.

4. Clinical picture

See Table 2.2 page 32.

Tinea Infections

Synonym: Ringworm.

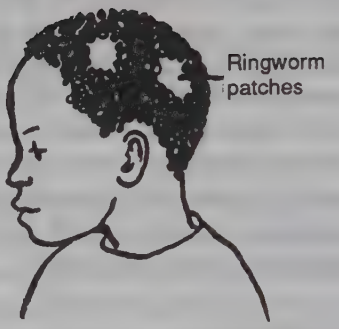


Figure 2.7 **Tinea capitis**

Tinea capitis (ringworm of the scalp) begins as a small papule which spreads to involve a larger area; hairs in the affected skin become brittle and break off easily. Occurs mainly in children under ten. It disappears after puberty.

Tinea corporis (ringworm of the body) is characterized by flat, ring-shaped, spreading lesions. The ring lesions are reddish, vesicular or pustular, and may be dry and scaly, or moist and crusted. The central area often clears, leaving apparently normal skin.

Tinea pedis (ringworm of the foot or athlete's foot) is characterized by scaling and cracking of the skin between the toes, particularly the fourth and fifth toes.

Tinea unguium (ringworm of the nails) is characterized by a thickening, discoloration, and brittleness of one or more of the nails. There is an accumulation of caseous material beneath the nail, which becomes chalky and disintegrates.

Tinea versicolor or *pityriasis* is a very superficial infection. The skin of the sides of the face, neck, and chest shows many irregular, round, light-coloured areas.

Candidiasis (see also section on AIDS)

Synonyms: Moniliasis, thrush, yeast infection.

There are four main manifestations:

(a) *Oral thrush:* White pseudomembrane (patchy white dots on a red background) on the mucous membranes of the mouth: the underlying epithelium is bright red; ulcers are rare; common in newborns, malnourished children or after antibiotic therapy, and in AIDS patients.

(b) *Vulvo-vaginitis:* Pruritus and vaginal discharge, thick and white.
(c) *Balanitis:* Itching and redness of the glans penis, swelling of the prepuce, sometimes phimosis; eventually secondary infection with ulcers.
(d) *Intertrigo:* In deep body folds, e.g., axillae, under the breasts, groins, anus. The skin is erythematous, moist, and eroded. The boundaries of affected areas are sharp; watch for predisposing factors (urine for sugar, AIDS).

Diagnosis

The diagnosis of fungal infection can be confirmed by laboratory investigations. The technique is easy and materials needed are simple. Clinical diagnosis is very inaccurate.

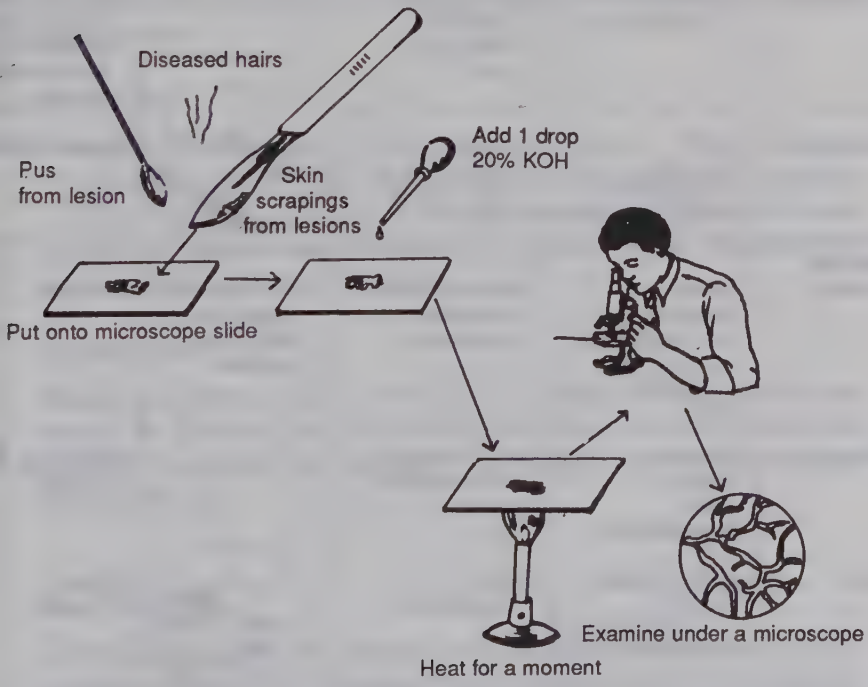


Figure 2.8 **Diagnosis of fungal skin disease**

Table 2.2 Characteristics of fungal skin diseases

Fungus	Risk factors	Main symptom	Diagnosis	Treatment
Ringworm				
Tinea capitis	Children under 10	Brittle hair; areas of broken hair on scalp		Whitfield's (Ung. acid. benz. co.) b.d. for 3 week
Tinea corporis	Excessive perspiration; hot humid areas	Ring-shaped lesions; central healing	*Branching filaments crossing borders of cells*	Keep dry and clean
Tinea pedis (athlete's foot)	AIDS	Scaling and cracking of skin between toes		Apply Whitfield's ointment daily
Tinea unguium	Nail injuries; corticosteroids	Thickening, dis-colouration or brittleness of nails		Surgical removal of nails plus Whitfield's ointment daily for 16 weeks
Tinea versicolor	AIDS Excessive perspiration; hot humid areas	Superficial small round light-coloured areas		Personal hygiene
Candidiasis				
Oral thrush	Measles; Newborn; Vulvo-vaginitis of mother after antibiotics; AIDS	Pseudomembranes on mucous membranes	*Short filaments, round thick-walled cells, budding*	G.V. paint
Vulvo-vaginitis	Pregnancy; Diabetes; AIDS; Post-antibiotics	Vaginal discharge white and thick, itching		Nystatin vag. tab. G.V. paint
Balanitis	Lack of personal hygiene (Acquired by sexual intercourse)	Itching and redness		Hygiene G.V. paint
Intertrigo	Fatness; Diabetes; Humid areas (armpits, breast folds, between toes)	Intertriginous areas of redness (like eczema)	*Long filaments, oval cells, budding*	G.V. paint (Diet in diabetes) Keep areas dry by exposure and talcum powder

5. Management of the individual patient

All patients with fungal skin diseases should be given health education on personal hygiene.

Scrubbing with water and soap, followed by the application of fungicidal drugs is the basis of treatment. Fungicides need to be applied for a long time—at least three weeks.

For specific treatment see Table 2.2 page 32.

6. Prevention and control

By general improvement of personal hygiene, and treatment of individuals. When many children with tinea capitis are seen, mass school treatment may be rewarding. Oral thrush of the newborn can be prevented by treating maternal vulvo-vaginitis in the third trimester of pregnancy.

7. Action

- Check your laboratory for diagnostic facilities.
- Check your knowledge of leprosy; do a KOH slide with appropriate skin diseases but also test for anaesthesia of skin in doubtful cases.
- Refer all skin diseases of uncertain diagnosis for investigation, but treat fungal infection yourself.

8. Summary

Dermatomycosis is caused by a wide variety of fungi. Infections should be differentiated from skin manifestations of leprosy and syphilis. Diagnosis and treatment can be done at health centre and dispensary level.

TRACHOMA

1. Trachoma is a chronic inflammation of the conjunctiva and cornea. It is characterized by follicular conjunctivitis followed by vascular invasion of the cornea (pannus), and in its later stages by scarring of the lids, with the eyelashes damaging the cornea and leading to blindness.

2. Occurrence and importance

Trachoma is a very important cause of blindness, causing severe, permanent disability and a lot of human suffering.

Trachoma is especially common in dry, dusty areas with poor hygiene, poverty, and crowded living conditions. It is mainly seen in areas where water shortage is a daily problem. The incidence of trachoma correlates well with mean annual rainfall.

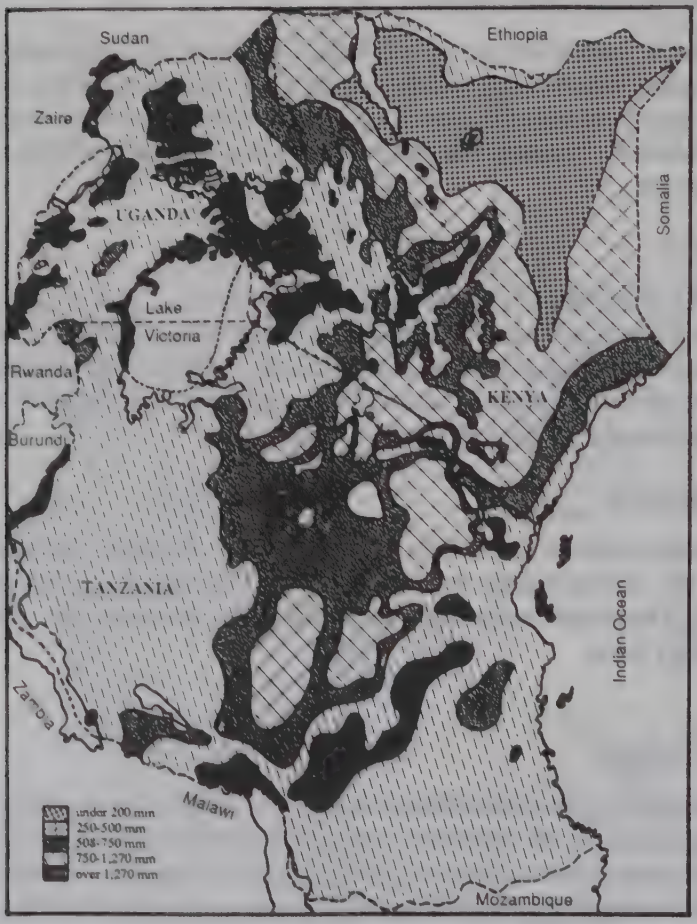


Figure 2.9 **Mean annual rainfall in eastern Africa**

3. Epidemiology

Trachoma is caused by certain serotypes of *Chlamydia trachomatis*, of the group of Chlamydiae. Other organisms belonging to this group cause inclusion conjunctivitis of the newborn and adults, non-gonococcal urethritis, cervicitis, salpingitis, epididymitis, atypical pneumonia of the newborn and lymphogranuloma venereum. It is now agreed that this agent is not a virus although it can only exist inside cells and can pass a bacterial filter. It differs from viruses in being sensitive to certain antibiotics, and in several other ways.

Transmission of trachoma occurs by direct contact with ocular discharges of infected persons.

C. trachomatis is present in large numbers during the early stages of the disease and disappears gradually.

Secondary bacterial infection increases the risk of corneal ulceration. If corneal ulceration occurs, scarring is more severe and more rapid, possibly resulting in perforation of the cornea and so leading to blindness in an early stage of the disease.

Flies, fomites (contaminated substances) and fingers are important in transmitting the secondary infection, but their role in the transmission of trachoma is uncertain.

In endemic areas children get the infection at pre-school age. Although the disease normally progresses very slowly, blindness by the age of 10 is not uncommon. Girls tend to be more affected than boys, probably because the boys more often leave the house where water is short, and get more opportunity for bathing.

Because the early stages are the most infective, transmission is high among children. Bacterial infection and Vitamin A deficiency exacerbate the disease and encourage scarring, leading to blindness.

4. Clinical picture

Trachoma develops in four stages.

Stage I

- Early trachoma begins with red, watery eyes like ordinary conjunctivitis.
- After a month or more, small pinkish-grey lumps, called follicles, form inside the upper lids. To see these, turn back the lid as shown in the drawing.

- The white of the eye is mildly inflamed.
- At this stage there is little pus unless bacterial superinfection has started. The pre-auricular glands also begin to enlarge.
- Confirmation can be made by demonstrating the chlamydiae inclusion bodies in scrapings from the conjunctiva stained with any Romanowsky Stain and/or Lugol's.

Stage II

- At this stage of the disease, if you look very carefully or with a magnifying glass, you may see that the top edge of the cornea looks greyish because it has many tiny red blood vessels in it (pannus).
- The combination of both follicles and pannus is almost certainly trachoma.

Stage III

- Trachoma is a self-limiting disease. After several years the follicles begin to disappear leaving whitish scars on the conjunctiva.
- In the cornea the small vessels retrogress. A diffuse haze remains on the cornea. Some vision may remain unless gross damage, like rupture of the cornea from corneal scars, has occurred.

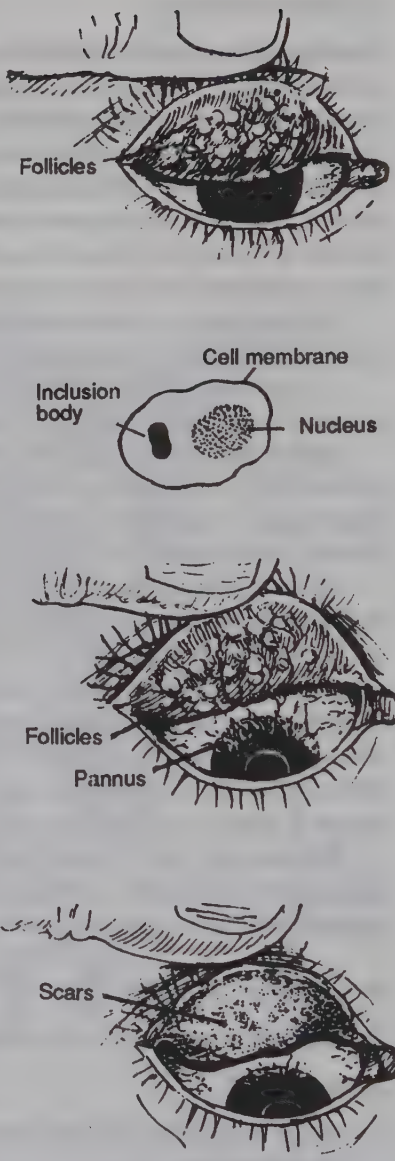


Figure 2.10 Stages in the development of trachoma

Stage IV

- It is the scar formed after the healing process, years after the onset of the disease, that does most damage. The scars are on the inner side of the conjunctiva.
- Scar tissue always retracts so the eyelid becomes thick and turned inwards. This is known as entropion.
- These scars make the eyelids thick and narrow, and may keep them from opening all the way, or they may pull the eyelashes down into the eye, scratching the cornea whenever the patient blinks. This is called trichiasis. The combination of entropion and trichiasis will completely destroy the cornea, resulting in blindness.

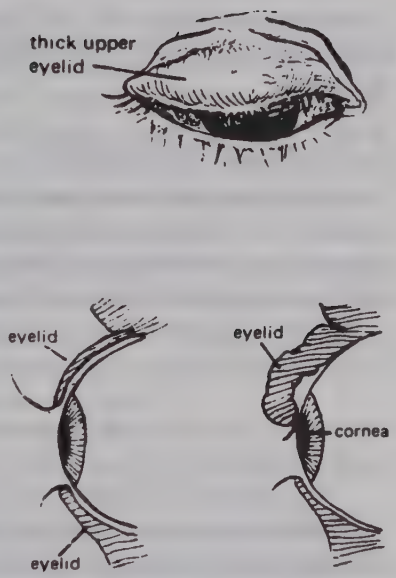


Figure 2.10 Stages in the development of trachoma

5. Management of the individual patient

C. trachomatis is sensitive to tetracycline, erythromycin and sulphonamides. The treatment of choice is 3% tetracycline topical eye ointment. This is stronger than the usual 1% tetracycline eye ointment used for general eye infections, and it stings, but it is important to use it whenever it is available because it is much more effective. (Sulphonamides are potentially dangerous drugs and can only be given under guidance and close supervision.) Effects of the treatment are slow and the success of treatment can only be judged after three months. One method of administration of the eye ointment is to apply twice a day for 5 days each month for 6 months.

Patients in stage IV of the disease with entropion must be treated surgically. It is essential to do this as soon as possible because every blink increases the damage. If no surgery is possible in your health centre, you can remove the inturned eyelashes by pulling them out with forceps.

This should be done before you refer the patient. Entropion operations are simple and can be carried out at health centre level, but pannus and opacity of the cornea have to be dealt with by an eye specialist.

6. Prevention and control

Trachoma is a clear example of a disease caused by lack of water. The quantity of water is more important than the quality. The most effective way of dealing with the trachoma problem is to have enough water near people's homes. (If water was supplied to every house it would eradicate trachoma completely.)

Improve water supply

Until such time as water supplies are improved, other preventive measures must be taken. Health education can teach people a few basic principles that will go a long way towards reducing the incidence of the disease.

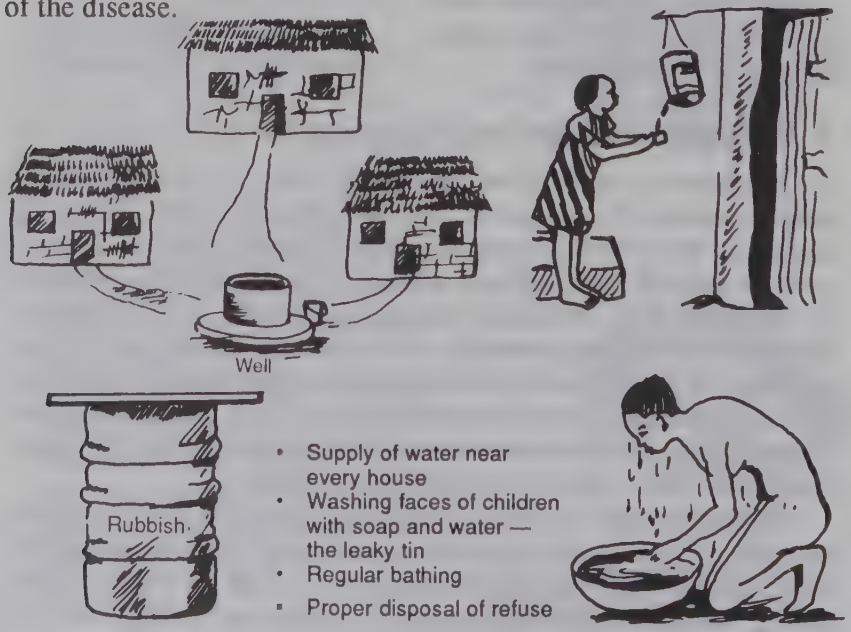


Figure 2.11 **Control of trachoma**

Health education

The importance of regular bathing, and especially of washing the faces of children with soap and water, should be stressed. The "leaky tin" is a good way to get the maximum hygienic benefit out of a little water. A tin with a small hole near the bottom, plugged with a small spike of wood, can be filled with water and hung up. When the spike is pulled out, a fine jet of water flows. This can be used to wash the face and eyes without contaminating the rest of the water.

Improper refuse disposal will encourage fly breeding. Reduction of the fly population will reduce bacterial superinfections, and also the severity of the disease.

Early treatment will reduce transmission because trachoma is most infectious during Stage I.

To detect as many cases as possible, school surveys should be done yearly in endemic areas. A school teacher can give treatment to all children regardless of whether they are already infected or not.

For such mass treatment WHO recommends the intermittent treatment schedule (application of 3% tetracycline eye ointment for 3–5 days per month for 6 months).

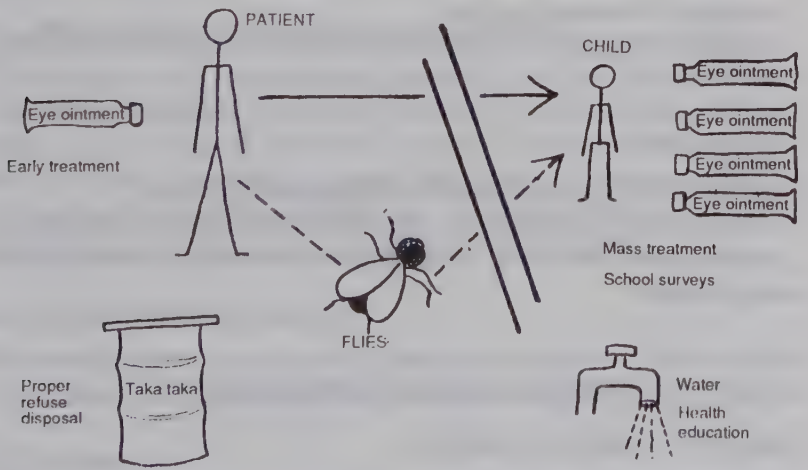
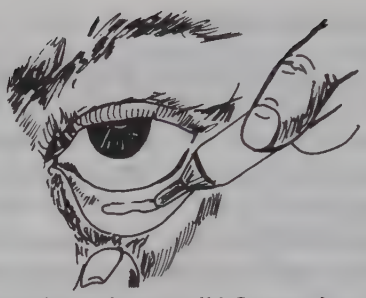


Figure 2.12 **Breaking the trachoma transmission cycle**

7. Action

When trachoma is a problem in the catchment area of your HC do the following:

- Arrange with head teachers for a day to do a school survey. If you find many trachoma cases, ask the help of the teacher in treating them. Explain to him how to clean the eyes and how to apply eye ointment;
- Supply the head teacher with enough tetracycline eye ointment to treat all children for 3–5 days each month for 6 months;



Pull down lower eyelid. Squeeze 1 cm of ointment into inner surface of lid without touching the cornea. Close eye.

Figure 2.13 **Putting tetracycline ointment in the eye**

- Stress to the local development committee the importance and the urgent need of improvement of the water supply to provide plenty of water nearby;
- Give health education talks at MCH clinics on the transmission of trachoma and how it can be prevented and treated. Stress the danger of the complications;
- Explain to the head teacher how to clean eyes and how to apply eye ointment.

8. Summary

Trachoma is an important cause of blindness in Africa.

Treatment with 3% tetracycline eye ointment should start early.

Main control measures include improvement of water supply, mass treatment.

ACUTE BACTERIAL CONJUNCTIVITIS

Synonyms: Pink eye, sore eye.

1. Acute bacterial conjunctivitis is a clinical condition starting with watery eyes and redness of the conjunctiva, followed by oedema of the eyelids and mucopurulent discharge.

2. Occurrence and importance

Conjunctivitis is common and widespread throughout Africa, particularly in hot and dry areas. It is usually self-limiting and does not lead to corneal or lid scarring.

3. Epidemiology

Transmission is by contact with ocular discharges or secretions from the upper respiratory tract of infected persons through contaminated fingers, clothing, and other articles.

Flies may transmit the disease. Transmission is favoured when living conditions are poor, where rainfall is low, and where dust and flies are plentiful.

4. Clinical picture

The patient presents with red eyes because of vascular injection of the conjunctiva. Usually there is oedema of the eyelids. The eyes are watery and the patient complains of a gritty feeling. There is no actual pain or loss of vision. Normally both eyes are affected. If this is not so, other conditions causing a red eye must be carefully excluded, particularly a foreign body in the eye.

5. Management of the individual patient

Cleaning the eyes regularly with water is often sufficient and should be the first priority in treatment. Penicillin eye ointment should not be used since it causes penicillin allergy. Chloramphenicol (or 1% tetracycline) eye ointment is best. Drops are more convenient for the patient but their action is short-lived. A relative of the patient should be instructed on how to apply the ointment or drops.

6. Prevention and control

Personal hygiene; care and treatment of affected eyes. Proper refuse disposal to prevent fly breeding; health education.

7. Action

When conjunctivitis is a problem, give health education in schools and at the health centre, stressing the importance of personal hygiene and the dangers of improper refuse disposal.

8. Summary

Conjunctivitis is caused by a number of different organisms, especially in hot areas where hygienic conditions are poor. Treat by cleaning the eye regularly with boiled water and with chloramphenicol or tetracycline eye ointment.

Note
Most cases of conjunctivitis are caused by viruses (conjunctivitis is a common feature in systemic viral infections, e.g., measles). They do not need application of antibiotics. Only frequent cleaning of the eyes is important. Bacteria are only likely to be the cause when there is a mucopurulent discharge, so only give antibiotic eye ointments to purulent conjunctivitis. In these cases, the patient must use the antibiotic ointment for at least five days. A single application of eye ointment is useless.

Antibiotic eye ointment only for purulent conjunctivitis

Chapter three

Sexually Transmitted Diseases and AIDS

INTRODUCTION

The communicable diseases belonging to this group are usually transmitted during sexual intercourse; hence the name sexually transmitted diseases, or STDs.

The number of germs known to be sexually transmitted has increased in recent years. Some of the diseases have already been covered in the previous chapter because they are contagious. During sexual intercourse there is close body contact, which is an ideal situation for transmission.

The causative organisms of STDs are very easily killed by drying or by changes of temperature. Therefore, transmission of these agents from one person to another can only occur under very special circumstances, mostly during sexual intercourse.

STDs are common in adults, but they are often hidden for fear of the opinion of others. Self medication is common and, when sought, treatment is often obtained from private practitioners, quacks, pharmacists, nurses, and traditional healers.* This results in under-reporting of STDs.

Single young men and women, as well as divorcees, are a high risk

*STD is the most important cause of illegal purchase and use of antibiotics.

group for STDs, as they may satisfy their sexual needs with many sexual partners. Certain occupational groups are also at high risk, and these include prostitutes, barmaids, soldiers, long-distance truck drivers, seasonal labourers and students. These may form reservoirs for STDs.

RISK FACTORS

1. Age

Fifteen years and older. The potential for sexual activity is greatest from adolescence to the third decade. The greatest risk occurs in the age group 18–35.

2. Marital status

Unmarried or divorced people are likely to change partners more often and are, therefore, more frequently exposed to STDs.

3. Occupation

Soldiers, prostitutes, taxi drivers, long-distance truck drivers, seasonal labourers, policemen and students tend to expose themselves to STDs more easily.

4. Residence

Due to industrialization and consequent urbanization, there is usually a large group of single, poorly paid and unemployed young people who live in shanties in towns. Sexual intercourse, for pleasure and for gain, assumes an important role in such circumstances. Prostitution and promiscuity flourish under these conditions and so do STDs.

5. Promiscuity

Promiscuous sexual behaviour is closely associated with the acquisition and spread of STDs in any group of people. There are certain factors whose presence may tend to reduce promiscuous behaviour in individuals. These factors are shown in Figure 3.1. The more of these factors an individual has, the less the risk of STDs.

CONTROL

STDs are difficult to control because the co-operation of those infected is needed, and this is often difficult to obtain.

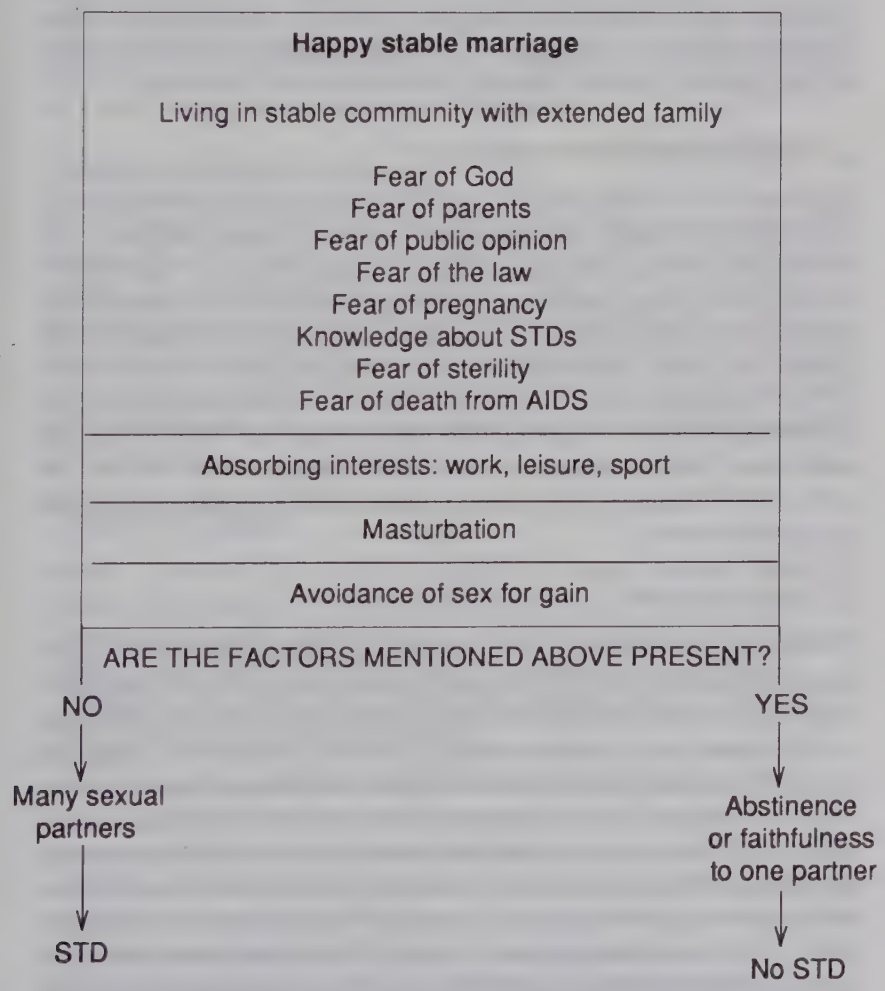


Figure 3.1 **Factors influencing absence or presence of STD**

1. Importance of early diagnosis and treatment

This is one of the most important measures and yet most difficult to achieve in many developing countries because of lack of resources such as money, trained manpower, laboratory equipment and drugs. Facilities for early diagnosis and treatment of STDs are not freely available and accessible to everyone. These facilities should be made available and freely accessible, and confidentiality strictly maintained, so that patients are not afraid to present for early treatment.

2. Elimination of the reservoir

The reservoir is exclusively human. It includes asymptomatic patients, both male and female, and untreated, or poorly treated patients, who have many sexual partners. Regular examination and treatment of known prostitutes, barmaids, soldiers or long-distance truck drivers would reduce the reservoir, but would not completely eliminate the risk of infection. Contact tracing of consorts of patients for treatment is very important, but this is particularly difficult in many developing countries because of population movement and lack of proper addresses. Urban migration, civil strife, droughts and famine are some of the causes of movement of people. STD transmission is high in these situations.

3. Sex education

There is a general lack of public awareness of the problem and lack of specific knowledge of STDs in all target groups. It is important to ensure that all children receive information about sexuality, STDs and AIDS before they become sexually active, so that they learn the necessary skills to adopt safe sexual behaviour and avoid promiscuity. This information can be imparted through school or family-life education. Children can learn about masturbation as a safe alternative to sexual intercourse before marriage. They should be told that masturbation is a normal activity and so it should not raise feelings of guilt, and that it does not cause disease. They should learn about the early symptoms and signs of STDs, and the need to present early for diagnosis and treatment. They should avoid self-medication, treatment by untrained personnel and chemoprophylaxis. They should know of the various dangers of STDs and AIDS and their effect on family life. They should know about the correct use of condoms.

4. Male motivation

It is important to reach male adults with information about STDs and AIDS because they are often the decision-makers about sex, and they may be having sexual partners outside marriage.

They must learn safe sex practices, including faithfulness to one partner, and the use of condoms. They should also know the early signs and symptoms of STDs and the complications which may arise due to non-treatment or inadequate treatment.

5. Women and development

Many women find it difficult to make sex safer because traditionally they do not have power to insist on safe sex in a male-dominated society. They are less educated, less well paid, and less employed in the formal sector than men. They are therefore open to sexual abuse. Women's development projects and education can help them to adopt safer sex by offering them alternative sources of income, thus strengthening their economic position to increase their negotiating power with men.

6. Other measures

Provide quarters for married couples in work camps; provide recreational facilities for soldiers, students, etc.

Encourage health units to use laboratory facilities for correct diagnosis so that they can give correct and adequate treatment.

What action can you take from your health centre to reduce the incidence of STDs in the community?

- Arrange the line of flow in your OPD in such a way that confidential service is guaranteed for every patient.
- Arrange with the head teachers and parent-teacher associations of the primary schools in your area for lectures to be given on sexual behaviour, family planning, and STDs to pupils in the last years of primary school before they leave. Do this every year.
- Give health education on STDs to groups at risk (students, soldiers, labourers, hotel and bar workers).
- Suggest periodical check-ups for STDs for barmaids and other women at risk. (This would be ideal, but is very difficult in practice.)

CLINICAL PICTURE

STDs show in three different ways:

1. STDs showing as genital discharge

- Gonorrhoea.
- Urethritis due to *Chlamydia trachomatis* and occasionally other bacteria and viruses (previously known as non-specific urethritis or NSU).
- Trichomoniasis.
- Candidiasis.

Note
Candidiasis in women is often not an STD.

2. STDs showing as genital sores or lumps

- Syphilis.
- Chancroid.
- Lymphogranuloma venereum.
- Condylomata acuminata (venereal warts).
- Herpes simplex.
- Molluscum contagiosum.
- Scabies.
- *Pediculosis pubis* (crab lice infestation).
- Granuloma inguinale.

3. STDs showing as disease elsewhere in the body (especially important in infants)

- Syphilis.
- Gonorrhoea.
- AIDS.
- Chlamydia infection.

It has been a frequent practice to diagnose all genital sores on clinical grounds as syphilis and all cases of genital discharge as gonorrhoea. This leads to inappropriate treatment.

A better approach is to make wet preparations and Gram stains of all cases of genital discharge, and to carry out serological tests on all cases of genital ulcers so that a proper diagnosis is made before treatment.

STDs of importance are listed in Table 3.1.

When the only generally recognized STDs were syphilis, lymphogranuloma venereum, granuloma inguinale, gonorrhoea, and chancroid, these used to be called the venereal diseases (love diseases), because they, with NGU (NSU), are only transmitted by sexual intercourse. The term STDs is preferred these days because it also includes other conditions which are mainly transmitted sexually but can also be transmitted in other ways.

Table 3.1 **The important sexually transmitted diseases**

	Causative agent	Disease
Bacteria:	*Treponema pallidum*	Syphilis
	Neisseria gonorrhoeae	Gonorrhoea
	Haemophilus ducreyi	Chancroid
	Chlamydia trachomatis	Non-gonococcal (non-specific) urethritis (NGU, NSU),
	Chlamydia trachomatis	Lymphogranuloma venereum (LGV)
	Donovania granulomatis	Granuloma inguinale
Viruses:	Herpes simplex virus	Herpes simplex of genitals
	Human papilloma virus	Condylomata acuminata
	Pox virus	Molluscum contagiosum
	Human immunodeficiency virus (HIV)	AIDS
Protozoa:	*Trichomonas vaginalis*	Trichomoniasis
Fungi:	*Candida albicans*	Candidiasis (moniliasis, thrush)
Ecto-parasites:	*Sarcoptes scabiei*	Scabies
	Phthirus pubis	Pubic lice

Note: Different serotypes of *C. trachomatis* cause NGU and LGV.

GONORRHOEA

1. Gonorrhoea is an acute or chronic purulent infection of the urogenital tract

Synonym: Abbreviated: "gc".

2. Occurrence and importance

Gonorrhoea is by far the commonest STD, both in urban and rural areas. It can cause sterility in both males and females and causes a serious decline in birth rate in some communities. "One-child sterility" is often seen in some parts of eastern Africa, and this is usually due to tubal obstruction caused by chronic gonococcal salpingitis.

Gonorrhoea in females may cause death due to salpingitis and peritonitis or predispose to ectopic pregnancy. In males, it may cause urethral stricture resulting in urine retention, hydronephrosis and uraemia, also causing death. The frequency of these fatal complications is not known. Gonococcal ophthalmia of the newborn may cause blindness.

3. Epidemiology

Gonorrhoea is caused by the gonococcus, a Gram-negative kidney-shaped diplococcus, which can only grow intracellularly. This type of gonococcus is known as *Neisseria gonorrhoeae*.

The gonococcus is not able to penetrate intact skin or squamous epithelium. It prefers columnar epithelium, such as in the urethra, endocervix, rectum and conjuctiva.

Contact of mucous membranes with gonococcal pus is usually only possible during sexual intercourse.

The following conditions are exceptions to this rule:

(a) *Gonococcal ophthalmia neonatorum*. This is an acute inflammation of the conjunctiva of the newborn. The infection is contracted during passage through the birth canal.

(b) *Gonococcal vulvo-vaginitis*.The vagina of adult women in the reproductive age is lined with squamous cells which contain glycogen. Through bacterial action this glycogen is metabolized into lactic acid, thus producing a low pH. This low pH protects the vaginal wall from invading gonococci. The glycogen content of

the cells is determined by oestrogen levels and is therefore low before puberty and after menopause. So before puberty and after menopause, the vagina is less resistant to gonococcal infections. This accounts for the fact that pre-pubertal girls and post-menopausal women can get vulvo-vaginitis, while women of reproductive age rarely do. The sharing of towels or clothing promotes the spread of the infection from one girl to another (the gonococcus can survive for up to 24 hours in a discharge on a moist surface such as a towel).

The gonococci multiply on the mucosal surfaces and subsequently the infection can spread to other structures lined with columnar epithelium, such as Littre's glands, Cowper's glands, the prostate, seminal vesicles and epididymis in the male, and Skene's glands and the fallopian tubes in the female. The infection spreads along the epithelial and subepithelial layers and generates a white blood cell

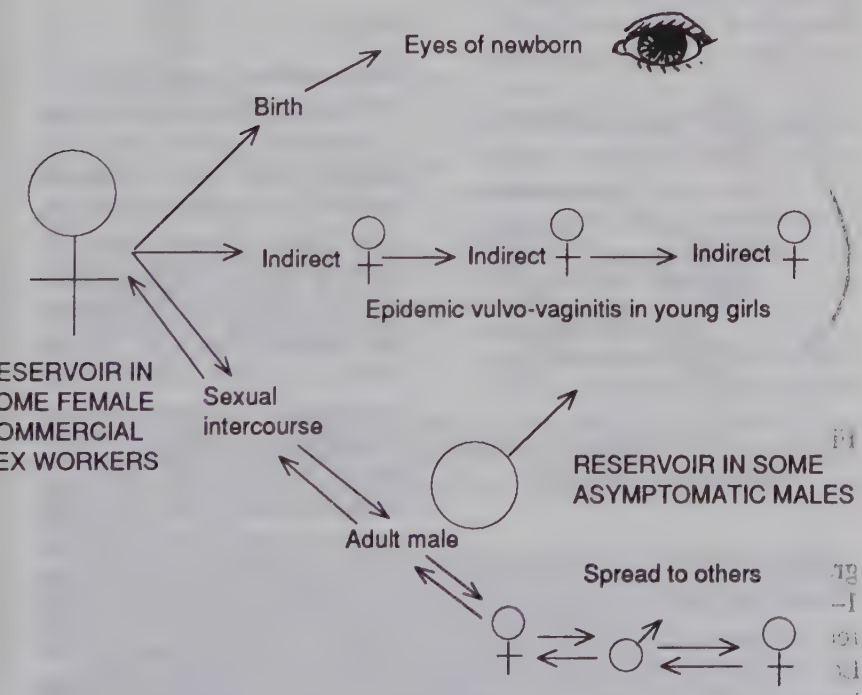


Figure 3.2 **Transmission of gonorrhoea**

(neutrophil) response which accounts for the pus discharge in acute stages in the typical case. Tissue response is often minimal in females, so that pus discharge may also be minimal. Resolution by fibrosis may occur in the male urethra, epididymis and fallopian tubes, if the inflammatory process is not treated adequately.

4. Clinical picture

The risk of infection after a single exposure to an infected partner ranges from 20 to 35% in males and from 60 to 90% in females. After an incubation period of 2–10 days, symptoms of urethritis develop in the great majority of infected men. However, asymptomatic urethral infection also occurs in men, and may persist for months if untreated. The asymptomatic male carrier is a reservoir of gonococcal infection and may also be at increased risk for disseminated disease (see page 54). Many women with endocervical or urethral infection also remain asymptomatic or have only mild symptoms.

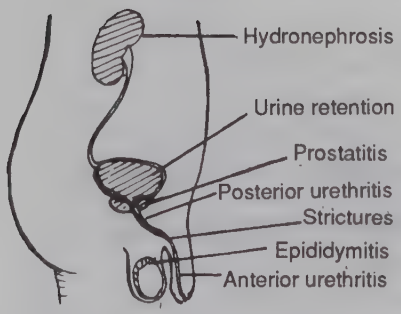


Figure 3.3 (a) **Clinical picture of gonorrhoea in males**

Males

The earliest symptom is irritation at the urinary meatus. A burning sensation when passing urine and a purulent yellow and profuse discharge soon follow. Dysuria may be slight or very severe (especially when the infection has reached the posterior urethra). Severe dysuria is often accompanied by urgency and frequency, and occasionally terminal haematuria (blood in the urine). In untreated cases, the discharge which was purulent and profuse gradually becomes scanty and less purulent and may be absent after 1–3 months, or may be present only in the mornings. Some cases may recover spontaneously, but most develop one or more complications. Local complications include infection of para-urethral glands, resulting in peri-urethral abscess and urethral stricture with associated urinary

retention. Extension of infection to the posterior urethra may lead to infection of the Cowper's glands, the prostate, seminal vesicles, vas deferens and epididymis. Failure to treat these complications adequately results in chronic infections which are often difficult to eradicate. Infection of the vas deferens may result in male infertility.

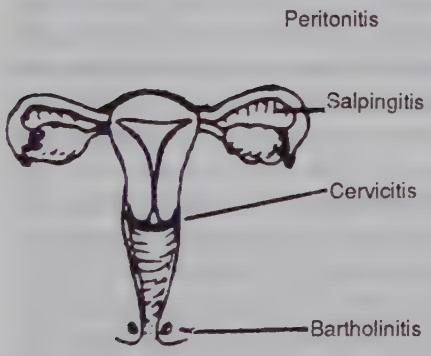


Figure 3.3 (b) **Clinical picture of gonorrhoea in females**

In acute pelvic inflammatory disease: look for gc and consider Chlamydia also

Females

About 50% of infected women have no symptoms. The female urethra is short and the urethritis and discharge often go unnoticed. Cervicitis may cause a discharge which may be dismissed as normal. As a result of these signs, many women are only seen in clinics when they have developed symptoms due to complications.

When these symptoms occur, they may be due to the primary infection, after the same incubation period as in the case of men, or due to secondary infection and complications.

Primary infection of the urethra causes a burning sensation during micturition. Frequency and urgency indicate bladder infection. Infection of Bartholin's ducts is common and may present with acute pain on one side of the vulva, but often it may swell gradually without any pain.

In untreated cases, the infection in the majority of women remains latent for several months or years, during which time they form an important reservoir for the spread of the disease.

Some women develop local and systemic complications. In a small proportion of cases, the infection may resolve spontaneously.

Local complications. These are common, especially in women with asymptomatic primary infection who have usually been harbouring the gonococci for a long time without treatment.

Spread of infection to the para-urethral glands may lead to abscesses or cyst formation. Peri-urethral abscesses may also form as a result of invasion of peri-urethral tissues posteriorly. Urethral stricture is a rare complication in females.

Infection of Bartholin's glands may lead to abscess formation if acute, or cyst formation if chronic.

Pelvic infection. Salpingitis develops in 15–30% of women with urogenital gonorrhoea. The gonococcus can be isolated from the endocervix in only about 50% of women with salpingitis. Isolation of gonococci from the pouch of Douglas and tubes is a less common occurrence. This is mainly because of secondary invasion by other organisms such as anaerobes and *Chlamydia trachomatis*, which are important in pelvic inflammatory disease. The isolation of gonococci in chronic salpingitis with associated infertility, is much rarer. Partial or complete tubal blockage is a common consequence of untreated or inadequately treated gonorrhoea in women in many African countries. As a result, there is increased incidence of ectopic pregnancies and infertility in many parts of Africa.

Disseminated gonococcal infection. This develops in 1–3% of patients with mucosal infection. Dissemination rates appear to be higher in patients with asymptomatic primary infections, partly because of the long period during which the organism may gain access to the bloodstream. In women, this may occur in association with menstruation, pregnancy or delivery.

Septicaemic spread involves a variety of distant organs, resulting in arthritis, endocarditis or meningitis.

Infants

Gonococcal ophthalmia neonatorum is characterized by a short incubation period (3 days or less) and mucopurulent discharge from one or both eyes.

Diagnosis

The diagnosis is confirmed by examination of a urethral smear or deposit of urine after centrifugation (man and woman) or endocervical smear — cervical smear (woman). Gram's stain will show Gram-negative diplococci lying inside pus cells (intracellular). Some may escape and lie outside cells, but the diagnosis should not be made unless intracellular diplococci are definitely seen. If there are facilities for culture, this should be done to confirm the diagnosis.

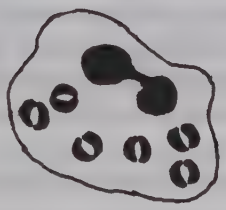


Figure 3.4 Microscopic appearance showing intracellular diplococci

5. Management of the individual patient

The value of penicillin in the treatment of gonorrhoea has been greatly reduced by the emergence of penicillinase-producing *Neisseria gonorrhoeae* (PPNG). The penicillinase thus produced renders penicillin ineffective for the treatment of such gonorrhoea. Since its appearance in 1976 in West Africa, PPNG has spread to many other African countries, thus giving an unacceptably high failure rate (75%) with penicillin treatment. Penicillin, therefore, cannot continue to be used as a first line of treatment in these cases. The second line of drugs such as spectinomycin and cephalosporins are, unfortunately, very expensive and beyond the budgets of many African countries, compounding the problem of control of gonorrhoea in their populations even further.

Uncomplicated urogenital infection

The WHO-recommended treatment regimen for *N. gonorrhoeae* is as follows:

Group A drugs

(a) Benzyl penicillin 5 mega units (3 g) intramuscularly, with 1g of probenecid* orally, or

*Probenecid blocks the excretion of penicillin in the kidneys. This will result in a longer-lasting high level of penicillin in the blood. It is the high level of penicillin which is the essential part of the treatment of gc.

(b) Procaine benzyl penicillin 4.8 mega units (4.8 g) intramuscularly, with 1 g of probenecid orally, or
(c) Ampicillin 3.5 g with 1 g of probenecid orally, or
(d) Amoxycillin 3 g with 1 g of probenecid orally, or
(e) Tetracycline hydrochloride 500 mg four times a day orally for 7 days, or
(f) Doxycycline hydrochloride 100 mg twice a day orally for 7 days.

These first-line drugs are only useful in areas where gonorrhoea resistance remains low. In many African countries, resistance to these drugs is now high and therefore their use, especially the penicillins, should be minimal.

Note
Treatment of gc with penicillin can mask concurrent syphilis (see p. 65).

Group B drugs

This second line of drugs is useful in areas with high gonococcal drug resistance to the first line of drugs:

(a) Spectinomycin (Togamycin) 2 g intramuscularly, or
(b) Cefoxitin 2 g intramuscularly, with 1 g of probenecid orally, or
(c) Cefotaxine 1 g intramuscularly, or
(d) Ceftriaxone 250 mg intramuscularly.

These drugs are expensive and are unlikely to be available in health centres and dispensaries.

Group C drugs

These drugs are active against many resistant gonococci and are less costly and more widely available than group B drugs.

Unfortunately, they are not recommended for pregnant women.

(a) Kanamycin 2 g intramuscularly, or
(b) Thiamphenical 2.5 g orally, or
(c) Cotrimoxazole (Septrin or Bactrim) four tablets twice daily for 2 days.

Recently a number of new drugs have become available which, in combination with group A drugs, have been shown to give good cure rates. These drugs inhibit penicillinase production by the gonococcus. The regimens include:

(a) Amoxycillin 3 g with clavulanic acid 250 mg orally, or
(b) PPF 4.8 mega units intramuscularly, plus 1 g of probenecid, plus amoxycillin 250 mg and clavulanic acid 125 mg orally, or
(c) Amoxycillin 3 g, clavulanic acid 250 mg, plus probenecid 1 g orally, or
(d) PPF 4.8 mega units intramuscularly, plus probenecid 1 g orally and sulbactam 500 mg intramuscularly.

These combinations may be worth trying in African countries.

There are five important causes of treatment failure in gonorrhoea:

- Wrong diagnosis: non-gonococcal urethritis, chemical urethritis, venereophobia;
- Drug resistance;
- Too low dose of antibiotic;
- No treatment given to sexual partner, resulting in reinfection;
- Post-gonococcal urethritis (see page 59).

Single dose treatment in an uncomplicated case

Ophthalmia neonatorum

Specific treatment: Crystalline penicillin by injection plus 1% tetracycline eye ointment, or chloramphenicol eye ointment.

6. Prevention and control

Gonorrhoea in adults — see Introduction to this chapter.

Ophthalmia neonatorum can be completely prevented. This can be done by the use of one drop of 1% silver nitrate in each eye of every newborn. The problem with silver nitrate solution is that it has to be freshly prepared every week. If fresh silver nitrate is not available, the eyes of the newborn can be washed out with normal water; this will remove the gonococci because they usually remain on the surface of the mucous membrane for some hours before they invade the tissues.

Eye wash for every newborn

7. Action

- Do not treat urethritis routinely as gc; confirm diagnosis with smear; non-gonococcal urethritis may be more frequent than frank gonorrhoea in some cases.
- Check all gc cases for concurrent syphilis after eight weeks.
- Treat all cases of salpingitis and PID as a combination of gonococcal and non-gonococcal salpingitis. They are clinically indistinguishable. Do not use single dose therapy because of the high failure rate. The following is recommended:
  Ampicillin 3 g orally after probenecid 1 g orally, then 500 mg four times a day for 1 week.
  Alternatively, give:
  Tetracycline 500 g four times a day for 7–10 days. Add Flagyl 400 mg three times a day for 7 days to eliminate anaerobic organisms. Admit or refer severe cases.
- Always try your best to treat the partner or partners.
- Do not miss an opportunity to give health education. You may prevent the individual from picking up AIDS.

8. Summary

Gonorrhoea is an infection of the urogenital tract transmitted through sexual intercourse.

Uncomplicated gc sensitive to penicillin can be treated with a single dose of penicillin. However, many strains of gonococci (PPNG) now produce penicillinase which inactivates penicillin so they are resistant to this treatment and require other drugs such as spectinomycin. Prevention is by health education and early diagnosis and treatment of patients and contacts.

Gonococcal ophthalmia neonatorum is an eye infection of newborns, easily prevented by routine eye washing at birth.

NON-GONOCOCCAL (NON-SPECIFIC) URETHRITIS

1. Non-gonococcal urethritis (inflammation of the urethra) was commonly known in the past as "non-specific urethritis" (NSU) because the cause had not been established. However, specific organisms have now been identified as causing many of these cases. *Chlamydia trachomatis* is responsible for at least 50% of infections.

Other organisms responsible are mycoplasma, anaerobic bacteria and various viruses, e.g., herpes simplex. Mechanical or chemical irritation may cause urethral pain and mimic infectious urethritis.

2. Occurrence and importance

NGU is at least as common as gonorrhoea, but unfortunately urethral discharge is generally automatically diagnosed as being gonorrhoeal and treated with penicillin, which has little or no effect on chlamydiae. This may lead to unjustified doubts as to the value of penicillin in gonorrhoea, and claims of penicillin resistance.

3. Epidemiology

Post-gonococcal urethritis

Patients successfully treated for gonorrhoea may have a slight continuing urethral discharge. If no gonococci are found in this discharge, it is probably because there was a concomitant chlamydial infection which is not sensitive to penicillin and requires a course of tetracycline. Laboratories with experienced technicians and a good microscope are sometimes able to confirm chlamydial infection. Field's stain shows blue-staining intracellular "inclusion bodies" lying inside monocytes or polymorphs.

Fear of having caught an STD (venereophobia) may lead to excessive use of antiseptics. Also, squeezing of the penis may result in a slight discharge, apparently justifying the patient's fear.

4. Clinical picture

The discharge of NGU is often thinner and whiter than typical gonorrhoeal discharge, but it may look just like typical thick yellow gc pus. On the other hand, gonorrhoea may sometimes present with a thin watery discharge. There is no way of being certain which is which without microscopy. A careful history may help the diagnosis (history of catheterization, foreign bodies introduced into the urethra, excessive masturbation, use of antiseptics to prevent STD, recently treated gonorrhoea). If there is no suggestion of chemical or mechanical urethritis, and no gonococci are seen microscopically, the urethritis is most likely to be chlamydial.

5. Management of the individual patient

Chlamydiae are sensitive to tetracycline. The dose is 250 mg four times a day for 7 days. Remember to treat the partner whenever possible. Chemical or mechanical irritation will improve when further trauma of the epithelium is avoided. Reassurance and rest (that is, abstinence from sexual intercourse) are all that are needed.

6. Prevention and control

As for other STD.

7. Action

Always order a wet smear (for trichomonads) and a Gram stain in all cases of urethritis; treat according to findings.

8. Summary

NGU is inflammation of the urethra. It is usually due to chlamydiae. Sometimes, it is due to other organisms and non-specific irritants.

TRICHOMONIASIS

1. Trichomoniasis is a protozoal infection of the genital tract of males and females.

2. Occurrence and importance

Trichomoniasis is a very common infection of the female genital tract. It affects 10% of all women. In males, it may cause a urethritis, often misdiagnosed as gc and consequently wrongly treated with antibiotics.

3. Epidemiology

Transmission is by sexual intercourse or by indirect contact through contaminated clothes and other articles. The parasite likes an acid environment (pH 4) as found in the vagina of adult women during the reproductive period.

4. Clinical picture

Trichomonas infection in women is usually symptomless, but it may cause itchy vaginitis and increased vaginal discharge. The discharge is greenish and foamy. In most males the infection is completely asymptomatic.

Diagnosis

The diagnosis is easily confirmed once one thinks of it. A fresh drop of vaginal or urethral discharge or a high vaginal swab are examined directly for moving protozoa. It is essential to do the examination with a fresh specimen; therefore the specimen must be taken personally to the laboratory technician and the reason for immediate examination explained.



Figure 3.5 **Microscopic appearance of *Trichomonas vaginalis***

If a patient complains of urethral discharge it is best to make a Gram stain to exclude gc and at the same time examine a fresh specimen for trichomonas.

5. Management of the individual patient

The treatment of choice is single dose metronidazole (Flagyl) 2 g (10 tablets of 200 mg or 5 tablets of 400 mg) swallowed under supervision, or 200 mg three times a day for 5 days. It is important to treat both partners even if one partner is asymptomatic.

Disadvantages

Metronidazole is incompatible with alcohol and must not be given to women in early pregnancy. Women with trichomonas vaginitis in pregnancy may be treated topically with Tinidazole pessaries to be inserted in the vagina. A disadvantage of pessaries is that they are expensive and often difficult to obtain.

6. Prevention and control

- As for other STD — see Introduction page 45.
- Improvement of general hygiene.

7. Action

Always order a wet smear together with a Gram stain in all cases of urethritis; treat according to findings.

8. Summary

Trichomoniasis is a common infection of the genital tract in both males and females. It may cause urethritis in males. Treatment when indicated should be cheap and simple and given to both partners. Antibiotics are of no use.

SYPHILIS

1. Syphilis is a disease characterized by a primary lesion, a later secondary eruption on the skin and mucous membranes, then a long period of latency, and finally late lesions of skin, bones, viscera, CNS and cardiovascular systems.

2. Occurrence and importance

Yaws and syphilis are closely related. In a community with yaws there will be no syphilis, but while yaws has been practically eradicated in most of Africa, the incidence of syphilis is increasing. Syphilis is said to occur more often in urban societies, although a high incidence is also found in some rural communities.

Syphilis is a very slowly progressing disease, but it is disabling and may be fatal after 10–20 years.

The same groups are at risk as for other STDs (see Introduction, p. 44), but medical and laboratory workers have to be included as they can, rarely, acquire the infection accidentally. The precautions they must take are the same as those to prevent AIDS.

3. Epidemiology

Syphilis is caused by a spirochaete known as *Treponema pallidum*. The spirochaete enters the body through the mucous membranes and skin. Outside the body the spirochaetes die rapidly.

Conditions for transmission are optimal during sexual intercourse, but skin-to-skin contact can be enough for transmission if there are lesions.

Spirochaetes invade the bloodstream after infection and so blood may be infective.

Routes

- Usual transmission — sexual intercourse.
- Congenital infection may occur before birth, if the mother is infected.
- Unusual transmission — accidental, by touching infective tissues (dentists, midwives, and other medical personnel).
- Via blood transfusion.

4. Clinical picture

The incubation period varies from 10 days to 10 weeks, with an average of 3 weeks. Patients usually present with a genital sore, the hard chancre. This is part of the primary stage of the disease.

Primary syphilis

This consists of the hard chancre, the primary lesion of syphilis, together with enlargement of regional lymph nodes.

The hard chancre is a single, usually painless, ulcer on the genitalia or elsewhere (lips, tongue, breasts); even when not treated it will heal spontaneously in a few weeks. The lymph glands are bilaterally enlarged and not painful. There will usually be no suppuration (see also comparison between soft and hard chancre, p. 68). Syphilis and chancroid can occur together. If secondarily infected, the chancre and regional lymph glands may be painful.

Secondary syphilis

In most patients (but not all), 4–6 weeks after the primary infection a generalized secondary skin eruption appears, often accompanied by

mild constitutional symptoms. These early rashes tend to be symmetrical, transient, and do not itch. These lesions are very infective and many spirochaetes can be demonstrated in them.

The manifestations of early secondary syphilis disappear spontaneously within a few months. Asymmetrical moist, flat, round, warty lesions appear after some time in the moist areas of the body. The commonest sites are the anus, vulva, perineum, mouth and axilla, and the areas between the buttocks and underneath the breasts. These highly infectious lesions are known as condylomata lata, and should be differentiated from condylomata acuminata which are of viral origin (see below). Other manifestations of late secondary syphilis include generalized lymphadenopathy, patchy loss of scalp hair (alopecia areata) and snail-track ulcers of the tongue and oral cavity.

These lesions also tend to disappear spontaneously after some time, even without treatment. The patient then enters the stage of latent syphilis during which he is no longer infectious and shows no symptoms at all. After a variable period ranging from 2 to 20 years, the manifestations of tertiary syphilis set in.

Tertiary syphilis

This stage is characterized by destructive, non-infectious lesions of the skin, bones, viscera, and mucosal surfaces.

Other disabling manifestations occur in the cardiovascular system (aortic incompetence, aneurysms), or central nervous system (dementia paralytica, tabes dorsalis).

Syphilis in pregnant females

A pregnant woman who has syphilis may infect her unborn baby. This is more common in the primary and secondary stages of the disease. Infectivity usually diminishes after 5 years of the disease.

Depending on the severity, congenital syphilis can result in congenital abnormalities (early or late manifestations), stillbirth, or repeated abortions.

Notes

- Primary syphilis in females is often missed because the chancre is symptomless and not easily seen.

- No primary genital lesion will be present when infection is acquired through infected blood (congenital, blood transfusion).
- Gonorrhoea and syphilis are sometimes acquired at the same time. When the gc is treated promptly with PPF, the first stage of syphilis may be suppressed. The patient will feel cured and safe but his syphilis will progress into the second and third stages. Therefore, it is wise to screen cases of gc routinely for concurrent syphilis. An RPR test or VDRL should be done 8 weeks after infection.

After gc watch for syphilis

Diagnosis

- Serological tests are based on antibodies formed. These will be positive only 6–8 weeks after infection. Most hospital and some health centre laboratories are able to perform Kahn, VDRL or RPR tests, which are non-specific but useful for screening.
- The spirochaete causing syphilis is difficult to stain. It can be demonstrated in smears by using a special illumination technique: dark-ground illumination. Smears from the primary lesion (hard chancre) or from skin lesions in the early secondary stage will show the spirochaetes. As the technique requires special skills only a few laboratories will perform this examination. Dark-field examinations are easy if the laboratory has a microscope with inbuilt power source and a dark-field stop. The technique can be learned.
- Jarisch-Herxheimer reaction: Occurs after penicillin injection. Due to a large number of killed spirochaetes, a patient with syphilis may react with fever. This may be considered as confirming the clinical diagnosis.
- Other highly specific and sensitive tests such as the TPHA test are used to confirm the diagnosis after screening tests such as VDRL.

5. Management of the individual patient

During treatment the patient should, of course, abstain from sexual intercourse.

The drug of choice is procaine penicillin (watery suspension) 0.8 mega units daily for 10 days. An alternative is benzathine penicillin 2.4 mega units (1.2 m in each buttock) as a single dose.

Syphilis is cured by low blood levels of penicillin, but only if the penicillin is in the blood for a period of at least 10 days. Patients allergic to penicillin should be given tetracycline 500 mg four times a day orally for 15 days.

Erythromycin can be used in pregnancy if there is allergy to penicillin. For syphilis of longer than one year's duration, use benzathine penicillin G 2.4 mega units intramuscularly weekly for three successive weeks. Procaine penicillin 0.8 mega units daily for 3 weeks should be given if there is any evidence of central nervous system involvement.

It is vital to trace contacts and treat them as well.

Penicillin for:
Syphilis: low level, long time
Gonorrhoea: high level, short time

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6. Prevention and control

In general the same methods apply as in the control of gonorrhoea. Condoms give better protection against gonorrhoea than against syphilis because the spirochaetes can penetrate the intact skin.

Medical workers and midwives should be careful in examining suspect lesions and should wear gloves when doing vaginal examinations.

If there are many cases of syphilis in your area, it is wise to screen all pregnant women routinely in order to prevent congenital syphilis. Arrange for this with the nearest laboratory which is able to perform the VDRL or Kahn test, but remember that these tests are non-specific and may give false positive results. If uncertain, refer the patient to a bigger laboratory.

7. Action

- Check all gc cases for concurrent syphilis.
- Think of syphilis in all poorly understood skin diseases, heart disease, psychiatric and neurological disorders.

8. Summary

Syphilis is a long-lasting disease. It is transmitted by sexual contact. Early diagnosis and treatment of cases and contacts are essential for control.

CHANCROID

1. Chancroid is an acute venereal infection which typically presents as a ragged, painful, necrotizing ulcer on the genitalia, frequently accompanied by inflammatory swelling and suppuration of the regional lymph nodes.

Synonym: Soft sore.

2. Occurrence and importance

Most common in tropical countries, especially in urban areas. Important in eastern Africa, and incidence is increasing.

For high-risk factors, see Introduction on page 45.

3. Epidemiology

Chancroid is caused by Gram-negative bacilli shaped like small rods and which form chains, *Haemophilus ducreyi*. These bacteria can penetrate the genitals very easily. Women may harbour the organisms in the vagina without symptoms. They form the reservoir of infections. Transmission is by sexual intercourse. Infected males usually do not pass the infection further because of the painful ulcer.

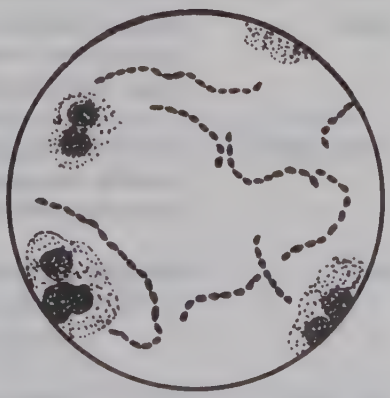

Figure 3.6 **Chancroid: microscopic appearance of *H. ducreyi***

4. Clinical picture

Three to five days after a sexual contact an ulcer develops on the skin of the penis. The ulcer can enlarge in every direction. Often other parts of the skin are infected from the first ulcer and new ulcers develop. The ulcers are painful, and soft on palpation. There may be a suppurative painful regional lymphadenitis. In most cases only one side is affected. Males might present with urethritis and females might have a vaginal discharge.

For differences between the soft chancre (chancroid) and the hard chancre of syphilis, see Table 3.2.

Diagnosis

Laboratory diagnosis is important as clinical diagnosis is unreliable. Typical Gram-negative rods can be seen from smears taken from the base of the ulcer or material aspirated from enlarged inguinal lymph glands. The slides, however, need to be prepared carefully and an experienced person should do the microscopy.

Table 3.2 **Comparison of the clinical features of chancroid and syphilis**

	Chancroid	Syphilis
Ulcer	Single or multiple, painful, soft, irregular edge, pus and oedema	Usually single, painless, hard, well-defined edge, clear discharge
Lymph glands	One-sided or bilateral, painful enlargement, often suppurative	Bilateral, painless enlargement, never suppuration
Lab.	*Haemophilus ducreyi* in smear (Gram stain)	Spirochaetes seen on dark-field illumination VDRL positive 6–8 weeks after infection
Incubation time	3–5 days	10–70 days or longer

5. Management of the individual patient

Sulpha was the drug of choice, e.g., sulphadimidine 2 g at once, followed by two tablets (1 g) four times a day for 1 week. Now an alternative treatment is cotrimoxazole two tablets twice a day for 7 days. Do not incise lymph glands, even when there is fluctuation; they will heal completely with antibiotics only. All patients must be advised on their future sexual behaviour as they might contract AIDS. (Many cases seen may have HIV infection as genital ulcers probably make entry for the virus easy.)

Note
Because large ulcers may be double infections with both chancroid and syphilis, many health units prefer to treat both diseases by giving benzathine penicillin 2.4 million units for syphilis as well as cotrimoxazole for chancroid.

6. Prevention and control

Condoms might not always prevent chancroid. When condoms are used, an ulcer could develop at the base of the penis which is not covered by the condom: "condom chancre". Thorough washing of genitalia with soap and water promptly after intercourse is probably helpful in prevention.

7. Action

See Introduction to this chapter.

8. Summary

Chancroid is an acute venereal infection characterized by painful, soft, destructive, growing, multiple ulcers.

Treatment: sulpha or cotrimoxazole.

Prevention is as for other STD.

LYMPHOGRANULOMA VENEREUM

1. Lymphogranuloma venereum is caused by certain serotypes of *Chlamydia trachomatis* which are more invasive than the ones causing trachoma.

2. Occurrence and importance

This disease occurs throughout tropical Africa and is more common in towns and seaports and amongst promiscuous people.

3. Epidemiology

The organism enters through abraded skin of the genitals and spreads to the lymph glands. There is a small local lesion which does not last for more than a few days and which is easily missed. There is an incubation period of 4–20 days.

4. Clinical picture

After the initial ulcer or vesicle (which may be missed) the next signs and symptoms are swelling of the inguinal lymph glands in males. In females, the spread is to internal glands and may be followed by rectal stricture. Following the damage to the lymphatics and lymph glands there may be swelling and oedema of the external genitals.

5. Management of the individual patient

Tetracycline is the drug of choice in doses of 500 mg four times daily for 2 weeks. In pregnancy, erythromycin in similar dosage should be used.

Abscesses in the glands should not be incised. If in doubt about the diagnosis refer to hospital.

6. Prevention and control

As for other STD.

7. Action

Exclude this disease in patients with swelling of the inguinal lymph glands, especially in men, if they are enlarged both above and below the fold of the groin. Arrange for contact tracing. This condition must be considered in the differential diagnosis of genital ulcers and lymph-node enlargement.

OTHER GENITAL SORES OR NEW GROWTHS

Many skin diseases can be localized in the skin or mucous membranes of the genital organs. Some infectious skin diseases are transmitted during sexual intercourse. Table 3.3 below summarizes the most common ones.

Herpes genitalis

This is caused by herpes simplex virus type 2 (HSV-2), and is usually sexually transmitted. After an incubation period of 3–7 days, the early lesions appear as an itchy rash, which quickly develop into crops of painful vesicles on the external genitalia or cervix (where they are often symptomless). The vesicles on the external genitalia break down to form shallow, often confluent superficial ulcers. These ulcers are usually painful with the first attack, but become less painful with subsequent attacks. Recurrences tend to be less severe and self-limiting and may be triggered off by worries, anxiety, pregnancy, menstruation, depression, fevers or trauma.

Secondary bacterial infection of the ulcers can occur as a complication. At one time the virus was thought to be closely associated with cancer of the cervix in women, but this association is no longer strong.

Genital herpes should be differentiated from other causes of genital ulceration such as syphilis, chancroid, lymphogranuloma venereum and granuloma inguinale.

Table 3.3 **Other causes of genital sores**

Pathogenic organism	Disease	Main sign/symptom
Viral	Herpes simplex	Blisters
	Venereal warts	Tumours
	Molluscum contagiosum	Papules
Fungal	Candidiasis	Redness and itching
Bacterial	Balanitis	Redness and irritation
Parasitic	Scabies	Papules and tunnels, itching
	Pediculosis pubis	Itching, pyoderma

Treatment is symptomatic and consists of analgesics and sedatives for first attacks; prevention of secondary bacterial infection with frequent saline wash and application of gentian violet (GV). Recently a drug known as acyclovir (Zovirax) has been found to be useful, especially in severe herpes as seen in AIDS patients. Both systemic and topical applications exist, but this drug is very expensive and so may only be available in some big cities and hospitals.

Patients with active lesions are advised against sexual intercourse. Pregnant women with herpes should be delivered in hospital by Caesarean section, to avoid infecting the baby during vaginal delivery.

Venereal warts (Condylomata acuminata)

Venereal warts are caused by infection with human papilloma virus (HPV), and are usually transmitted during sexual intercourse. The virus grows best in warm, moist areas of the body, such as the female genitalia, the uncircumcised penis, and the anal region.

Similarly, genital discharge, of whatever origin, in either sex also encourages its growth and spread. Recently, the virus has assumed significance because it is now thought to be very closely associated with cancer of the cervix in women.

After an incubation period of 1–6 months, the warts appear as fleshy irregular-shaped growths on the coronal sulcus, glans penis, external urinary meatus and prepuce in males, and on the labia and vagina with extension to the cervix and anal regions in females.

Treatment depends on the extent and distribution of the lesions. Medical treatment consists of local application of corrosives such as podophyllin and trichloracetic acid (TCA), and 5-fluorouracil cream. Podophyllin 25% solution in spirit or tincture of benzoin usually gives good results in smaller lesions not situated on the cervix or inside the male urethra. The drug is contraindicated during pregnancy. When first applied, podophyllin is left for 4 hours, and thereafter it is left for longer periods (up to 12 hours) and then washed off. Take care to cover the surrounding areas with protective ointment (e.g., Vaseline) to prevent corrosion of normal tissues. The patient is treated twice a week for 1 month. If there is no improvement, or if the warts are large, or situated in tricky areas, refer the patient to hospital. In hospital, surgical excision, electrocautery and cryosurgery may be used to treat large warts. Recurrences are common. Always differentiate condylomata acuminata from condylomata lata, which is a

manifestation of secondary syphilis. Do VDRL to exclude syphilis and treat accordingly.

Molluscum contagiosum

Shows as a round, smooth, papule. The papule has a central depression (umbilicated).

Treatment is local with careful application of pure phenol or trichloracetic acid.

Balanitis

Balanitis is an inflammation of the glans penis. It may be caused by candida or bacteria. Lack of personal hygiene is the main promoting factor. Phimosis may be the reason that the glans penis cannot be cleaned. In candida infection, diabetes mellitus must be excluded. Candida infection may be acquired from a woman with a vulvo-vaginitis caused by candida (see also Chapter 2 on Dermatomycosis).

Therapy: Improved personal hygiene, health education, eventually circumcision. Antibiotics are contraindicated.

AIDS

1. The Acquired Immune Deficiency Syndrome is a systemic viral disease with a latency period of often many years. It culminates in a state of immune deficiency of the body which results in death from unusual infections and tumours.

Synonyms: There are different names in various parts of Africa, e.g., Slim in Uganda, Ukimwi in Tanzania, SIDA in Francophone Africa and VIGS in South Africa.

2. Occurrence and importance

AIDS is an important new disease that appears to be spreading steadily in the general population in Africa and throughout the world. It is more common in urban communities, and in the sexually active age groups. The same groups are at risk as for other STD, including medical and laboratory workers who may acquire the infection accidentally. In addition, there is a new group of patients who are

acquiring the disease: the newborn. The presence of congenitally acquired infection indicates a high level of disease in the community.

AIDS is a more difficult disease to control than the other STDs as the infected person can transmit the virus to others during a long latent period which can be without symptoms.

There is also no immediate evidence of infection as there is no genital lesion on acquiring the infection through sexual intercourse.

3. Epidemiology

AIDS is caused by a retrovirus (Human Immunodeficiency Virus — HIV). The virus is present in the body fluids of an infected person, in highest concentration in blood, semen and vaginal secretions.

The virus can only be transmitted through contact of infected fluids with the blood stream of another person through minute lacerations of the skin or mucous membranes. Conditions for transmission are optimal during sexual intercourse.

However, the virus can also be transmitted through infective blood transfusions and sharing of needles, syringes and surgical instruments, and via the placenta to the foetus.

Routes of transmission

- Usual transmission — sexual intercourse.
- Unusual transmission — accidental contact with infective body fluids on cuts/sores, etc. (doctors, midwives, dentists, laboratory personnel).
- Via blood transfusion.
- Via sharing of needles, syringes, surgical instruments, blades, etc.
- Congenital infection of the foetus or newborn may occur before or during birth if the mother is infected.

4. Clinical picture

The infection with the causal virus is shown by the development of antibodies which are demonstrated by special tests now available in most big hospitals. Unfortunately, the antibodies are not protective and the virus remains in the individual who becomes a healthy carrier with the potential to infect others for a long period.

The incubation period from contact to the appearance of antibodies is usually 3 weeks to 3 months, although a period of greater than 1 year

has been reported. Shorter incubation periods occur after transfusion of infected blood. At this time, the patient may develop an acute illness with fever, headache, cough and lymphadenopathy, which resolves spontaneously. Patients may present with an acute mental illness (dementia) which also resolves.

Patients then remain asymptomatic for months or many years, although antibodies can be demonstrated in the blood. Ultimately, most patients develop the disease AIDS. Babies who acquire infection from their mothers usually develop the disease within a year.

The symptoms and signs of AIDS may develop gradually until the full-blown picture of the disease is seen. This consists of general symptoms and signs of infections (e.g., fever, swelling of lymph glands, night sweats, headache and cough), and presence of unusual (opportunistic) infections and tumours, such as Kaposi's sarcoma.

Some of the opportunistic infections which occur as the immune deficiency appears are herpes zoster, oral and oesophageal thrush (Candida), tuberculosis and rare types of pneumonia and bowel infection.

These infections may be accompanied by weight loss (more than 10%), chronic diarrhoea and cough, prolonged fever (which may be intermittent), unusual rashes, and generalized lymphadenopathy. Some patients develop progressive deterioration of mental function which includes psychiatric disturbances, tremors and slowness, leading to paralysis. Some of the symptoms and signs of AIDS are identical to those of other wasting diseases, e.g., tuberculosis, malnutrition and cancer. It is vital not to miss these treatable conditions.

Tuberculosis is one of the infections which progresses faster when there is lowered immunity.

AIDS in pregnant women

Pregnancy may accelerate the disease, or may cause a healthy seropositive mother to develop AIDS.

Diagnosis

- Serological tests to detect antibodies are usually positive within 3 months of infection, but may be negative for up to 1 year. These tests require sophisticated equipment and are not available in all laboratories. New, more simple, tests are being developed.
- There is no other simple way to detect the virus.

- Clinical diagnosis is not always accurate. Suspected patients should be confirmed with a serological test.
- The diagnosis has to be kept confidential.

5. Management of the individual patient

There is no cure for the AIDS virus. Patients should be treated for their symptoms and for opportunistic infections.

Treatment includes:

- Gentian violet or mycostatin for oral candidiasis;
- Cotrimoxazole for acute respiratory infection;
- Full treatment of tuberculosis;
- Symptomatic treatment of pain with paracetamol.

6. Prevention and control

The most important preventive measure is health education. No one should die of ignorance.

The most important method of control applies to all STDs: reduction of sexual activity outside one faithful partnership. Condoms give some, but incomplete, protection against the virus. Latex rubber condoms should be used. Contraceptive foams and jellies may offer some protection against the virus. The presence of other STDs, and especially genital ulcers, encourages virus transmission.

Diagnose and treat STDs promptly and correctly

Medical workers and midwives must be careful when handling body fluids. They should always wear gloves during procedures in which they will be in contact with body fluids, and especially cover wounds or abrasions.

Medical workers must learn and perform correct sterilization procedures for needles, syringes, blades and other surgical instruments (see Table 3.4, page 78).

Blood for transfusion must be screened routinely for HIV infection and discarded if positive. Avoid unnecessary transfusions. Give a

blood transfusion only when it is absolutely necessary to save the patient's life.

Women of childbearing age who are seropositive must be counselled before they embark on another pregnancy.

7. Action

- Any apparently healthy person may be carrying the AIDS virus. This is more likely in patients presenting with STDs. Take great care in handling genital specimens, taking blood, or giving injections to these patients.
- AIDS must be confirmed with blood tests before the diagnosis is told to the patient. Patients with AIDS require careful counselling when they are told the diagnosis. Laboratory results and medical records must be kept confidential.

8. Summary

AIDS is a fatal disease which, once the symptoms and signs develop, causes death in under 2 years. The AIDS virus is transmitted most commonly by sexual contact. There is no treatment or vaccine. Communities must be taught how to prevent AIDS by limiting sexual activity.

Table 3.4 Procedures for disinfection/sterilization

Item	First choice	Others
1. Skin		
(a) Injections	70% alcohol	Savlon
(b) Wounds	Eusol	Hibitane
(c) Prior to surgical procedures	0.25% iodine	Savlon
2. Washing hands (after working with infected material)	0.5% hypochlorite* Scrub nails with brush	Soap and water or 2% Lysol
3. Thermometers, re-usable tongue depressors, attachments for diagnostic instruments	Soak in 70% alcohol between use	Hibitane, Savlon
4. Syringes, needles, lancets	First flush with water immediately after use	
(a) Plastic disposable	To be discarded/incinerated	
(b) Autoclavable/re-usable plastic, glass	Autoclave	Boil for 20–30 min
5. Soiled linen (infected)	Soak in 0.5% hypochlorite for 30 min	Soak in 2% Lysol overnight or autoclave or boil for 20–30 min
6. Soiled plastic sheeting	Wipe with 0.5% hypochlorite	Wipe with 5% Lysol
7. Surgical instruments	Autoclave	Soak in 4% formalin, 2% glutaraldehyde or 0.5% hypochlorite for 30 min. Wash thoroughly then boil for 20–30 min

8.	Disposable infected material (cottonwool, gauze), disposable containers	Incinerate	
9.	Infected re-usable containers	Autoclave container including contents prior to disposal and washing	Soak in 4% formalin, 2% gluteraldehyde or 5% Lysol before disposal in a pit latrine
10.	Bedpans (infected)	Add an equal volume of neat Lysol or 4% formalin or 0.5% hypochlorite for 30 min prior to disposal of contents and washing	
11.	Placentae, surgical tissue	Incinerate	
12.	Laboratory glassware (contaminated)	Autoclave	Soak in 2% gluteraldehyde, 0.5% hypochlorite, 4% formalin or 5% Lysol prior to washing
13.	Wire loops, blades, points of forceps	Flame until red hot	
14.	Benches and floors (following spillage of infected material) interior of fridges, freezers, beds, etc.	Wipe with 0.5% hypochlorite	Wipe with 4% formalin or 2% gluteraldehyde

*Commercially supplied Jik is 3.5% sodium hypochlorite. Dilute 1:7 with water to obtain a 0.5% solution.

Chapter four

Vector-borne Diseases

INTRODUCTION

Definition of vector: Generally speaking, a vector is any carrier of disease, but in the case of the "vector-borne diseases" we restrict the word to those invertebrate hosts (insects and snails) which are an essential part of the life cycle of the disease organism. The disease organism actually undergoes a period of development inside the vector, and the time taken for this is called the extrinsic incubation period. A house fly, which just carries bacteria or amoebic cysts on its feet and contaminates food with them, is not regarded as a vector: this is simply mechanical transmission of disease.

Insect vectors usually acquire the disease organism by sucking blood from infected persons, and later pass it on by the same route. There are other transmission routes, however. Infection may enter skin cracks or abrasions either from infected insect faeces deposited when feeding, or from body fluid when an insect is crushed.

Table 4.1 Classification of some vector-borne diseases according to causative organisms

Viruses	Bacteria	Protozoa	Worms/flukes
Dengue	Plague	Malaria	Filarial diseases: Filariasis bancrofti Onchocerciasis
Yellow fever	Relapsing fever	Trypanosomiasis	Schistosomiasis

DISTRIBUTION

Vector-borne diseases are limited to areas where suitable conditions exist for the vectors. Most vectors have quite specific breeding, feeding, and attacking behaviour.

Endemic and epidemic vector-borne diseases

In Africa many vector-borne diseases are endemic, which means that they are present all the time in a given geographic area or population group, e.g., malaria, schistosomiasis, filariasis and trypanosomiasis. Some of the diseases are present all the time in animals in certain areas and are enzootic, e.g., yellow fever in monkeys. Many of these diseases can become epidemic, that is, there is an increase of cases in excess of what is expected because of environmental or other changes leading to increased transmission. Examples are plague, yellow fever, and typhus. Before this happens, there might have been an epizootic (an epidemic in animals) when there is an unexpected increase in occurrence of the disease in the animals concerned, e.g., in plague.

Some of the very serious epidemics in Africa have been due to yellow fever (Nigeria, Ghana, Ethiopia, Sudan), trypanosomiasis (Uganda), kala azar (Kenya, Sudan), plague (Uganda, Kenya, Tanzania), and typhus (Burundi, Rwanda, Ethiopia).

Table 4.2 **Diseases and their vectors**

Vector	Causative organism	Disease
Mosquitoes	*Anopheles funestus* *A. gambiae*	Malaria
	Culex and *Anopheles*	Filariasis bancrofti
	Aedes spp.	Yellow fever Dengue fever
Sandflies	*Phlebotomus* spp.	Visceral leishmaniasis (kala azar) Cutaneous leishmaniasis
Buffalo gnats or Black flies	*Simulium damnosum* *S. naevei*	Onchocerciasis (River blindness)
Biting flies	*Glossina palpalis* *G. morsitans*	Trypanosomiasis (sleeping sickness)
Cyclops	*Dracunculus medinensis*	Dracunculosis (guinea worm)
Lice	*Pediculus corporis*	Relapsing fever (louse-borne variety)
	P. capitis	Epidemic typhus
Fleas	*Xenopsylla cheopis*	Plague
Soft ticks	*Ornithodorus moubata*	Relapsing fever (tick-borne variety)
Hard ticks	*Rhipicephalus* spp.	African tick-borne typhus

CONTROL

All organisms causing vector-borne disease have a stage in their transmission cycle outside the human being.

This offers an opportunity of controlling the disease without interfering directly with human beings and their behaviour, but this task is not as easy as it may seem. To be successful, a thorough study of the behaviour of the vector is essential, e.g., in the case of a flying

insect, breeding places, resting places, feeding patterns, and flying distance should all be known.

Control of vector-borne disease may be brought about by:

- Reducing the host reservoir;
- Controlling the vector;
- Protecting the susceptible individual.

Table 4.3 **Control of vector-borne diseases**

Host	Vector	Susceptible individual
Reduce human reservoir by mass treatment	Vector control	Vector avoidance Screening houses Protective clothing Repellents Health education to change personal behaviour
Reduce animal reservoir		Protection from parasite Immunization Chemoprophylaxis Health education to use above properly

There are three main methods of vector control:

- Killing of adults — insecticides;
- Killing of larvae — larvicides;
- Prevention of breeding — environmental sanitation.

Insecticides

Insecticides fall into two groups: residual and non-residual. Of the non-residual insecticides, the best known is pyrethrum which is the base of ordinary sprays. Pyrethrum is a natural insecticidal mixture, derived from pyrethrum flowers. The dried powdered flower petals can be used as dusting powder. An extract of the powder is often used in solution in kerosene as a spray. The insecticidal components of pyrethrum are unstable in light and air and therefore have no residual effect. Pyrethrum is still the quickest-acting insecticide known and is of considerable use in the immediate handling of a biting nuisance.

When pyrethrum is sprayed in a room which is then kept closed for 10 minutes, it will kill most insects present at the time of spraying. But when the room is opened again new insects entering will be unaffected and further spraying will be necessary. In most tropical houses it is impossible to keep a room closed. Efficient screening and daily use of pyrethrum are recommended.

Larger insects like cockroaches tend to recover from the almost immediate knockdown by pyrethrum. Therefore a residual insecticide is added to most products for domestic use. Pyrethrum is also incorporated in slow-burning insect coils. These coils produce an insecticidal smoke which is both toxic and repellent to insects. Such coils burn for about 9 hours and are widely used.

Residual insecticides are stable organic chemicals which, when applied to a surface, remain toxic for some time, usually several months. Particles of these insecticides are picked up by the feet of the insects when they rest or walk on the surface. The particles dissolve in the waxy outer layer of the insect and then penetrate into it. These insecticides affect the nervous system of the insect causing paralysis and death. Their action is slow: often contact of several minutes is necessary and death may not occur for several hours. Thus a room treated with residual insecticides may not show any dead insects because the affected insects may leave the room and die elsewhere.

Two main types of residual insecticides are used:

Chlorinated hydrocarbons:	DDT (Dicophane)
	Dieldrin
	Gammexane (gamma-benzene hexachloride BHC, Lindane, HCH)
Organophosphorus compounds:	Diazinon
	Malathion
	Parathion
	Fenitrothion

Chlorinated hydrocarbons, particularly DDT, are irritating to insects so they may leave the treated surface before they have picked up a lethal dose. Most of the organic phosphates are highly toxic for man and therefore cannot be used in house spraying. The newer ones, such as malathion and diazinon, are less toxic, but also less effective, especially when used on mud surfaces.

DDT is not biodegradable, i.e. it is not broken down and destroyed in nature. Hence it can contaminate the environment, be concentrated in the food chain, and become a health hazard to man, animals, and the ecosystem. For this reason, its use has been banned in many countries.

Larvicides

The residual insecticides have little residual effect when used as larvicides in water. This may be due to dilution, absorption by mud and vegetation, or the insecticide being washed away. Therefore, the application of these insecticides has to be repeated regularly.

Oil works by spreading over the water surface as a continuous film and preventing the larvae from "breathing". Therefore, it can be used only in open water. When there is vegetation, emulsions or pellets must be used. Stomach poisons such as Paris green are effective only against larvae which feed on the water surface — this includes all mosquito larvae. Temephos (Abate) granules can be applied on water once monthly and are relatively non-toxic to man, animals and fish. The ability of an oil to spread is enhanced by dilution with a thinner oil such as vegetable oils or kerosene.

Environmental sanitation

Prevention of breeding of mosquitoes is an effective and cheap form of mosquito control. It also has the advantage that most measures that can be used to prevent them breeding can be carried out at village level by self-help.

These methods are best applied in densely populated areas where the number of breeding places is limited.

The measures have to be carried out as far as the flight range of the mosquito (1–2 km for *Anopheles* spp.). Most flying vectors follow an odour and hence their effective range is always against the wind — only a few hundred metres for *Anopheles* spp. but further for tsetse flies.

Control measures are:

- Draining water holes, ditches, and any accumulation of water around the village, or filling in holes and ditches so that water will

not accumulate (effective against *A. gambiae* and *Culex* spp.).
- Clearing bush and grass along water banks (*A. funestus*).
- All containers likely to hold water to be collected and disposed of (*Aedes* spp.).
- No water container should have water in it for longer than one week. This can be achieved by emptying the container once a week (*Aedes* spp.). Water containers should have lids or be covered with a larvicide, e.g., a few drops of oil.
- Clearing bush, replacing the bush by cultivation, will prevent breeding of *Glossina* spp.
- Snails can be controlled by disturbing their habitat: changes in water level, filling or draining the habitat, clearing the habitat, and use of molluscicides — some from local plants, e.g., Endod (*Phytolacca dodecandra*).

Protecting the susceptible individual

Individuals should be advised to screen their houses with mosquito gauze and to make ceilings in their houses. Mosquito nets should be used at night. Screens and nets should be of proper construction. Bed nets should be rectangular because when they are cone-shaped the sleeper is more likely to come into contact with the net and so risk being bitten. Bed nets can be impregnated with an insecticide to make them more effective as the insecticide kills and has an additional repellent effect. Bed nets are very important for infants and children. In houses unsuitable for nets, impregnated curtains may be used.

When screening of the whole house is too expensive, a part may be screened, e.g., the bedroom. Screening of only part is less effective because it does not keep out insects which are in search of the kitchen (notably flies).

Repellents give a valuable form of protection for those who have to be out in tsetse-infested areas or at night in mosquito-infested areas. Repellents act for 3–5 hours. In such circumstances, it is advisable to wear long trousers and long-sleeved shirts.

MALARIA

1. Malaria is an acute infective illness caused by protozoa of the genus *Plasmodium*.

The infection is often accompanied by attacks of fever which may be periodic.

Synonym: Homa ya mbu.

2. Occurrence and importance

Malaria is the commonest disease in tropical Africa, both in hospitals and the community. There are 400 million people at risk of infection, developing approximately 100 million clinical episodes per year. Of these, it is estimated that one million people, mainly children, will die.

Malaria is a very important cause of fever, convulsions, anaemia, and death. In pregnancy it results in low birthweight, abortion and maternal death, the former contributing to maternal and infant mortality. Malaria is also a major cause of economic loss through working and learning days lost.

The geographical distribution of malaria depends on the climatic conditions necessary for the survival of the vector (the *Anopheles* mosquito) and the parasite. The vector needs a humid climate, a warm average temperature, and suitable breeding places. The parasite only develops successfully in the mosquito at mean daily temperatures of 16°–21°C.

3. Epidemiology of malaria in Africa

There are four different species of the malaria parasite which infect man. These are: *Plasmodium falciparum*, *Plasmodium malariae*, *Plasmodium vivax* and *Plasmodium ovale* (see Table 4.5, page 96).

(a) In some areas transmission occurs throughout the year, e.g., in the warm humid coastal belt and the low-lying areas.
(b) Other areas are free of malaria, e.g., those above 1,500 metres altitude or with a very dry climate.

In many areas, the situation is between (a) and (b), and malaria transmission occurs during part of the year (the rainy season).

Spleen rate and endemicity: To classify malaria endemicity in an area, we measure the spleen rate. Repeated attacks of malaria often

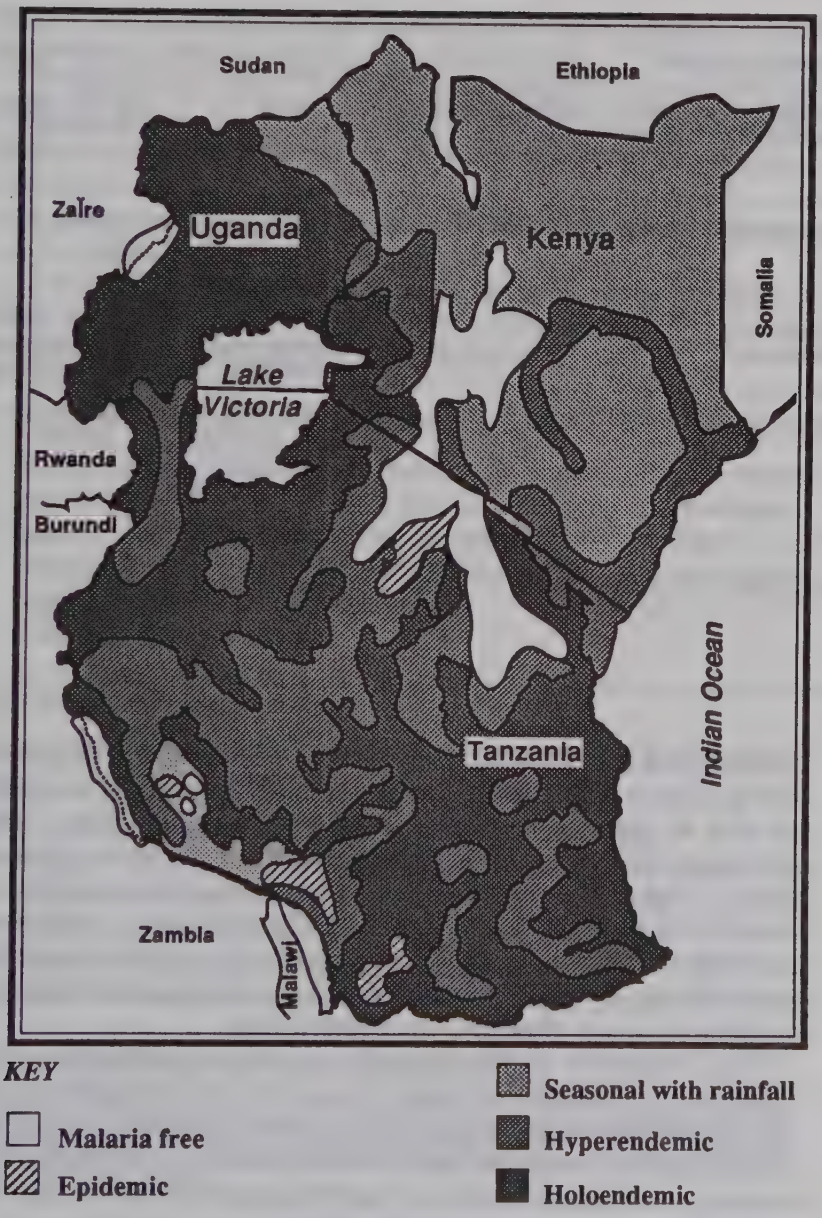

Figure 4.1 Distribution of malaria in eastern Africa

Table 4.4 Malaria endemicity, annual period of transmission and clinical picture

Malaria endemicity	Spleen rate in children 2–9	Spleen rate in adults	Months of transmission annually	Clinical features of malaria patients	Immunological description
Hypoendemic	<10%	Low	< 3 months	Liable to epidemics Clinical attacks severe in all age groups	Non-immunes
Mesoendemic	11–50%	<25%	3 –6 months	Mainly children Some adults	Moderate in adults Low in children
Hyperendemic	51–75%	>25%	>6 months	Clinically severe malaria mainly in children (occasionally in adults)	Adults semi-immune
Holoendemic	>75%	Low	All year	Severe malaria in children; mild malaria in adults	Adults immune

cause progressive enlargement of the spleen. Identification of splenic enlargement in different age groups in a particular area can therefore be used to estimate malarial endemicity. The spleen rate is the percentage of children between 2 and 9 years of age with an enlarged spleen. Depending on the spleen rate, an area is classified as hypoendemic (low transmission), mesoendemic (medium transmission), hyperendemic (high transmission), or holoendemic (transmission all year). This classification correlates more or less with the clinical picture of malaria in different areas.

The differences in this clinical picture are attributed to the development of malarial antibodies (immunity). This immunity results from frequent exposure to malaria infections and is partially protective. In this situation people are semi-immune. The effect of this partial immunity on malaria attacks is best illustrated by the following clinical patterns in a malaria-endemic area:

Birth to 6 months: Few attacks due to the malaria antibodies inherited from the mother (passive natural immunity); lack of para-

amino benzoic acid in breast milk (required for parasite folate metabolism), and the presence of haemoglobin F.

6 months to 5 years: Frequent severe attacks of malaria due to declining maternal antibodies. Spleen usually palpable and blood slides show a high parasite density (the number of infected erythrocytes is high).

5 to 10 years: Clinical symptoms diminish; the parasite density lowers (the immunity is developing).

Adult: Few clinical signs of malaria, spleen no longer palpable, a blood slide may reveal few parasites (low parasite density) without the person suffering from malaria; semi-immune state.

Other methods used to assess endemicity

- Prevalence and seasonal incidence of the disease, often restricted by the parasite rates which may be measured using blood slides or some of the modern "tools", e.g., ELISA or DNA probes;
- Vector species and their characteristics, e.g., density, sporozoite rate, biting habits, longevity;
- Climatic conditions.

Special risk groups

The people who are especially at risk from malaria and its consequences are:

- Children under 5 years of age (little or no immunity);
- Women in their first pregnancy (the malaria parasite thrives in the placenta causing abortion, low birthweight and death);
- Travellers from non-malarious areas (no immunity);
- Splenectomized patients (no immunity);
- Those with sickle-cell anaemia (causing sickle crisis).

Transmission and life cycle of malaria

Malaria is transmitted by the female *Anopheles* mosquito which requires blood for the development of her eggs. The male mosquito does not suck blood.

1. *Sporozoites* in the mosquito salivary glands are injected into the host via the proboscis of the mosquito. They rapidly migrate to the liver and are only in the circulation for about 30 minutes.

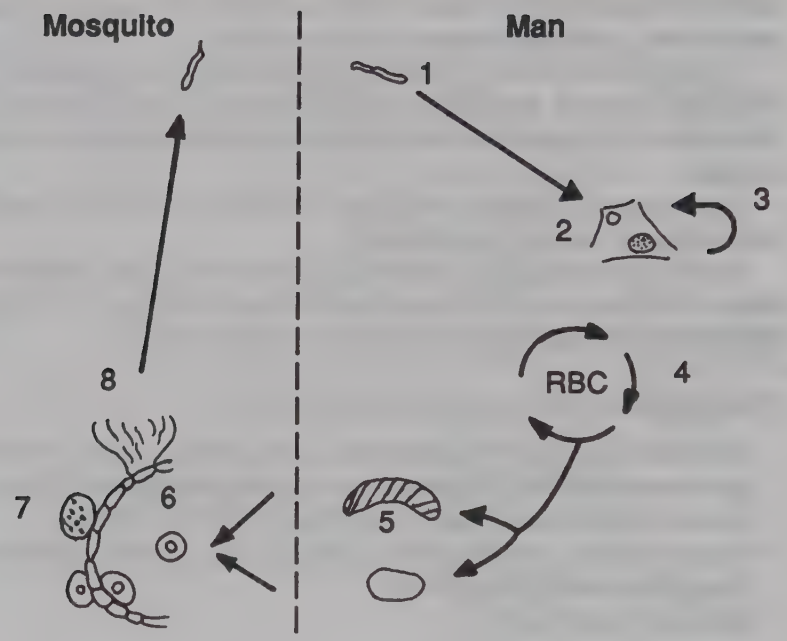

Figure 4.2 Simplified life cycle of malaria

Note

In man: 1. *Sporozoites* in mosquito salivary glands are injected while feeding and migrate to the liver, staying in the circulation for about 30 min. 2. *Hepatic (liver) stage* — the sporozoite, taken up by the Kupffer cells, passes into the hepatocyte and develops into a schizont, releasing 1000s of merozoites into the general circulation. 3. *Hypnozoites* only occur in *P. ovale* and *P. vivax*. They mature unpredictably and release new merozoites responsible for recurrent infections, possibly for life, if not specifically treated. 4. *Erythrocytic or blood stage* — hepatic merozoites invade red blood cells (RBC) and develop from 'rings' into blood schizonts. These schizonts rupture the RBC releasing more merozoites. 5. *Gametocytes* — at an unknown stage/time in the RBC cycle 'sexual' forms develop which are responsible for the transmission of the parasite. **In the mosquito:** 6. *Sexual reproduction* — the male and female gametocytes develop into gametes and fuse in the stomach of the mosquito (zygote). The zygote develops into a mobile ookinete which migrates through the stomach to form: 7. *Oocyst* which matures and releases: 8. *Sporozoites* which migrate to the salivary glands ready for delivery with the next blood meal.

2. *The hepatic stage*. The sporozoite is taken up by the Kupffer cells of the liver and then passes through into the hepatocyte or liver cell. There, it develops into a hepatic schizont, releasing thousands of merozoites into the general circulation. This takes 9–14 days.
3. *Hypnozoites*. This stage of the parasite only occurs in *P. ovale* and *P. vivax*. The hypnozoites mature unpredictably and release new merozoites responsible for recurrent infections up to 18 months after primary infection, even though earlier blood stages may have been cured. Without specific treatment, using primaquine, it is thought that this stage may remain in the liver for life.
4. *Erythrocytic or blood stage*. The merozoites released from the liver invade individual red blood cells (RBCs) and then develop within the RBCs from "rings" into blood schizonts. The schizonts then rupture the RBC releasing numerous merozoites which invade new RBCs. It is this part of the cycle that causes clinical illness and the length of time for each RBC cycle gives the periodicity of the symptoms.
5. *Gametocytes*. At an unknown stage/time in the RBC cycle "sexual" forms develop, which are in turn responsible for the survival of the parasite. These male and female gametocytes circulate for a few weeks and are taken up by the feeding mosquito.

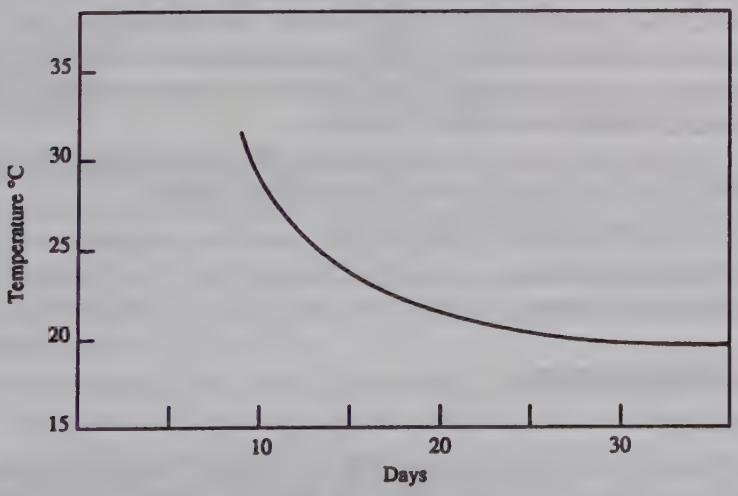

Figure 4.3 Time for parasite development in mosquito in relation to mean daily temperature

6. *Sexual reproduction.* The male and female gametocytes develop into gametes and then fuse in the stomach of the mosquito to form a zygote. This develops into a mobile ookinete which migrates through the wall of the stomach to form an oocyst.
7. *Oocyst.* This matures and releases sporozoites.
8. *Sporozoites,* which migrate to the salivary glands ready for delivery with the mosquito's next meal.

Major Anopheles *species of importance in Africa*

(a) *Anopheles gambiae* complex: this is the most important vector in Africa and is a complex of six closely related species. These mosquitoes breed in temporary water bodies exposed to sunlight such as pools and puddles which are mostly found during the rainy season. This mosquito is responsible for most seasonal malaria transmission.
(b) *Anopheles funestus:* breeds in permanent vegetation such as swamps and rice fields.
(c) *Anopheles melas:* is adapted to brackish (slightly salty) water.

The development of mosquitoes from eggs to larvae to adults is temperature-dependent such that at 31°C it will take 7 days and at 20°C it will take 20 days. The development of the malaria parasite in the mosquito is also temperature-dependent (see Figure 4.3) such that at 30°C it will takes 9 days, but below 16°C the mosquito is likely to have died before the parasite is ready for transmission as a sporozoite. Temperature, and hence altitude are, therefore, important determinants of malaria transmission (see Figure 4.1).

In each malarious area often only one or two species are important vectors. Your area may have its own adapted species.

Other ways of transmission are by:

(a) Blood transfusion;
(b) Via the placenta.

4. Clinical picture

Pathology

The sporozoites and liver stages do not cause clinical symptoms. Illness only begins once the invaded red blood cells (4) rupture to release new merozoites and vasoactive peptides which also activate

the immune system. Thereafter, late-stage trophozoites and schizonts are hidden in deep tissues (spleen, brain, liver) where they may cause significant damage. This involves immune complex formation, blockage of capillaries, breakdown of normal capillary lining and abnormal metabolic activity (lactic acidosis) which may lead to severe illness.

The merozoites of *Plasmodium falciparum* are able to invade erythrocytes of all ages (young and old). The merozoites of the other plasmodia prefer young erythrocytes. *P. falciparum* infections also multiply more efficiently, resulting in a higher parasite density and hence more severe clinical illness.

The combination of high parasite density and micropathology makes falciparum infections very dangerous — hence the name malignant malaria.

Note
The signs and symptoms of malaria are non-specific, and can mimic those of many other infections.

Symptoms	*Signs*
• Fever and chills	High temperature, rigors
• General malaise	Periods of sweating and diarrhoea
• Joint pains, backache	
• Nausea, vomiting and diarrhoea	Spleen enlargement, jaundice (haemolytic)
• Headache	Convulsions
• Dizziness	Anaemia (mainly haemolytic)

Fever: Periodic fever is usually a feature only of the less dangerous forms of malaria caused by *P. ovale*, *P. malariae* and *P. vivax*. In *P. falciparum* malaria, however, the erythrocytic stages of the parasites are frequently not synchronized, leading to a loss of fever periodicity.

A typical attack of fever has three stages: (a) cold, (b) hot, and (c) sweating stages:

(a) The cold stage: the temperature rises and the patient shivers; lasts 1–2 hours. In this stage, the infected red cells burst releasing merozoites, and many ring forms are seen in the blood smear.
(b) The hot stage: the temperature is high (about 40°C), the skin is dry and hot, there is severe headache, often nausea and vomiting; lasts 3–4 hours.

Table 4.5 Some characteristics of the different malaria species affecting man

Species in eastern Africa	Frequency as a cause of malaria	Incubation period (days)	Average parasitaemia per µl blood	First attack	Description
Plasmodium falciparum	>90%	9–10	200–500(000s) Dense	Severe in non-immunes	Malignant malaria
Plasmodium malariae	15%	28 (18–40) (usually mixed)	6,000	Mild	Quartan malaria
Plasmodium vivax	<1%	16+	20,000	Mild	Benign (Ethiopia)
P. ovale	<5%		9,000	Moderate	Tertian

Note
Parasitaemia is the number of parasites per microlitre of blood.

(c) The sweating stage: the temperature falls rapidly; the patient sweats profusely — his bed clothes are soaked; lasts 2–4 hours. The patient is relieved but exhausted.

In between attacks the patient has few complaints (in uncomplicated malaria).

Spleen: The spleen enlarges with each attack of malaria because the spleen has to clear the damaged red cells and produce antibodies. In early infections it may not be palpable, but in later attacks it is enlarged and may be painful during the attack. After the age of 10 years, splenic enlargement is rare in holoendemic areas.

When a child in a holoendemic area gets older he develops partially effective immunity. The spleen becomes smaller and probably no longer palpable. In a few cases, however, the spleen continues to increase in size. This is thought to be an abnormal immunological reaction to malaria. The condition is called tropical splenomegaly syndrome (TSS). The syndrome only occurs in endemic malarial areas and mainly in young adults.

The features of TSS are:

- Hypersplenism resulting in thrombocytopenia, anaemia, decreased immunity, increased bacterial infections;

- Attacks of haemolysis resulting in anaemia and haemolytic jaundice. The attacks are more common in pregnancy and may be fatal (see under treatment).

There is always a raised IgM level with high antimalarial antibody levels.

Anaemia: The anaemia of malaria results from two main factors:

(a) haemolysis of parasitized red cells, and
(b) some bone-marrow suppression.

As the iron of the red cells is recycled, a pure iron deficiency picture is rare. Instead, the blood picture is usually mixed and reflects a high turnover of cells superimposed on some elements of nutritional deficiency. The latter may be exacerbated by helminth or fluke infestations.

Jaundice: Mild jaundice is usually the result of haemolysis. However, hepatitis may occur as a complication of *P. falciparum* malaria. Hepatitis has also been caused by prophylactic use of amodiaquine (see treatment, below).

Diagnosis

Malaria presents as a flu-like illness.

- Clinical diagnosis — presenting *symptoms* may suggest malaria.
- Therapeutic diagnosis — patients' complaints respond to malaria treatment.

  A clinical or therapeutic diagnosis is not accurate and should *always* be supplemented by laboratory diagnosis.
- Laboratory diagnosis — malaria parasites in blood smear.

Each of these methods has its own limitations, as is shown in the following stories.

Story one: In a holoendemic area, an adult presented himself with fever at the health centre. The nurse sent him to the laboratory for blood examination. The report — "malaria parasites present" — was back before the patient was examined clinically.

The medical assistant was busy and so prescribed malaria treatment straight away. The next day the patient still complained of fever and slight abdominal pain. He was ordered to complete his full course of chloroquine before returning to the health centre again. Some days

later, he was brought in on a stretcher by his relatives. He now had manifest signs of generalized peritonitis. He was referred to the district hospital where laparotomy was done.

Diagnosis: typhoid with perforation

Moral: In a holoendemic area the presence of a few parasites may not explain the symptoms. Other causes must be ruled out by examining the patient.

Story two: In a holoendemic area, a healthy looking man donated one pint of blood for his sister. She was undergoing a Caesarean section for obstructed labour.

During the operation she was given one bottle of blood, one bottle of Dextran, and normal saline. Two days after the operation she developed some fever.

Suspected diagnosis: postoperative sepsis. Treatment was started with penicillin. After 2 days there was no improvement and the therapy was changed to broad-spectrum antibiotics. During the night the temperature rose higher. The nurse on duty took a blood slide as a routine, and the result was "malaria parasites seen". After chloroquine treatment the patient improved.

Moral: All donated blood should be screened for malaria.

Story three: A man from a mountainous area reported to the health centre complaining of having felt unwell for some weeks. The man looked ill and had a temperature of 38.5°C. Nothing significant was found and a thick blood film was negative.

The patient was sent home on chloroquine and when seen 3 days later he was better. Malaria seemed to be proven. Three months later the same patient was admitted, severely wasted, febrile, dyspnoeic and with obvious pain in the lower right chest. He had a massive right pleural effusion, the liver was enlarged to the umbilicus, smooth and very tender.

Diagnosis: amoebic liver abscess

Moral: Most fevers resolve spontaneously. Chloroquine has a potent anti-inflammatory action and has a non-specific effect in many illnesses, and a specific effect in some other parasitic diseases (e.g., hepatic amoebiasis). Malaria is very unlikely in mountainous areas.

Story four: A young boy came to the health centre with fever and was treated with chloroquine but no blood slide was taken. He was reviewed after 10 days as he was still unwell. A blood slide was done and was positive. What is the diagnosis? Is this now a first attack of malaria (the initial fever being due to a virus) or malaria resistant to chloroquine?

Moral: Always do a blood smear and you will know what you are dealing with.

The following facts should be kept in mind:

(a) A diagnosis of clinical malaria is basically a diagnosis of pyrexia of unknown origin which requires further investigation (see below, differential diagnosis).
(b) Everyone in a holoendemic malaria area may have a few malaria parasites in his blood but these are not necessarily responsible for his presenting symptoms.
(c) Improvement after a therapeutic (treatment) trial of antimalarials does not always confirm the diagnosis of malaria.

If malaria is suspected, the diagnosis should be confirmed by the laboratory, especially in areas where relapsing fever or trypanosomiasis occur.

Differential diagnosis: Fever in adults and children is common and can have many causes, e.g., viral infections, but the diagnosis is difficult to prove. A careful history and proper examination may reveal the right diagnosis.

The most common causes of fever, *apart from short virus infections like dengue*, are listed in Table 4.6, page 100. (In women in the reproductive age, conditions related to childbirth such as septic abortion, puerperal sepsis and pelvic inflammatory disease, must be excluded as causes of fever.)

5. Management of a malaria episode

Always try to confirm the diagnosis of malaria with a blood slide. Cases may be divided into uncomplicated or complicated (serious), and special attention should be paid to drug resistance.

Table 4.6 Differential diagnosis of fever

CHILDREN	Disease	Diagnostic criteria
Probabilities	Malaria	Blood slide
	Early measles	Koplik's spots
	Otitis media	Ear-drum examination
	Upper respiratory tract infection	Physical examination
Possibilities	Meningitis	Neck stiffness Bulging fontanelle CSF examination
	Urinary tract infection	Urine sediment examination
	Tonsillitis	Pus on tonsils
Rarities	Relapsing fever	Blood slide
	Trypanosomiasis	Blood slide Physical examination
ADULTS		
Probabilities	Malaria	Blood slide
	Relapsing fever	Blood slide
	Meningitis	Signs of meningeal irritation CSF examination
	Pneumonia	Dyspnoea Fine crepitations Rapid respiration
	Urinary tract infections	Urine sediment examination
Possibilities	Typhoid fever	Continual fever, blood culture, Widal test
	Acute bronchitis	Cough, sputum examination
	Rheumatic fever	Arthralgia, history of sore throat
	Hepatitis	Jaundice
	Subacute bacterial endocarditis	Heart murmurs, enlarged heart, blood culture
	Filariasis	Eosinophilia, night blood slide
	Septicaemia	White count, blood culture
	Abscesses	
	Tuberculosis	Sputum, physical examination
	AIDS	Physical examination Blood test
Rarities	Amoebiasis	Liver enlargement, white cell count, ESR
	Trypanosomiasis	Blood slide Lymphadenopathy
	Leptospirosis	Jaundice, bleeding, meningism
	Hodgkin's disease	Lymphadenopathy
	Leukaemia, reticulosis	White cell count, bone-marrow examination
	Appendicitis	Pain and rebound tenderness right lower abdomen
	Peritonitis	
	Visceral leishmaniasis	Endemic area: blood test, tissue and blood smears

Uncomplicated malaria (see definition of complicated malaria page 102)

The first line of treatment is oral chloroquine base 25mg/kg divided over 3 days (day 1, 10mg/kg; day 2, 10mg/kg; day 3, 5mg/kg; or day 1, 10mg/kg, followed by 5mg/kg after 6 hours; day 2, 5mg/kg; day 3, 5mg/kg). See below for uncomplicated chloroquine-resistant malaria.

Chloroquine is available as two different salts: chloroquine sulphate (trade names Resochin, Aralen, Malarex) and chloroquine phosphate (trade name Nivaquine). Both contain 150 mg of chloroquine base, the active ingredient, and are extremely well and rapidly absorbed when taken orally.

If the patient does not respond adequately or remains parasitaemic up to day 14 then consider a second-line antimalarial as this is chloroquine resistance.

Chloroquine resistance has spread throughout eastern Africa. *In vivo* studies in 1991 indicated that resistance has reached a level of approximately 40% in endemic areas. Resistance is commoner in non-immune persons as they have no immunity to help the drug attack the parasite. Amodiaquine resistance was found to be much lower, and there was less than 5% resistance to Fansidar and Metakelfin, and no confirmed resistance to quinine, Mefloquine or Halofantrine.

Choice of second-line antimalarials, in the following order is:

1. Amodiaquine: 10 mg/kg, 10 mg/kg, 5 mg/kg, i.e., 3 doses over 3 days;
2. Pyrimethamine-sulfa combinations: 3 tabs/60 kg single dose;
3. Quinine sulphate 10 mg/kg 8 hourly for 5 days.

Further blood slides and observation are necessary to ensure parasite clearance and clinical recovery.

NB: Do not use a second-line antimalarial if the blood slide is negative.

Antimalarial injections. These should only be used if the patient is unable to take drugs orally or is seriously ill. Care must be taken to avoid toxic overdosage.

The chloroquine dose is 5 mg/kg to a maximum of 200 mg — repeated 6 hourly until the patient is able to take oral medication.

Chloroquine injections can be dangerous

Management of severe malaria

Definition: Severe/complicated malaria includes any episode of slide-confirmed *P. falciparum* malaria with any of the following:

1. Hyperparasitaemia (more than 5%);
2. Severe anaemia (less than 5 g/dl);
3. Hypoglycaemia (less than 2.2 mmol/l);
4. Cerebral dysfunction — ranging from confusion to coma;
5. Convulsions;
6. Concurrent infections — pneumonia, septicaemia;
7. Major organ failure — renal, hepatic;
8. Pulmonary oedema;
9. Heart failure.

Parenteral antimalarials are indicated. Choices include:

1. Quinine dihydrochloride in 5% dextrose, 20 mg/kg loading dose and then 10 mg/kg 8–12 hourly thereafter for 5 days, both intravenously over 4 hours;
2. Intramuscular quinine 20 mg/kg loading dose plus 10 mg/kg maintenance dose 8–12hourly for 5 days;
3. Short-course quinine for 3 days, as above, plus a single dose of pyrimethamine sulfadoxine at the outset.

Change to the oral route as soon as possible. The addition of tetracycline/cotrimoxazole to quinine regimes appears to be unnecessary at present in eastern Africa.

Individual complications and their management

Hyperparasitaemia (>5%): Control parasitaemia as rapidly as possible; exchange transfusion not of proven value.

Severe anaemia (<5g/dl): Treat the malaria aggressively with quinine and add folic acid and iron replacement. If the patient is in cardiac failure, consider transfusion of packed red blood cells (HIV negative!) along with frusemide diuretic and digoxin.

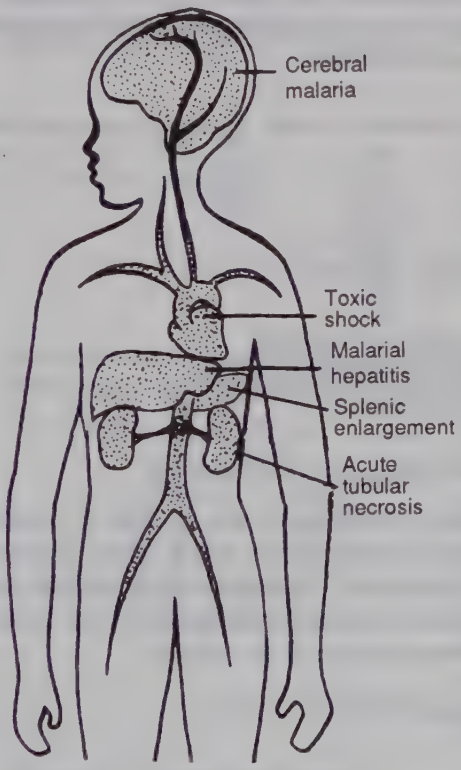

Figure 4.4 **Complications of severe malaria**

Brain: Mental disturbance appearing as acute psychosis, meningitis-like symptoms; coma.
Shock: Development of a shock syndrome caused probably by the amount of toxins produced (toxic shock).
Liver: Malarial hepatitis with hepatomegaly and jaundice.
Kidney: Acute tubular necrosis due to anoxia. Result: anuria with consequent uraemia.
Spleen: The normal function of the spleen is to take away old and abnormal erythrocytes. Due to the infection the spleen has to absorb a huge number of cells and increases in size. Therefore splenic enlargement is a common finding in acute malaria.

Transfusion volume: 6 ml/kg body weight per gram of desired haemoglobin increase. Drip rate: 2 ml/kg/hr. For example, for a 9 kg child requiring an increase of 3 g/dl:

Total volume = 6 ml x 9 kg x 3 g = 162 ml at a rate of 9 x 2 = 18 ml/hr.

Hypoglycaemia (<2.2 mmol/l): Give an intravenous bolus of 20% glucose (a dose of 0.33g/kg) and then maintain with an intravenous drip of 5% dextrose.

Convulsions: Establish the cause and treat accordingly. For control of convulsions give injections of paraldehyde or Valium (diazepam).

If the temperature is very high, antipyretics and tepid sponging are useful. Give appropriate treatment for hypoglycaemia and concurrent conditions such as epilepsy or meningitis.

Concurrent infections: Establish cause and give appropriate antibiotics intravenously.

Organ failure:

Renal: Acute tubular necrosis or nephritis
Hepatic: Toxic hepatitis
Cardiac: Cardiac failure/pulmonary oedema
Shock: Hypovolaemia, gastroenteritis.
Establish cause and treat accordingly.

Treatment of tropical splenomegaly syndrome

Long-term treatment: malaria prophylaxis for at least 1 year or sometimes for life. The best prophylaxis in these cases is causal prophylaxis with proguanil (see control). Splenectomy is not indicated as the immune disorder is not cured and hepatomegaly, severe malaria attacks, and sepsis may follow removal of the spleen.

6. Prevention and control

Levels of control:

(a) Vector control — see Introduction;
(b) Individual protection — see Introduction;
(c) Case finding and treatment;
(d) **Chemoprophylaxis** should only be used as an addition to vector avoidance in those at high risk. Widespread use in endemic communities should be avoided because that tends to accelerate the development of resistance.

Chemoprophylaxis is indicated for those with no immunity who are exposed to malaria through travel in endemic areas and who will not be resident there for life. Prophylaxis is also essential for pregnant women, especially those having their first babies, as it protects them from severe haemolysis, abortion, stillbirth or low-birth-weight babies, and death. Sickle-cell disease, splenectomy and malnutrition are also indications for chemoprophylaxis.

Drugs used for chemoprophylaxis include:

- Proguanil (Paludrine) 200 mg daily (adult dose).

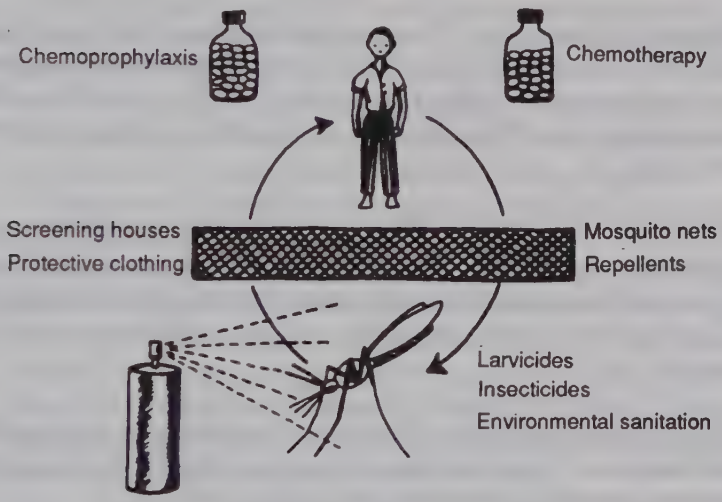

Figure 4.5 **Malaria control**

Malaria policy in East Africa

The endemicity of malaria in an area is an important factor in choosing the type of malaria control programme which is most suitable.

The World Health Organization (WHO) has set four strategies for the control of malaria in endemic and non-endemic areas.

Stable or endemic areas: Here transmission is more or less continuous and immunity is high except in young children. Eradication is almost impossible and most of these countries are poor.

WHO tactical variant No. 1: The aim here is to reduce or prevent deaths from malaria. This is achieved through community health workers and health workers in health units being trained in early

diagnosis and treatment, and making available suitable drugs for treatment. Vector control is usually not feasible, but personal protection, especially the use of insecticided mosquito nets, is important.

WHO tactical variant No. 2 : The aim here is to reduce both deaths and sickness. It includes chemoprophylaxis for pregnant mothers, children at risk and possibly certain key workers. This is possible when primary health care (PHC) facilities are available and communities are involved.

WHO tactical variant No. 3: This is where vector control methods, such as indoor spraying, are added to treatment and chemoprophylaxis. This is possible in areas with well-organized PHC, adequate trained staff, and sufficient funds, and where malaria transmission is not so intense.

WHO tactical variant No. 4: This aims at eradication and uses all control measures. It is only possible in countries with very good facilities and funds and where transmission is low.

Unstable or epidemic malaria areas: In these areas transmission is seasonal and immunity may vary depending on the conditions. Malaria epidemics are feared because they result in high mortality. Malaria eradication has succeeded most in these areas — and both tactical variants No. 3 and No. 4 are appropriate. Residual insecticides are applied just before and during the transmission seasons. This approach has been used most successfully in Africa, in Ethiopia and Zimbabwe.

7. Action

- Plan with the health auxiliary and the local development committee the introduction of measures to reduce mosquito-man contact, and vector breeding sites.
- Set up or improve your laboratory to diagnose malaria with a blood slide.
- Advise local shopkeepers to stock chloroquine, and provide trained community health workers with chloroquine. Make sure that these

shopkeepers and CHWs know the correct dose and understand the importance of keeping to it.

- Give malaria prophylaxis to all high-risk children and pregnant women who attend your MCH clinics.

  Start the campaign in the most densely populated areas, or the villages around your health centre, and gradually increase the coverage.
- Check the dormitories of schools and other institutions:
  — encourage the installation of window screens, and the use and maintenance of impregnated mosquito nets by the pupils.
  — suggest any improvements required to the heads of the schools or institutions.

  Curtains can also be treated with pyrethroids for better protection.
- Make sure injectable chloroquine is not misused by the staff in your health centre. Discuss the strict indications for the use of this drug in staff meetings, and also the right dose of oral chloroquine. Stress the need to refer patients for second-line drugs if there is no response to an adequate dose of chloroquine and that a blood slide must be made before using second-line drugs.
- Give health education on individual protection by chemoprophylaxis, where indicated, and the use of insecticided mosquito nets, especially for infants and children.
- Advise people on the need for ceilings and insect screens in houses.
- In urban areas larvicides can be used if funds are available — although old engine oil with or without vegetable oil is effective.

Chemoprophylaxis for all pregnant women, especially primigravidae, and children at special risk

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8. Summary

Malaria is an acute systemic infection caused by four types of plasmodia. It is characterized by fever, joint pains, headache, and haemolytic anaemia. Malaria is still the commonest disease in tropical Africa, and may be hypo-, meso-, hyper-, or holo-endemic.

Transmission is mostly by *nocturnal A. gambiae* and *A. funestus* mosquitoes.

Chloroquine is still the first line of treatment but resistance is increasing and other drugs often have to be used.

Eradication of malaria has proved to be very difficult in most parts of Africa. In eastern Africa, control measures include early treatment, chemoprophylaxis for pregnant women, patients with tropical splenomegaly syndrome and children at risk, and the use of insecticided mosquito nets for individual protection.

Blood slides are very important for diagnosis and correct management

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RELAPSING FEVER

1. Relapsing fever is an acute, infectious, bacterial (spirochaetal) disease, characterized by alternating febrile periods.

Synonyms: Recurrent fever, tick fever.

There are two types of relapsing fever:

(a) Louse-borne relapsing fever transmitted by lice.
(b) Tick-borne relapsing fever transmitted by soft ticks.

2. Occurrence and importance

Louse-borne relapsing fever

The disease is transmitted by the human head louse, *Pediculus capitis* and the common body louse, *Pediculus corporis*, the latter being the more common. It tends to occur in epidemics.

Pediculus corporis lives in clothes which are worn continuously without adequate laundering. The disease is likely to occur in conditions where many poor people huddle together in cold weather without changing or washing clothes. These conditions also exist in refugee camps, e.g., following war, famine or natural disasters. The louse is unable to survive without human contact. It is for the above reasons that the disease has an epidemic character.

Louse-borne relapsing fever has been reported in Rwanda, Sudan, Ethiopia, Somalia and Uganda.

Tick-borne relapsing fever

This is a zoonosis. It has been reported in western and north-west Tanzania, Uganda and Rwanda. It is transmitted by soft ticks (*Ornithodorus moubata*) which live in cracks and crevices of walls and floors. The disease can therefore be endemic in areas where housing conditions are poor.



Figure 4.6 **Most deaths from relapsing fever occur in children and pregnant women**

Adults in endemic areas are semi-immune. Children, newcomers and pregnant women are more susceptible to infection because of reduced or lack of immunity to relapsing fever. Therefore, most deaths from relapsing fever occur in these groups of people.

3. Epidemiology

Both types of relapsing fever are caused by spirochaetes of the genus *Borrelia* (louse-borne: *Borrelia recurrentis*: tick-borne: *Borrelia duttoni*).

Mode of transmission

Louse-borne relapsing fever is transmitted from person to person by the human louse. The spirochaetes are taken up when the louse feeds on the blood of an infected person. They then multiply within the body of the louse, but are not present in the saliva or coxal fluid of the louse. This louse only infects another person when it is crushed on the body near the bite wound.

The organisms are not transmitted to the offspring of the lice (in contrast to the cycle in ticks).

The transmission of tick-borne relapsing fever is different. When the tick sucks blood from an infected person, the spirochaetes are taken up and they multiply in the body of the tick. Within 1 week they appear in the tick's salivary glands and in the coxal fluid ready to be transmitted to a new host. The organisms can either be injected directly when the tick feeds on the host, or they invade the body through intact mucous membranes (e.g., in laboratory infections: Dutton, the discoverer of the disease, died from it).

The tick, *Ornithodorus moubata*, involved in the transmission of the disease in east Africa, needs a blood meal only occasionally.

The spirochaetes pass into the ovary of the tick and the offspring of an infected tick are automatically infected without having sucked infectious blood themselves (transovarian or vertical transmission). In this way, a house once inhabited by infectious ticks can remain dangerous for up to 10 years. There have been reports of people getting relapsing fever in a resthouse which was only visited for a few days in the year.

In humans, the spirochaetes can cross the placenta from mother to foetus. This may result in abortion, stillbirth, premature delivery or congenital infection in the newborn (compare with syphilis).

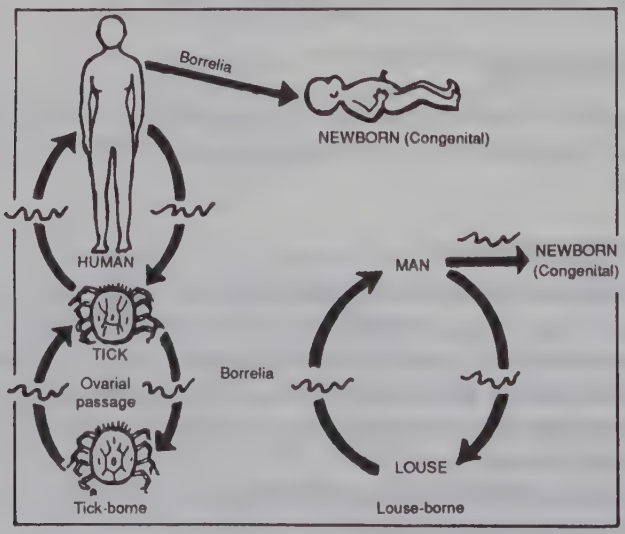

Figure 4.7 **Transmission routes of the relapsing fevers**

4. Clinical picture

The borreliae multiply in the body fluids. They produce endotoxins when they die. The toxins affect the cells of the liver, spleen, and endothelial cells of the capillaries.

After an incubation period of 2–10 days, the patient presents with a sudden onset of high fever, chills, headache, joint pains and body aches. There may be nausea and vomiting, jaundice and pain in the right upper quadrant.

After 5–7 days the temperature comes down by crisis. In two-thirds of patients a less severe relapse occurs 5–10 days after the initial attack. A second relapse may occur in about a quarter of these patients. Relapses are commoner with tick-borne relapsing fever than with the louse-

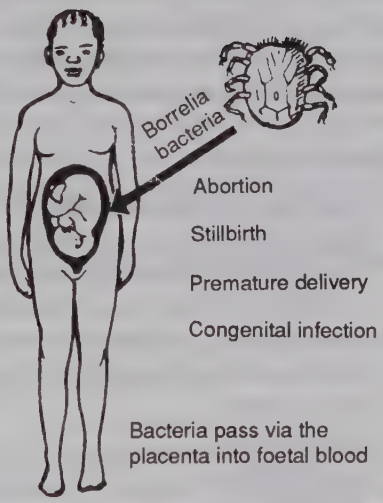

Figure 4.8 **Results of infection in pregnancy**

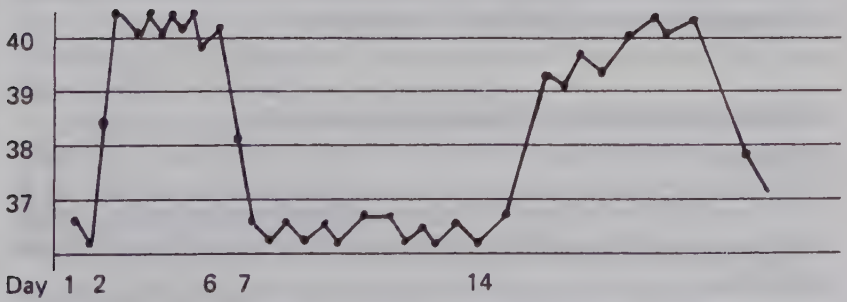

Figure 4.9 **The fluctuating temperature pattern in relapsing fever**

borne variety. In untreated cases, there may be up to ten relapses.

The fever and clinical signs regress each time after a relapse when immunity to the new strain of borreliae develops. But when another change occurs in the character of the organisms, they evade the immune system and this leads to another relapse.

The clinical picture may be so similar to malaria that only a blood smear can differentiate the two.

Complications

- Collapse and coma leading to death may occur in patients with little immunological resistance, pregnant women, and children. In louse-borne relapsing fever, deaths usually follow the first attack; in the tick-borne form, it follows subsequent attacks.
- Meningitis: lymphocytic meningitis.
- Eyes: iritis and atrophy of the optic nerve.
- Myocarditis.
- Liver failure.
- Bleeding.

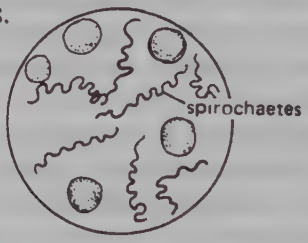

Figure 4.10 **Blood smear showing spirochaetes**

Diagnosis

Microscopic examination of a thick blood smear stained with Leishman or Giemsa, as is done for malaria, is useful in diagnosis. Malaria as a differential diagnosis can also be excluded at the same time. Differentiation between the two types of relapsing fever is not possible by microscopy.

5. Management of the individual patient

Treatment

First choice: Tetracycline 250 mg orally four times a day for 1 week, or 500 mg as a single oral dose (this was used successfully in Ethiopia in 1978).

Second choice: Injection PPF 0.4 mU for 5 days. A combination treatment of procaine penicillin 400,000 units intramuscularly followed

by tetracycline 500 mg four times a day for 5–7 days was also found to be effective and safe in the Sudan in 1977.

Attention

Some deaths occur after starting treatment as a result of a severe Jarisch-Herxheimer reaction (as in syphilis). The antibiotic suddenly kills a large number of spirochaetes which release toxins into the circulation causing the patient to collapse. This reaction is characterized by rapid breathing, chills and a fall in blood pressure. Patients must be nursed flat, given adequate fluids, and be confined to bed for at least 24 hours.

Tetracycline is contraindicated in pregnant women and in young children.

6. Prevention and control

Tick-borne relapsing fever

The best way to control the disease is to improve housing conditions. Houses in most parts of Africa are made of mud and wattle, and the roofs are grass thatched. Cracks appear in the walls when the mud dries, providing a hiding place for ticks. The application of plaster (sand and cement) over the mud walls would produce a smooth surface, thus eliminating the development of cracks. This is one feasible way of controlling tick-borne relapsing fever which is within the reach of everyone.

Another hiding place for ticks is grass thatched roofs. The use of corrugated-iron sheets for roofing would eliminate this problem but not everyone can afford them. It is hoped, however, that as the standards of living improve, more people will be able to afford better housing and the disease will disappear.

Insecticides can be used to kill ticks. Gammexane (Gammatox plus) is recommended. One kilogram of gammexane is enough to make 40 litres of solution which can then be sprayed or applied on the floor and walls and in cracks.

Control of tick fever can also be achieved by reducing exposure to the infection. People who sleep on the floor have the greatest risk of infection. The use of beds should therefore be encouraged.

Mass treatment of patients is not useful as the important reservoir

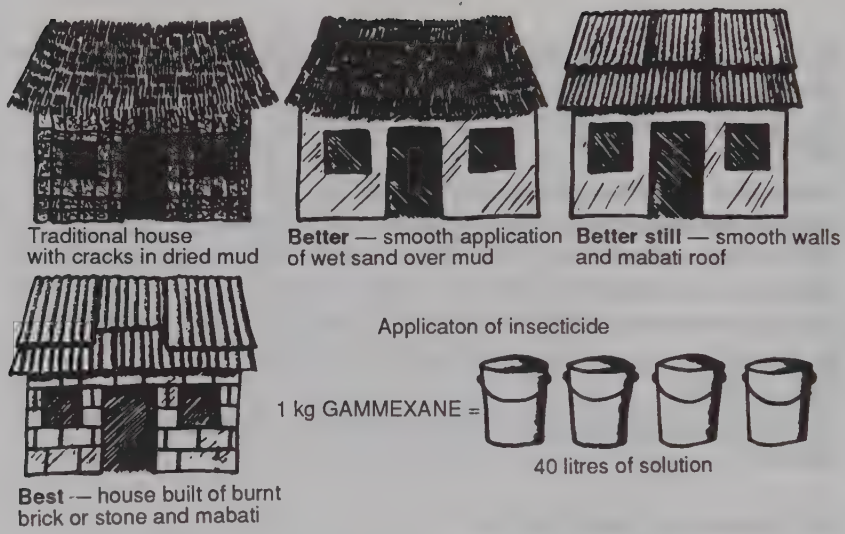

Figure 4.11 **Control of relapsing fever by better housing**

is the tick; once infected a tick is able to produce offspring which are also infectious.

Louse-borne relapsing fever

This can be controlled by improved personal hygiene and the frequent washing and changing of clothes. The vector population can be reduced by using insecticides such as DDT or malathion powder to disinfect clothes, or by boiling clothes to kill the lice and the eggs.

7. Action

- Do routine thick blood smears in all patients presenting with fever. In this way, you will not miss cases of relapsing fever.
- Encourage patients to spray their houses with insecticide in order to avoid re-infection.
- Give health education on the need for improved housing (walls, roof, bed) and personal hygiene.
- Pregnant women and children under 5 years are at risk of getting severe relapsing fever. Be on the alert for the disease and always

take thick blood smears so as to diagnose relapsing fever early and manage it accordingly.

8. Summary

Relapsing fever is an acute infectious disease caused by spirochaetes of the borrelia group. It is characterized by attacks of fever.

Transmission is by body lice and soft ticks in certain endemic areas of Africa. Treatment is with antibiotics, starting with low doses so as to avoid the development of Jarisch-Herxheimer reactions. Prevention is mainly by vector control.

BANCROFTIAN FILARIASIS

1. Bancroftian filariasis is a disease caused by filarial worms, and mainly affects the lymphatic system. It is transmitted by mosquitoes.

2. Occurrence and importance

In Africa, filariasis is most frequent in the tropical coastal belt and the lake regions. Since the disease is transmitted by mosquitoes, it is rare in high altitudes and dry regions. The disease is more disfiguring than dangerous. In some hot coastal places over 30% of adult males have hydroceles, a late manifestation of the infection. The treatment for hydrocele is surgical and this may be a severe drain on limited hospital resources.

3. Epidemiology

Bancroftian filariasis is caused by *Wuchereria bancrofti*, a worm belonging to the nematode family. Mosquitoes transmit the disease from person to person. Two main genera are involved: *Culex* in urban areas and *Anopheles* in rural areas. When a mosquito carrying the microfilarial larva bites a new host, it deposits it into the subcutaneous tissue of the patient. The larva finds its way into the lymph vessels where it takes about 1 year to mature. The adult worm is 4–8 cm long and 0.2 mm thick.

The mature female worms start giving birth to live microfilariae. During the day, these microfilariae hide in the large vessels; at night, when the vector mosquitoes suck blood, the microfilariae appear in the peripheral blood. The mosquito takes up the microfilariae when it sucks blood from an infected person. The microfilariae undergo several changes in the body of the mosquito. After 10 days the mosquito is able to transmit the infection to other human beings when it bites them.

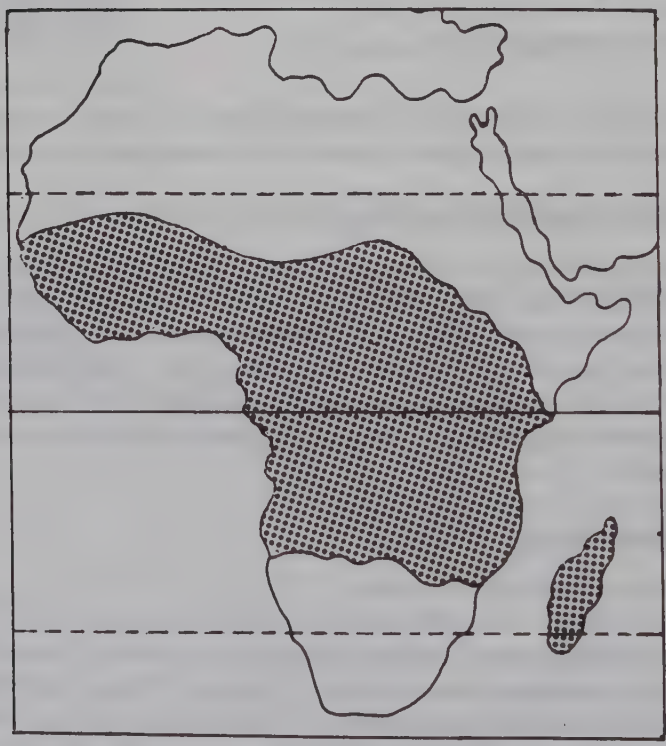

Figure 4.12 **Distribution of Bancroftian filariasis**

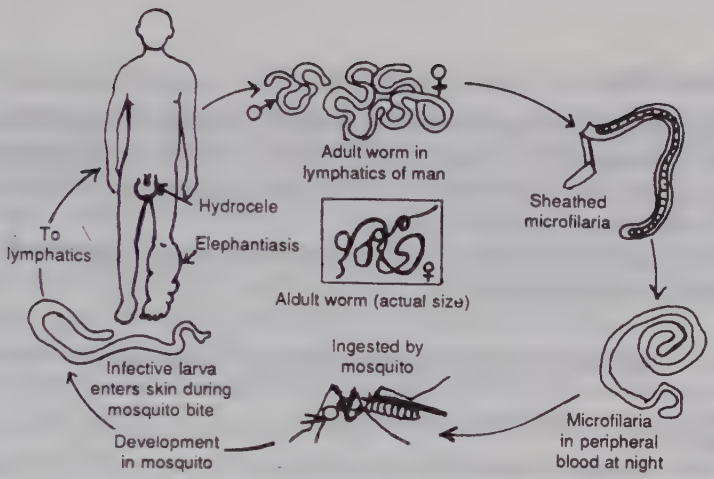

Figure 4.13 **Life cycle of *Wuchereria bancrofti***

4. Clinical picture

The presence of the worms in the lymph vessels gives rise to a foreign-body reaction. When the worms die, more foreign proteins are released causing an even more severe reaction. Three stages may be distinguished:

Acute phase

This starts a few months after the infection, and is characterized by fever, lymphadenopathy and eosinophilia.

At this stage, microfilariae are not yet demonstrable in the peripheral blood because the worms are not yet mature. The acute phase is mainly due to a hypersensitivity reaction.

Subacute phase

This stage sets in after about a year. The worms will have matured and microfilariae are present in the peripheral blood. Reactions to the adult worms cause attacks of fever with lymphangitis, funiculitis, and/or epididymitis. Recurrent attacks of funiculitis sooner or later result in the formation of a hydrocele.

Lesions caused by the microfilariae are less common and are associated with hypereosinophilia and lung symptoms (tropical pulmonary eosinophilia syndrome).

Chronic phase

After many years of repeated attacks, lymph glands and lymph vessels become obstructed. As a result lymphoedema develops. Lymphoedema is most often seen in the legs or scrotum (elephantiasis), but may also involve the vulva, breasts, or arms.

Since the adult worms have usually died, microfilariae are no longer seen in the blood.

Table 4.7 Summary of the clinical picture of Bancroftian filariasis

Bancroftian filariasis	Main signs and symptoms		Blood smear
Acute phase (first months)	Fever Eosinophilia Lymphadenopathy Lymphangitis		Negative
Subacute phase	Adult worms cause: Fever Lymphadenitis Funiculitis Epididymitis Hydrocele	Microfilariae cause: Hypereosinophilia Asthma-like attacks Fever	Positive
Chronic phase	Lymphoedema Elephantiasis Chyluria Hydrocele		Negative

Diagnosis

During the day time microfilariae are not present in the peripheral blood. To demonstrate the microfilariae blood slides should be taken either:

(a) Between 10.00 p.m. and 2.00 a.m.; or
(b) 45 minutes after a provocative dose of diethylcarbamazine 100 mg.

Puncture fluid from swollen glands or an hydrocele may also be examined.

Differential diagnosis: Filariasis should be differentiated from other causes of lymphangitis and recurrent fevers, such as acute bacterial lymphangitis, relapsing fever, malaria, septicaemia, TB, and AIDS. Elephantiasis is often not caused by filariasis but may be due to chronic damage of the feet by absorption of silicate crystals leading to obstruction of lymph flow. This condition (called lymphoconiosis) is prevented by wearing shoes, and occurs in Ethiopia, Cameroon, some coastal areas of East Africa and central Kenya.

5. Management of the individual patient

The drug of choice is diethylcarbamazine (Hetrazan or Banocide). This kills the microfilariae and has also some effect on the adult worm. It should not be given during the acute phase of the disease but afterwards.

The dose is 6 mg/kg in three divided doses, i.e., 150 mg (three tablets) three times a day for an average adult. Treatment should be given for 12 days. Other drugs are being investigated, e.g., Ivermectin.

To avoid reactions, antihistamine drugs and/or steroids can be given during the first days of treatment.

In the chronic phase of the disease, chemotherapy is no longer useful since there are no active worms. Surgical treatment is indicated for hydrocele and is sometimes possible for elephantiasis of the legs and scrotum. Refer patients for whom surgery might be indicated.

6. Prevention and control

- Reducing the vector population. *Anopheles* are vectors in rural areas. *Culex* mosquitoes often breed in septic tanks and pit latrines in urban areas. Therefore septic tanks and latrines have to be treated regularly with larvicides.

  A promising new larvicide, which has been used successfully in Zanzibar, is polystyrene powder. The powder expands into little beads of 1–2 mm diameter when put in boiling water. This mixture is then poured into the pit latrine where it acts as a larvicide to reduce mosquito breeding by 90% or more. This effect will last until the pit overflows.
- Reducing the human reservoir through organized mass campaigns. Campaigns should be continued for at least 10 years. Before

starting the campaign, however, a careful cost-benefit analysis should be made.
- Reducing exposure to the infection by screening houses, closing eaves, and using mosquito nets. Many bites are required to cause an infection, and so reducing bite frequency helps prevent cases.

7. Action

- If your health centre is located in an endemic area, make sure it is possible to take thick blood smears between 10 p.m. and 2 a.m. at night.
- Exclude filariasis in all cases of unexplained eosinophilia (with fever).
- With the help of your health auxiliary and the local development committee, organize a mosquito reduction campaign.
- Organize bulk purchase of local polystyrene powder for treating pit latrines.
- Give health education on personal protection, screening of houses, and the usefulness of mosquito nets.

8. Summary

Bancroftian filariasis is a chronic disease caused by the body's reaction to the presence of *Wuchereria bancrofti*, a nematode worm. Transmission is through mosquitoes. Diagnosis can be made by examining a thick blood slide taken at night. Control of filariasis is difficult. Individuals may protect themselves by screening their houses or using mosquito nets, and by protecting their latrines with polystyrene beads.

ONCHOCERCIASIS

1. Onchocerciasis is a chronic disease caused by *Onchocerca volvulus*, a filarial nematode worm. The disease manifests mainly as skin nodules on bony surfaces, and causes eye lesions which may result in blindness.

Synonym: River blindness.

2. Occurrence and importance

Onchocerciasis is a major cause of blindness in some parts of tropical Africa (especially the Volta River Basin in west Africa) where people are exposed to heavy and repeated infections. Eye lesions are uncommon in areas where infection is light.

The importance of this disease in eastern Africa has almost certainly been under-estimated and the disease is probably more widespread than has been reported.

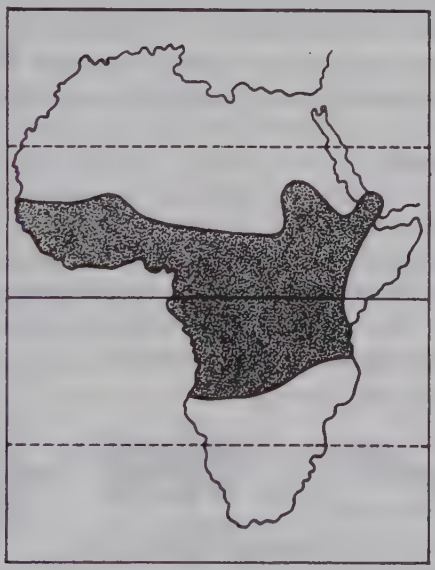

Figure 4.14 **Distribution of onchocerciasis in Africa**

The disease is transmitted by a small black fly called *Simulium damnosum*. The fly lays eggs in fast-running rivers or turbulent areas of a river. (When the river does not flow fast, the water does not contain enough oxygen to enable the eggs to develop into larvae.) The average flying distance of the fly is about 40 km (maximum 150 km). The disease is therefore likely to be present in the neighbourhood of fast-running mountain rivers.

3. Epidemiology

River blindness is caused by *Onchocerca volvulus*. The adult female worm is 33–35 cm long and 0.4 mm thick. The male worm is about 4 cm long and 0.2 mm thick. The adult worms live for 11–18 years and the female produces thousands of microfilariae which live for about 2 years.

The vector of onchocerciasis is the female *Simulium* fly. The fly takes up microfilariae when it sucks blood from an infected person. The microfilariae develop into infectious larvae which are passed on to a new host when the fly bites again. The *Simulium* fly attacks people outdoors during the day, but not in bright sunlight. Most bites occur around sunrise or sunset, or on cloudy days and in the shade.

4. Clinical picture

Pathology

The infectious larvae injected into the subcutaneous tissues develop into adult worms. The mature males and females then collect in balls, bound together by fibrous tissue which forms the typical nodule. The nodules are best seen on bony skin surfaces, e.g., elbow, shoulder, scapula, skull, ribs, and iliac crests.

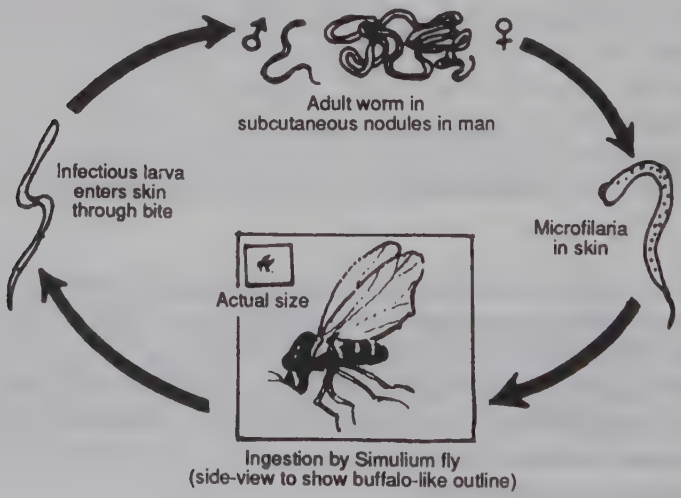

Figure 4.15 **Onchocerca cycle**

After 1 year, the adult worms start giving birth to microfilariae. The microfilariae migrate into the epidermis and subcutaneous tissues, and into the eye.

Onchocerciasis has three different clinical presentations:

(a) *Skin nodules:* these are caused by the adult worms; they are non-tender, rubbery and firm, ranging from 3 mm to 3 cm in diameter.

(b) *Dermatitis:* this is caused by a reaction to the presence of microfilariae in the epidermis, and manifests as itchy papules or macules. Afterwards the skin becomes loose, scaly, atrophic and depigmented.

(c) *River blindness:* this is a very serious complication of onchocerciasis caused by the presence of microfilariae in the cornea and the anterior chamber of the eye. At first, there is oedema of the conjunctiva, then corneal spots and a pannus begin to develop. The pannus starts at the lower limbus, unlike that of trachoma which affects the upper limbus. Finally, cataracts, iritis and glaucoma develop, leading to blindness.

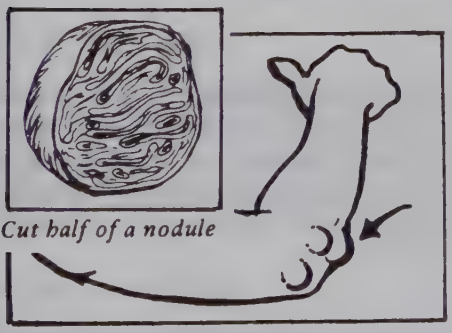

Figure 4.16 **Onchocerca nodules**

Diagnosis

The microfilariae of *Onchocerca volvulus* are not found in the blood. The diagnosis is made by demonstrating microfilariae in skin snips obtained from the thighs, buttocks, and iliac crests. The snip is placed in normal saline, shredded and examined under a microscope for microfilariae. Onchocerciasis skin nodules may be mistaken for sarcomatous or carcinomatous lesions if the surgically removed nodules are not cut open and properly examined for the presence of adult worms. This should always be done in areas where onchocerciasis is common.

5. Management of the individual patient

Onchocerciasis is not a fatal disease. If the patient has no serious complaints and is likely to be reinfected there is no urgency for treatment, since the traditional drugs used have been known to cause severe reactions.

The following groups of patients need treatment:

- Patients with eye lesions;
- Patients with severe skin lesions;
- Patients with heavy infections.

Two different kinds of treatment are needed:

- *To kill the microfilariae:* Diethylcarbamazine (Hetrazan) has been the traditional drug for treating onchocerciasis, but it has little effect on adult worms. It can cause severe reactions, and even death. Test doses are usually given in the first 3 days before the full dose of 2 mg/kg three times daily for 3 weeks is commenced.

  Ivermectin is a promising new drug which is very effective when given as a single dose; it will replace Hetrazan.
- *To kill or remove the adult worms* by surgical removal of nodules.

6. Prevention and control

Trials to control the disease by treatment of patients with Ivermectin are being conducted. Another promising control method is the continuous addition of insecticide to the water of rivers known to be breeding places of the *Simulium* fly. This method was used in Kenya to eradicate a focus of onchocerciasis.

7. Action

If river blindness is common in your community, inform your DMO and his health officer, and discuss your course of action.

8. Summary

Onchocerciasis is a chronic disease characterized by fibrous skin nodules, dermatitis and eye lesions. It occurs only near fast-flowing rivers, especially in highland areas, hence the name river blindness. The vector is the *Simulium* fly. Treatment is aimed at eliminating the

microfilariae and the adult worms using Hetrazan and Ivermectin. Vector control is the only method of prevention which has succeeded so far.

YELLOW FEVER

1. Yellow fever is an acute infectious disease caused by an arbovirus. It is characterized by sudden onset of fever, rigors, headache, nausea and vomiting, jaundice, and sometimes signs of liver and kidney failure.

2. Occurrence and importance

Tropical Africa is within what is internationally regarded as the yellow fever "endemic zone", though in fact there is no yellow fever in some countries, such as Tanzania. The vector, *Aedes aegypti* is, however, present in many countries, and if infection were introduced the results would be disastrous. The disease can spread very rapidly, and the case fatality rate may reach as high as 30% in non-immune populations.

3. Epidemiology

Yellow fever is a zoonosis of forest monkeys. The disease is transmitted among them by mosquitoes living in the tree tops. Man is infected when bitten by these mosquitoes in the forest; or he may be bitten by mosquitoes outside the forest which acquire the disease from monkeys leaving the forest to feed on banana and other agricultural plantations.

4. Clinical picture

Many infections are inapparent, leading to immunity without definite illness. In a classical case of yellow fever the onset is sudden with fever, headache and backache, nausea and vomiting. A bleeding tendency is common, resulting in epistaxis, bleeding of gums, haematemesis, and melaena. Liver-cell necrosis occurs and, depending on the severity, this may result in jaundice, liver failure and death. Involvement of the kidneys causes nephritis which results in albuminuria, and, occasionally, anuria and kidney failure. Patients who survive up to the seventh day of the disease usually recover.

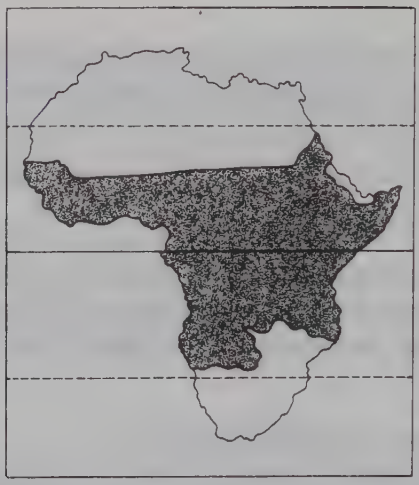

Figure 4.17 **Yellow fever endemic zone in Africa**

Diagnosis

In endemic areas, yellow fever should be suspected in patients who die or present with a febrile illness with jaundice and albuminuria.

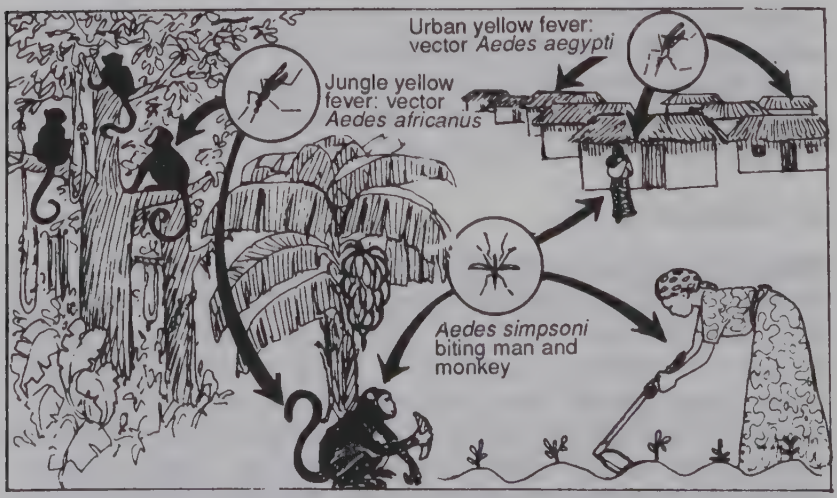

Figure 4.18 **Transmission of yellow fever**

Differential diagnosis:

Viral hepatitis, malaria, leptospirosis, relapsing fever, and Marburg, Ebola and Lassa fevers. Do appropriate laboratory tests to exclude these diseases.

5. Management of the individual patient

Since there are no specific drugs, supportive treatment is often all that is necessary. Avoid further spread of the infection by limiting contact between mosquitoes and the patient. The patient should be nursed in a well-screened room, or under a mosquito net. Inform the DMO immediately to come and see any patient you suspect to have yellow fever so that the diagnosis can be confirmed. Do not refer the patient unless he is very ill and needs hospital care. This way you limit spread of the disease.

6. Prevention and control

To avoid the spread of yellow fever, travellers to and from endemic areas need valid international immunization certificates. Immunization against yellow fever is carried out at city health services, regional hospitals or international airports. The vaccine contains a live attenuated virus and gives good protection for at least 10 years. Side effects are rare.

To avoid importation of infected mosquitoes, aircraft coming from endemic areas should be sprayed with aerosol insecticides.

Provided these measures are carried out correctly, an outbreak of yellow fever is unlikely in eastern Africa, and therefore routine immunization of the entire population is not necessary. In countries where epidemics occur, mass immunization of the population at risk is necessary to control the disease.

7. Action

Whenever you see a case of yellow fever:

- Isolate all the contacts in screened quarters to prevent spread of the infection by mosquitoes;
- Inform the DMO;
- Start a mass immunization campaign to cover all the surrounding areas infested with *Aedes* mosquitoes;

- Spray the houses of the patient and his neighbours with insecticides;
- Apply larvicides to all possible breeding places of *Aedes* mosquitoes, including small receptacles and the axils of certain plants.

8. Summary

Yellow fever is an internationally notifiable disease. It does not occur in all countries of Africa but the vector is present. Prevention is through immunization of travellers, and spraying of aircraft to avoid importation of mosquitoes carrying the virus.

TRYPANOSOMIASIS

1. Trypanosomiasis is a disease caused by a protozoan haemoflagellate transmitted to man, cattle, and wild animals by the *Glossina* fly (tsetse fly). Two trypanosoma species infect humans: *Trypanosoma brucei gambiense* (*Tbg*) and *Trypanosoma brucei rhodesiense* (*Tbr*). The disease is characterized by chronic fever, general weakness, cerebral involvement, and death if not treated adequately. *Tbg* causes a slow progressive "sleeping sickness", while *Tbr* causes an acute and rapidly progressing illness, usually leading to cardiac failure.

2. Occurrence and importance

Trypanosomiasis has interfered with the economic development of many parts of Africa because of its effect on the population of both cattle and people. The disease affects both cattle and man. It is invariably fatal over varying periods of time if not treated.

3. Epidemiology

T. b. brucei infects only cattle.

T. b. gambiense (*Tbg*) causes a slow chronic illness, resulting in death in several months or years from "sleeping sickness". Reservoirs include pigs, dogs, and antelope. *Tbg* is found in western and central Africa.

T. b. rhodesiense (*Tbr*) causes an acute, rapidly progressive illness with death from cardiac complications within several weeks or months. Reservoirs include antelope and pigs. *Tbr* is found in eastern Africa, now mostly in south-east Uganda.

The vectors

The disease is transmitted by tsetse flies, which live in areas of wooded vegetation and are usually not found on flat plains, closely cultivated areas, or areas densely inhabited by people.

There are two types of tsetse flies of importance:

- The riverine type which breeds along rivers and lakes;
- The woodland type which lives in open lightly wooded parkland away from water.

Tsetse flies of the riverine type are the main vectors of *T. brucei gambiense*. In eastern Africa, *Glossina palpalis* is the most important species. *Trypanosoma brucei gambiense* transmission occurs especially at river crossings and water holes where contact between man and fly can be very intense.

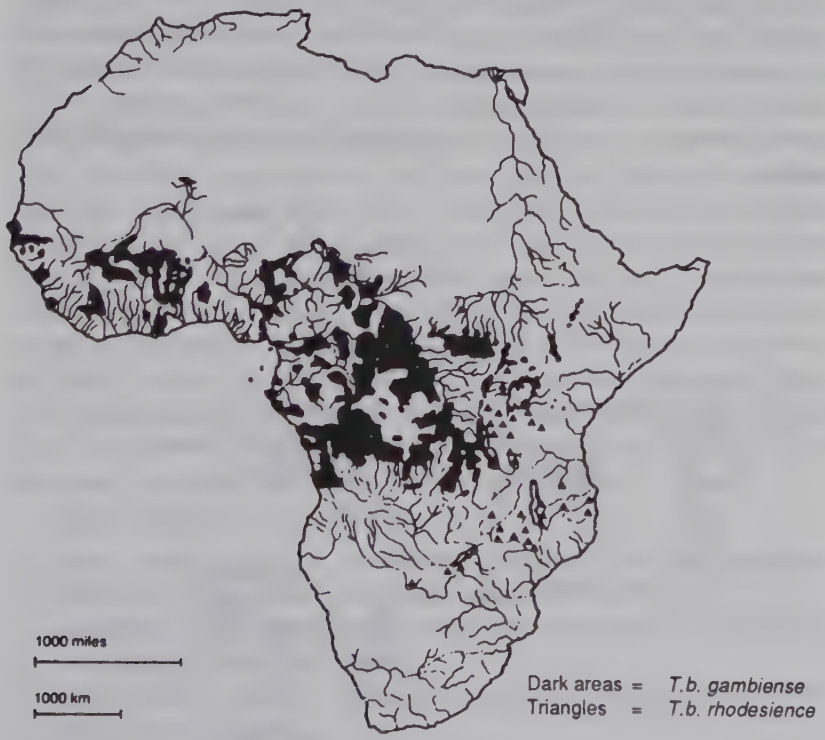

Figure 4.19 Distribution of different forms of sleeping sickness in Africa

The main vector of *T. brucei rhodesiense* is *Glossina morsitans*; it prefers to bite cattle and game but will also bite man. (In Busoga in Uganda an outbreak of *Tbr* appears to have been caused by *G. palpalis* at river crossings as well as in gardens.)

In the dry season the fly population is at a minimum and concentrated in certain limited areas which are known as permanent breeding places, primary foci or "homes". Tsetse flies hunt by sight and are attracted by moving dark objects, hence they follow buffaloes and vehicles.

Tsetse flies become infected with trypanosomes when they take a blood meal from infected persons or animals. After a period of time, during which the trypanosomes undergo developmental changes, the fly is able to transmit the disease when it bites another susceptible host. In heavily infested areas, about one fly in every 3,000 is infected with trypanosomes. Contact between man and fly is not always frequent because of the behaviour of the *Glossina* species and therefore trypanosomiasis may occur sporadically in people who live close to infested areas or people who, due to their profession, enter tsetse fly infested areas, e.g., wood cutters, honey collectors, game rangers, and hunters.

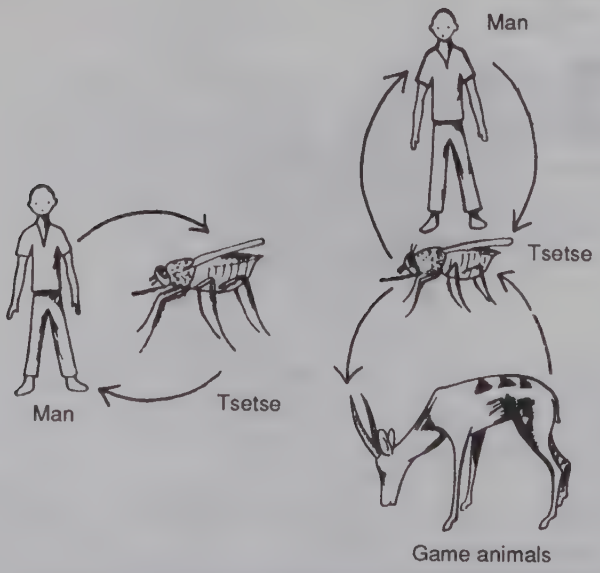

Figure 4.20 **Transmission of trypanosomiasis**

4. Clinical picture

Both types of trypanosomiasis run a similar clinical course, but *T.b. rhodesiense* infections are more rapid with death ensuing within a year without treatment, while in *T.b. gambiense* infections, death ensues after 2–3 years.

The disease presents in three stages: the primary or chancre stage, the systemic or blood stage, and the cerebral or sleeping-sickness stage. The incubation period is 2 weeks for *Tbr*, and may take months in *Tbg* infections.

Chancre stage

The chancre stage is more commonly seen in *Tbr*. It is characterized by the appearance of a painful indurated erythematous nodule at the site of the bite within a few days of the tsetse bite. The chancre may last 1–2 weeks and then resolve. This stage occurs in 70% of cases in Europeans but is rare in Africans.

Systemic illness or blood stage

This stage is characterized by the dissemination of the trypanosomes to blood, lymph and lymph nodes. There is an associated fever which has no typical pattern but tends to recur at intervals of days or weeks. The fever may last for weeks and then resolve, or may be continuous without remitting. This is common in *Tbr* infections. Debilitation, anaemia and general weakness follow. The spleen and several groups of lymph nodes become enlarged. The supraclavicular and lower posterior cervical lymph nodes are visibly affected in 80% of cases of *Tbg* infections (Winterbottom's sign). The nodes are rubbery, often painless, but rarely may form abscesses.

Other signs and symptoms are:

- Rash, usually pruritic, beginning around 6–8 weeks; migratory and lasts 1–2 months; seen better on white skins;
- Hepatosplenomegaly not common early but more frequent with increasing duration of illness;
- Anorexia resulting in weight loss, debility, pitting oedema of the face and lower legs;
- Impotence and menstrual irregularities;
- Cardiac failure (especially in *Tbr*).

Cerebral or sleeping sickness stage

This terminal stage of trypanosomiasis is due to invasion of the brain by the trypanosomes. In *Tbg* infections, this stage appears about 2 years after the initial infection. In *Tbr* infections this interval can be very short, taking only a few months. It is characterized by progressive mental deterioration, coma and death if not treated. Convulsions and localized signs such as hemiplegia and facial palsy, may occur. Patients are very weak; they sleep during the day, but are restless at night. As the disease progresses they become worse and eventually die if not treated.

Diagnosis

- Microscopic examination of chancre fluid for live organisms.
- Examination of the buffy coat of whole blood for organisms after spinning it in a centrifuge to sediment the red cells.
- *Wet blood smear:* Put a drop of blood on a slide, cover with a coverslip, and look for trypanosomes. This is a simple and inexpensive method, but it is not very sensitive. It is, however, useful for diagnosing *Tbr* which has waves of parasitaemia approximately every 8–15 days. With daily wet blood smears one might see trypanosomes. In *Tbg*, parasite peaks may be months apart, making wet blood smears less useful for diagnosis.
- *Thick blood smear*: stain, and look for trypanosomes. Thick blood smears are good for large population surveys.

  Trypanosomes are detectable in thick blood smears at lower levels of parasitaemia than in wet blood smears.
- Blood counts reveal anaemia, thrombocytopenia, and monocytosis. The ESR is raised.
- *Serological tests*: IgM levels are elevated in both blood and cerebrospinal fluid. Both false-positive and false-negative results are common, especially with serum IgM levels. The card agglutination trypanosomiasis test is a new test used for detecting *Tbg* infections. There is no antigen for *Tbr*. The test gives presumptive evidence of infection, but does not confirm the diagnosis.
- *Lymph node aspiration:* Look for live organisms in the aspirate. This used to be the most sensitive method of diagnosis before other newer methods were developed; it is still important for confirming

the diagnosis after a positive serological test. Aspirates should be obtained from every enlarged lymph node (this is often only possible when the lymph nodes have not yet become sclerosed and hard early in the disease). If the organisms are not found, and the diagnosis is still suspected, or the serology test is positive, continue periodic examinations using the above diagnostic techniques.

- *Other methods include*: CSF analysis, animal inoculation, and culture techniques.

Because of the serious side effects of medications, never start treatment without a confirmed diagnosis

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Differential diagnosis

Fever stage:

- malaria
- relapsing fever
- typhoid
- tuberculosis

Visceral stage:

- meningitis
- leukaemia and reticulosis
- psychosis
- hookworm anaemia
- cachexia from other cause (TB, malignancies).

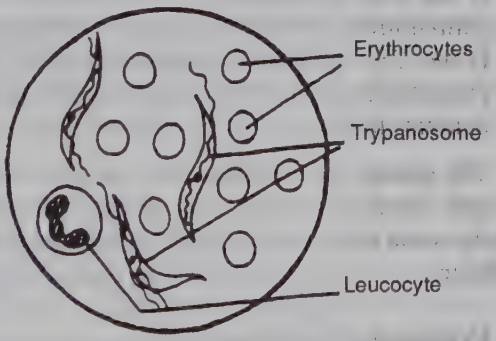

Figure 4.21 **Trypanosomes in blood smear**

5. Management of the individual patient

Patients with trypanosomiasis should be treated under strict observation in hospital because the drugs used are highly toxic, and need to be administered with care. The choice of treatment depends on whether the disease is at an early stage or advanced (cerebral) stage, and on the causative trypanosome. Cerebrospinal fluid (CSF) analysis will determine whether the CNS is involved or not, and this can be used to decide the type of treatment.

The limits of normal CSF are recognized as:

- 5 cells/cubic millilitre (lymphocytes);
- 37 mg/dl of total protein (dye-binding protein assay); or 25 mg/dl of total protein (Sicard Cantaloube method);
- Absence of trypanosomes (any method).

In *T.b. rhodesiense* infections:

- If the CSF is normal, give a course of suramin.
- If the CSF is altered, give a combined course of suramin and melarsoprol (Mel B). Melarsoprol is given intravenously as a 3.6% solution.

In *T.b. gambiense* infections:

- If the CSF is normal, give a course of suramin or a course of pentamidine, provided it has not been used locally for prophylaxis.
- If the CSF is altered, give a course of melarsoprol, preferably preceded by two or three injections of suramin or one injection of pentamidine.

The dosage for children must be calculated on the basis of body weight. Detailed information on these drugs, including indications, dosages and side effects, should be obtained from the District Medical Officer.

6. Prevention and control

The control of trypanosomiasis focuses on:

- Early detection and treatment;
- Vector control.

Bush clearing is an effective method of vector control but may harm the environment. Use of insecticides is expensive and only of temporary benefit. Baited fly traps are now being used to successfully reduce the fly populations by as much as 95% in 6 months in some communities.

The establishment of settlements will in the long run reduce the number of breeding places for the vectors of sleeping sickness. By turning bush land into agricultural land the habitats of the tsetse will be destroyed.

Chemoprophylaxis: There are no very effective drugs available to prevent the development of the disease, although pentamidine has been used.

7. Summary

Trypanosomiasis is an important disease in many parts of Africa because it retards economic progress by affecting both humans and cattle. The disease in humans is caused by two types of trypanosomes, *T.b. gambiense* and *T.b. rhodesiense*. *T.b. gambiense* causes classical sleeping sickness and occurs in west and central Africa and certain small areas in eastern Africa. *T.b. rhodesiense* causes an accelerated disease which may not present the classical picture of sleeping sickness. Tsetse flies are the vectors of the disease.

The animal form of the disease is also transmitted by tsetse flies and is caused by *T.b. brucei* which is pathogenic to cattle (Nagana).

Control consists mainly of destruction of the habitat of the vector by bush clearing, and establishing agricultural land, by fly trapping and by surveillance. Selective insecticide spraying has also been used.

PLAGUE

1. Plague is a highly infectious bacterial disease transmitted by fleas which live on rodents. It occurs in outbreaks which can be fatal in a large proportion of patients if not promptly treated. The initial cases of an outbreak are usually of the bubonic type, while subsequent cases may present as pneumonic plague.

2. Occurrence and importance

Plague was one of the major diseases recorded as causing extensive epidemics in the Middle Ages. These epidemics do not occur nowadays, but east Africa continues to be one of the few remaining foci of plague in the world. Small outbreaks have occurred in Uganda, Kenya, Tanzania, eastern Zaire and Namibia.

The disease is rare, but its importance lies in the fact that it can spread very rapidly unless the very first case is recognized early and appropriate action taken. The first cases are likely to present at a health centre. Health workers in areas where outbreaks occur should

therefore be able to recognize and quickly diagnose the disease and treat the cases promptly. The case fatality rate can be as high as 60% if adequate treatment is not given.

3. Epidemiology

Plague is caused by a Gram-negative bacillus called *Yersinia pestis* (formerly *Pasteurella pestis*). The disease is endemic in wild rodents living especially in the highlands. It is spread by fleas from the rats.

In the past, great epidemics of plague were caused by the fact that when wild rats died from the disease, their fleas looked for other rat hosts and moved to domestic rats as substitutes. When the domestic rats also died, the fleas started to bite human beings. When the first human being is infected, the disease causes bubonic plague. In the terminal stages of the disease, there is septicaemia and plague pneumonia, which can spread directly from man to man. For example, a person working in the fields is bitten by an infected flea. He may develop symptoms of bubonic plague and later develop septicaemia and pneumonic plague. By coughing infective plague bacilli he infects his relatives and suddenly many people die from an undiagnosed disease.

4. Clinical picture

There are three clinical presentations of plague:

Bubonic plague

This is the commonest form. It is characterized by swelling of the lymph glands (bubo) which appears within 24 hours of the onset of the disease. The glands most affected are those of the groin, although the armpit and other places may also be affected. The buboes vary in size, and they may be tender or non-tender. Bubonic plague may be complicated by septicaemia.

Septicaemic plague

Septicaemic plague may be due to an initial infection with highly virulent organisms or it may be a terminal complication of bubonic plague. When it is an initial infection, glandular involvement is minimal.

The illness is of sudden onset. The patient is prostrated, febrile, weak, pale and apathetic. Stupor, coma and death may follow on the

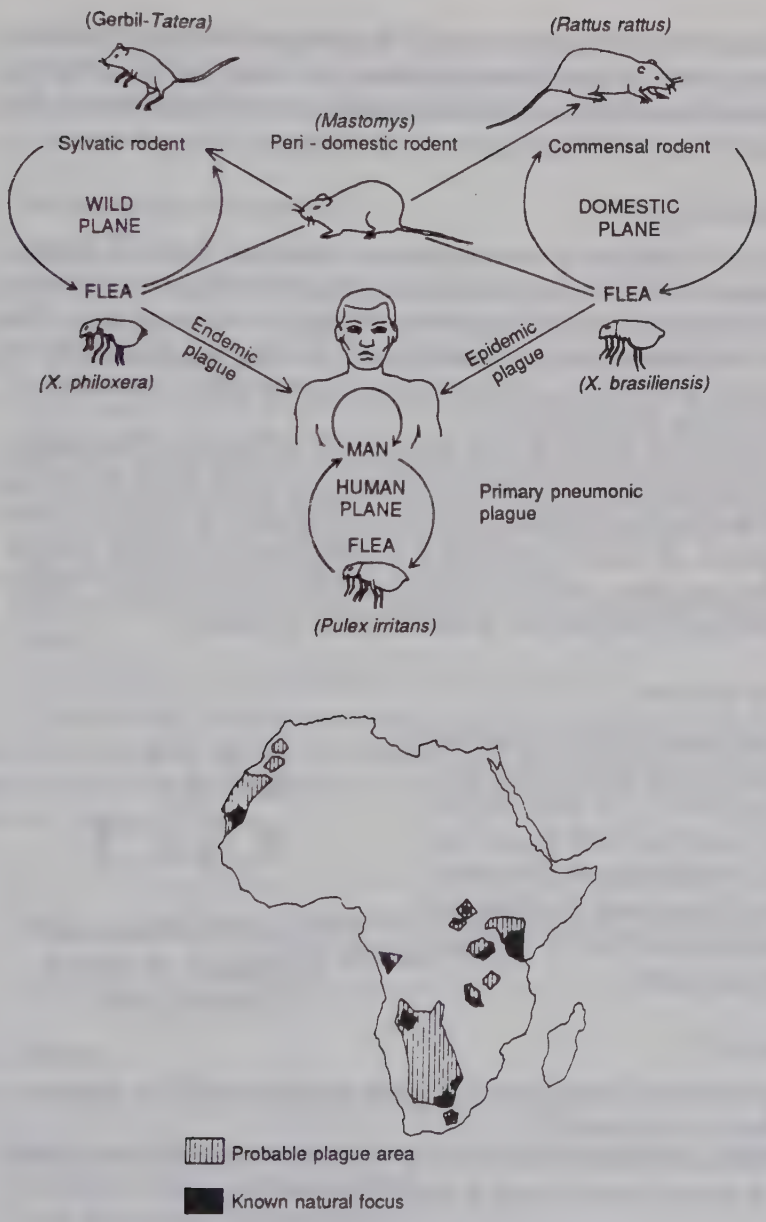

Figure 4.22 **The plague cycle and foci of the disease in Africa**

first, second or third day, or later. In the septicaemic stage the bacilli are everywhere in the organs, including the lungs. The patient may cough and spread the bacilli to his attendants who then develop the pneumonic type of plague.

Pneumonic plague

This usually follows or complicates the other two forms of plague. Attendants and visitors are at especially high risk of infection because of the numerous bacilli the patient coughs out all round him. The illness is characterized by sudden onset of rigors, malaise, intense headache, body aches, fever and severe prostration. This form of plague is rare in the tropics.

In the early stages it may be difficult to diagnose pneumonic plague, but the extreme illness of the patient in the absence of definite physical signs should raise the suspicion of this type of plague. At first the sputum is watery, but soon it becomes blood-stained. Pleural effusion may form, and death usually occurs within 1–2 days. This is the most infectious and the most fatal form of plague.

Minor plague

A few people who contract bubonic plague may not appear to be very sick. The fever comes down when the bubo bursts and starts discharging pus. The lesion can be differentiated from lymphogranuloma venereum by making a smear of the pus.



Figure 4.23 **Microscopic appearance of stained *Yersinia pestis***

Diagnosis

The presence of vast numbers of Gram-negative bacilli in sputum or pus from lymph nodes.

The bacilli are short and thick with rounded ends; the extremities stain a deeper colour giving a bipolar appearance.

5. Management of the individual patient

The bacilli are very sensitive to most antibiotics except penicillin.

Early treatment with streptomycin 1 g 12-hourly for 3 days and then 1 g daily for 7 days, or tetracycline or chloramphenicol 500 mg four times a day by mouth for 7 days are all effective. Cotrimoxazole is also effective in doses of two tablets twice a day for 7 days.

6. Prevention and control

Early diagnosis is most important. In cases of suspected plague patients should not be referred to the hospital but the DMO must be informed immediately. Plague is an internationally notifiable disease and should be reported to the World Health Organization (WHO).

- Chemoprophylaxis of all contacts must be started forthwith (tetracycline or cotrimoxazole) if there is no plague pneumonia.
- The area where the disease occurs must be quarantined (isolated from all neighbouring places); the police can help with this. Cases must be isolated and contacts quarantined for 10 days.
- Insecticides should be distributed to kill fleas.
- Encourage people to kill rats, and deal with rubbish which attracts them.
- Vaccination during an epidemic is not effective.

It is hoped that you will never see a case of plague, but remember that most probably the initial recognition of the disease must be by health staff working in small units, so your quick action may save the lives of many people.

Early recognition of plague followed by correct action is a matter of life or death

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7. Action

If you suspect a case of plague, do the following:

- Notify your DMO; ask him to come immediately; plan to inform the public on their role in control;
- Treat the patient with antibiotics;
- Give prophylaxis to all contacts (including yourself);
- Request supplies of insecticide and rat poison (e.g., Warfarin).

8. Summary

Plague is a disease of rodents such as rats. It is transmitted by the rat flea. When man becomes infected, the first case(s) present as bubonic plague. Septicaemia may result in pneumonic plague. Pneumonic plague spreads very rapidly. Early recognition and notification to the authorities is vital.

SCHISTOSOMIASIS

1. Schistosomiasis is a chronic disease caused by trematodes of the genus *Schistosoma* which infect the large bowel or the urinary bladder, depending on the species. The clinical disease is caused by the body's reaction to the presence of the eggs of these worms, and will therefore depend on the location of the adult worms.

Synonym: Bilharzia.

2. Occurrence and importance

Schistosomiasis is the second most frequent disease, after malaria, in some countries. In Egypt, 37% or more of the population may be infected. Infection, however, even though it generally causes symptoms

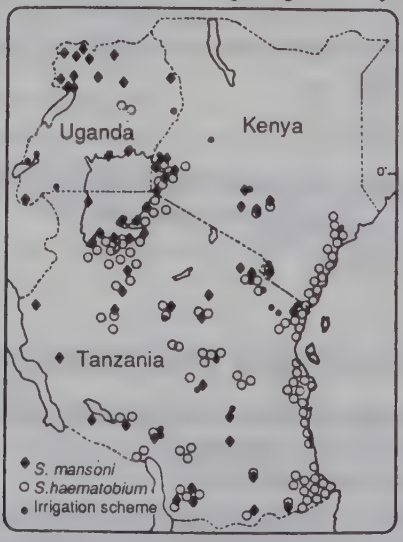


Figure 4.24 **Distribution of schistosomiasis in east Africa**

(e.g., haematuria) does not necessarily lead to disabling clinical disease. This depends on the worm load, duration of infection, the immune state of the patient and other concurrent diseases. Anaemia, to which schistosomiasis may contribute, certainly causes some morbidity in children, and lack of productivity in adults. Although schistosomiasis can result in killing conditions, such as portal hypertension, uraemia, and heart failure, it is not known how often these occur. There is, therefore, no general agreement on the seriousness of the ill effects of schistosomiasis on the general health of the people.

The incidence of schistosomiasis is related to water use. Development of water projects for irrigation or electricity provides the habitat for the snail vectors. Such projects may cause epidemics of schistosomiasis. The rise of socio-economic levels with improved agricultural techniques has often been accompanied by an increased incidence of schistosomiasis.

3. Epidemiology

Schistosomiasis is caused by the tissue reaction against the eggs of the schistosome worm. Depending on the species, the eggs are excreted in urine or faeces. The main schistosomes which infect man in Africa are *Schistosoma mansoni* and *Schistosoma haematobium.*

When the eggs reach water, miracidia hatch out. Miracidia are free-swimming larvae which have to reach a snail host within 24 hours or die. In the snail, the miracidia develop and multiply into many cercariae. These are the infective agents of schistosomiasis. They are shed from the snails in 4–7 weeks. They can only live up to 48 hours unless they infect man. Man becomes infected when he enters cercariae-infested water. This can occur while bathing, playing, cultivating or fishing. The cercariae penetrate the skin and enter the bloodstream from where they are carried to the liver or the bladder where they develop into adult worms.

Two types of schistosomiasis are found in Africa, each with a different clinical picture. Each type of schistosomiasis has a different distribution, depending on the type of snail vector and snail-host behaviour.

Schistosoma haematobium lives in the venous plexus of the urinary bladder. The eggs are excreted in the urine. The vector snails belong to the *Bulinus* genus which lives in temporary water bodies like ponds,

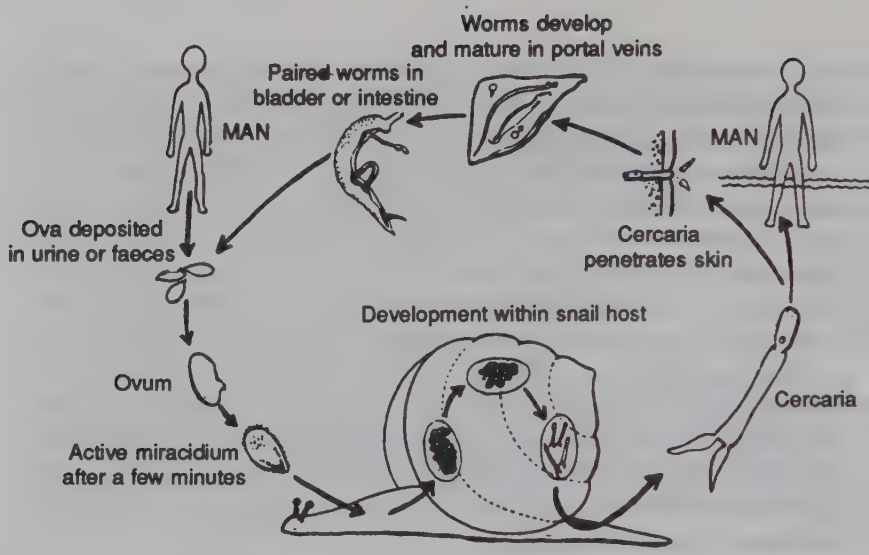


Figure 4.25 **Transmission of schistosomiasis**

dams, and paddy fields. It adapts to adverse conditions during the dry season by inactivity (aestivation).

Schistosoma mansoni lives in the mesenteric plexus of the large intestine. The eggs are excreted with faeces. The vector snails belong to the genus *Biomphalaria* which prefers permanent water like streams, irrigation schemes, and lakes.

The differences between *S. haematobium* and *S. mansoni* are shown in Table 4.8.

4. Clinical picture

Schistosomiasis as a disease has several stages: (a) invasion; (b) maturation; (c) established infection; and (d) late stage.

(a) Invasion

In this stage the cercariae penetrate the skin. This causes cercarial dermatitis with itching papules and local oedema. It is more severe when caused by bird and animal schistosomes (swimmers' itch). Very often, however, it is not noticed, or not reported.

The cercariae enter the circulation, and reach the liver via the right side of the heart and the lungs by an unidentified mechanism.

Table 4.8 **Distinguishing between *S. haematobium* and *S. mansoni***

	S. haematobium	*S. mansoni*
Adult worm	Venous plexus of bladder	Mesenteric veins of bowel
Egg (microscopic appearance)	 Terminal spine Found in urine	 Lateral spine Found in faeces
Vector	*Bulinus* (actual size)  Temporary water bodies, e.g., paddy fields, ponds	*Biomphalaria* (actual size)  Permanent water bodies, e.g., lakes, rivers, dams, irrigation schemes
Early disease	Terminal haematuria	Bloody diarrhoea "Mansoni dysentery"
Late disease	Bladder calcification Carcinoma of bladder Hydronephrosis Kidney failure Pulmonary hypertension leading to CCF	Bowel fibrosis Portal hypertension with hypersplenism Oesophageal varices CCF
Treatment	Metrifonate	Oxamniquine or praziquantal if funds available

(b) Maturation

The schistosomes mature in the liver. This stage is associated with fever, eosinophilia, abdominal pain, and transient generalized urticaria. It is known as the Katayama syndrome. After maturation, the adult worms descend the portal vein. *S. mansoni* migrates to the mesenteric veins in the intestinal wall, and *S. haematobium* finds its way to the bladder venous plexus. This stage often passes largely unnoticed.

(c) Established infection

This is the stage of egg production. To continue the life cycle, the eggs have to reach the lumen of bladder or bowel. Eggs are produced by the female worms after moving as far down the small veins of the bowel or bladder wall as they can go. Some eggs do not penetrate the tissues and are carried with the bloodstream to the liver and lungs. Other eggs penetrate into the tissues with the help of their spines, but fail to reach the lumen of the bowel or bladder. The eggs which do not reach the lumen provoke an inflammatory reaction and the formation of granulomas. It is this inflammatory reaction that causes the early signs and symptoms of schistosomiasis: signs of colitis with bloody diarrhoea and cramps in *S. mansoni* infection; terminal haematuria and dysuria in *S. haematobium* infection.

(d) Late stage

This is the stage of fibrosis and calcification which occurs where there are many eggs in the tissues. Around the bladder this may result in:

- Obstruction to and dilatation of the ureters (hydro-ureter) and kidney (hydronephrosis), possibly leading to kidney failure;
- Pyelonephritis can also follow;
- Calcification of the bladder which shows on X-ray or ultrasound investigation;
- Cancer of the bladder has been seen in Egypt and Mozambique.

In the liver, the fibrosis is periportal, resulting in portal hypertension. Portal hypertension leads to hypersplenism and anaemia, oesophageal varices and often massive bleeding.

In the lungs the fibrosis results in pulmonary hypertension and so leads to congestive cardiac failure (CCF).

The diagnosis of schistosomiasis is confirmed by finding eggs in stools or urine. If these are repeatedly negative a biopsy can be done (rectal snip, bladder biopsy).

Concentration techniques can be used to improve the detection rate of the eggs in stool, as plain stool examination can sometimes be misleading.

5. Management of the individual patient

Drugs in common use are shown in Table 4.9 (page 146).

6. Prevention and control

Infection of snails may theoretically be decreased by using pit latrines. However, only a small amount of contamination of the water is needed to keep the infection going. So pit latrines are more important in preventing other diseases than in control of schistosomiasis.

Control of snails

The control of snails is one method for reducing transmission of schistosomiasis, but snail control has proved to be very difficult. Schistosomiasis is increasing due to new agricultural methods. For example, irrigation schemes form an ideal breeding place for snails and very often the introduction of irrigation methods is followed by an epidemic of schistosomiasis.

Chemicals (molluscicides)

Molluscicides are expensive, and need to be applied repeatedly under close supervision to be of use in the control of vector populations. These chemicals are often toxic to humans and also kill fish. They should be applied during seasons when snail populations tend to peak so as to ensure maximum kill.

The most widely used molluscicides are:

Copper sulphate: is safe to handle; it kills both snails and their eggs, and is not as toxic to fish as other compounds. It is most effective at low pH. It is corrosive to equipment, and is absorbed by soil and organic matter.

Table 4.9 Drugs used in the treatment of schistosomiasis

Possible drugs in use	Dose	Route	Contra-indications	Side effects	Remarks
Metrifonate (Bilarcil)	7.5 mg/kg once every 2–3 weeks Total 3 doses	Oral	None	None if correct dose	For *S. haematobium* Not effective for *S. mansoni* Cheap
Oxamniquine (Vansil)	15-30 mg/kg for 1–2 days 20 mg/kg for children	Oral	Epilepsy Not effective for *S. haematobium*	Occasional or psychosis Dizziness	For *S. mansoni* Drowsiness
Praziquantal (Biltricide)	40 mg/kg one dose	Oral	None (Drug of choice if liver disease)	Mild giddiness	Equally effective for *S. mansoni* and *S. haematobium* Expensive
Niridazole (Ambilhar)	25 mg/kg for 7 days	Oral	Severe liver disease History of epilepsy or psychosis	Nausea Vomiting Occasional convulsions and psychosis	Seldom used now *S. haematobium* and *S. mansoni*

Notes

Drugs previously used include sodium antimony tartrate by injection, stibocaptate by injection, lucanthone oral and hycanthone injection. These all have side effects, some potentially serious.

S. mansoni, drugs of choice: oxamniquine, and praziquantal if there is liver damage.

S. haematobium, drug of choice: metrifonate (praziquantal also effective but costly).

Praziquantal is easy to administer as a single dose. It has no absolute contraindications, but it is expensive.

Niclosamide (Bayluscide): is safe to handle and use; it kills both snails and eggs. It is the cheapest chemical per volume of water treated, but tends to clog in equipment used. Effectiveness is slowly reduced by sunlight, absorption by mud, or high concentration of minerals (hard water).

In some areas, plant molluscicides are being tried, e.g., the fruit of Endod (*Phytolacca dodecandra*) in Ethiopia.

Environmental sanitation

Prevention of snail breeding may be more important than killing of snails. This requires radical alteration of snail habitats by:

- Draining or filling of water bodies;
- Clearing of vegetation in water bodies to deprive snails of food and resting places;
- Irrigation schemes: intermittent irrigation will result in sudden changes of water level and wave action;
- Flooding (harmful for all snails);
- Changing speed of water flow in channels and rivers;
- Protecting ponds;
- Straightening and deepening margins of water bodies.

When irrigation schemes are planned, these should be organized in such a way that at least no increase in snail population will occur. Sanitation engineers or public health specialists should be involved.

The supply of piped water to the people will not directly affect snail breeding, but is most effective in reducing contact between man and snail and so will greatly reduce transmission of schistosomiasis.

Snail control through environmental sanitation and or molluscicides needs maintenance over a long period (at least 10 years). It is impossible to use either of these methods generally in most of Africa because of prevailing economic conditions.

Prevention of infection of human beings

The most effective way to protect human beings from schistosomiasis is to educate them about the disease and give them an ample supply of safe water. Water from other sources can be made safe by keeping it in a container for 48 hours using the three-pot system. Within this period the cercariae will have died.

Treatment of individuals and mass treatment

Now that effective treatment is available, both morbidity and the level of infection in a community can be rapidly reduced. Treatment can be targeted at children in the 5–14 year age range because they have the highest prevalence of schistosomiasis. Where possible in a community, quantitative microscopic techniques should be used in epidemiological surveys to determine the level of endemicity. This information is then conveyed to the community at schools and public meetings, after which decisions are made about treatment and its costs. If endemicity is above 66%, then mass treatment of all children is indicated, if affordable. If endemicity is below 66%, then individuals should be examined and treated.

Treatment of schistosomiasis requires community participation in terms of organization, cost-sharing, and deciding on any further control measures (e.g., water and sanitation and health education). Mass treatment and treatment of all heavy worm loads is an effective way of reducing endemicity rapidly. Endemicity will then take several years to rise again, during which time other measures can be strengthened.

Treatment of all heavy infections will considerably reduce the burden of morbidity. It will also reduce the effects of schistosomiasis on the genito-urinary tract and the liver of individual patients and improve the health of the community in general.

7. Action

When there is a high prevalence of schistosomiasis, treat all those with symptoms, but retain some of your limited resources for the following control measures:

- Give frequent health education on how the disease is spread;
- Stress the importance of a safe water supply;
- Emphasize the need for building pit latrines.

8. Summary

Schistosomiasis is a common disease in Africa. Its public health importance is often local, at district or even village level. A cheap, safe, and effective drug is now available for *S. haematobium*, but treatment of *S. mansoni* is still expensive. Therefore, mass treatment

will either require a strong political commitment or cost sharing with community participation.

DRACUNCULOSIS

1. This is a disease due to a worm called *Dracunculus medinensis*. It occurs in areas with very poor water supply.

Synonyms: Guinea worm, dracontiasis.

2. Occurrence and importance

It occurs in Uganda and Sudan and in west Africa (Ghana, Cameroon and Nigeria especially). It is often seen in localized tropical areas with poor water supply. For example, in Uganda it is mostly found in villages in the north of the country.

3. Epidemiology

Man becomes infected by drinking water containing *Cyclops* which are infected with *Dracunculus medinensis*.

The *Dracunculus medinensis* larvae get into the connective tissue and slowly develop. The whole cycle takes about 1 year. After mating, the male dies and the female, now about 1 metre in length, moves to the surface of the body, and discharges new larvae, usually through an ulcer on the leg, which enter the water and infect the cyclops again. After 2–3 weeks, the larvae which have developed in the cyclops are infective. The cycle in the *Cyclops* requires a temperature of about 25°C.

In some areas, transmission may be greater in the dry season when many people visit a few pools, while in other areas it may be greatest at the start of the rains.

4. Clinical picture

An itching and painful blister develops at the site where the adult female worm emerges, thus making an ulcer. Ulcers can occur anywhere, but they are most common in areas which are most likely to come into contact with water, such as the feet, legs and the genitals. The thread-like worm can be seen in the ulcer. After discharging

larvae for 2–3 weeks the female worm dies and is extruded or absorbed.

Complications include infection, with cellulitis, arthritis, and possibly tetanus. The worm often erupts at critical times, especially during the rains when agricutural work is at its height. The patient usually experiences pain and inability to work. This can have serious adverse economic effects in heavily infected areas.

5. Management of the individual patient

Patients usually try to tease the worm out by winding the protruding end on a small stick daily until the whole worm is extracted, but the worm can break, causing a severe reaction. Drugs such as niridazole, metronidazole and thiabendazole have been tried, but probably only help to reduce the inflammation.

6. Prevention and control

This is an example of a condition that can be eradicated if there is stability and community education and involvement. Filtering and boiling water removes or kills the *Cyclops*. Insecticides can be used to kill the *Cyclops* in infected pools. In Uganda, one of the more effective measures was the drilling of boreholes and the provision of pumps so that people did not have to use small pools. However, in countries where there is war, civil unrest and population movements, the condition increases in frequency and may even produce epidemics because there are no organized control programmes.

7. Action

- Filter drinking water through a cloth or boil the water.
- Improve water supplies; this is by far the most important measure.
- Educate the population.
- Stop people with ulcers from stepping in water sources.

8. Summary

Dracunculosis is caused by a tropical worm found in areas with insanitary water supplies containing *Cyclops*, the vector of the disease. It is a condition of economic importance as it can cripple many people at the time of intensive agricultural work. It can be eradicated by health education, filtering water or providing protected water sources.

LEISHMANIASIS

This is an infection caused by parasites of the genus *Leishmania*. Its zoonotic hosts are mainly canines and rodents. Man acquires the infection when he interrupts the natural cycle. Sandflies (*Phlebotomus* spp.) are the main vectors which transmit the disease between animals, from animals to man, and from man to man. In Africa, there are two forms of leishmaniasis: visceral and cutaneous.

VISCERAL LEISHMANIASIS

Synonym: kala azar.

1. This is a severe chronic infection of the reticulo-endothelial system which presents with fever, chills, anaemia, leucopenia, splenomegaly, weight loss and general debility.

2. Occurrence and importance

Kala azar is a disease found in many parts of the world, but in Africa it occurs on the Mediterranean coast and in a belt across the arid and semi-arid areas from Lake Chad in the west to Somalia in the east, excluding the highlands of Ethiopia. There are two main foci of the disease — the Sudan and eastern Africa.

Sporadic cases occur in Somalia, along the Juba river, in Karamoja in Uganda and in the Rift Valley area of Kenya. The main endemic area is in eastern Kenya along the Tana river and in Machakos District along the Athi river.

In the Sudan the disease is found in the flood plains of the Nile and its tributaries, and in the early 1990s caused a large epidemic and thousands of deaths.

Without treatment the disease is usually fatal because of intercurrent infections and general wasting.

3. Epidemiology

Visceral leishmaniasis is a zoonosis which circulates among wild and peri-domestic animals and sandflies, and only occasionally affects man. The animals most involved are canines, such as jackals and dogs, and rodents, such as rats.

The parasite which causes visceral leishmaniasis is called *L. donovani* and it is spread by sandflies of the species *Phlebotomus orientalis* in the Sudan and *P. martini* in Kenya. In the Sudan the rat is the zoonotic host, while in Kenya dogs, rodents and lizards may form the reservoir of the disease.

The disease mainly affects male teenagers, and may affect special groups such as soldiers and hunters who enter endemic areas.

4. Clinical picture

The incubation period of visceral leishmaniasis is 4–10 months or longer. Often before the definitive symptoms begin, a papular skin lesion, known as a leishmanioma, appears. This may precede the onset of the disease by 4–6 months.

The disease may set in gradually or suddenly. If the onset is sudden, it presents with intermittent or remittent fever which lasts for 2–6 weeks before becoming persistent and low grade. If the onset is gradual, it starts with discomfort in the left hypochondrium as the spleen enlarges. During this time the patient may present with acute intercurrent infections such as dysentery or pneumonia. The disease then runs a chronic course lasting 1–2 years and is associated with chronic fever, hepatosplenomegaly, lymphadenopathy, anaemia and emaciation. In 96% of cases, if not treated, the patient dies of intercurrent infection.

Diagnosis

Diagnosis is by finding the parasites in stained smears from splenic puncture or by immunological tests such as the formal gel test, leishmanin test and complement fixation test.

5. Management of the individual patient

The drugs used for treating visceral leishmaniasis are rather toxic and so should only be administered in hospital.

The parasites are sensitive to several drugs, but are beginning to show resistance to antimonial drugs forcing authorities to use pentamidine which is more expensive and has more side effects.

The drug of choice is sodium stibogluconate intravenously or intramuscularly in doses of 0.1–0.2 ml/kg (1 ml = 100 mg) daily for 6–30 days. Alternately, pentamidine isothianate may be given

intramuscularly in doses of 0.1 ml/kg (1 ml = 40 mg) three times weekly until ten doses have been administered.

Amphotericin B is reserved for cases resistant to other drugs and it is given by slow intravenous infusion in 5% dextrose in doses of 1 mg/kg on alternate days until a total of 2 g has been given.

Exacerbation of kala azar by HIV infection and AIDS is now causing some concern. The two diseases also look alike.

Supportive treatment

- Ensure bed rest;
- Good nursing;
- Adequate nutrition;
- Correct anaemia;
- Treat intercurrent infections.

6. Prevention and control

Although sandflies are susceptible to insecticides, control of the vector is difficult because the flies breed away from homes. However, in Baringo District of Kenya some measure of control has been achieved with the use of fine insecticide-treated curtains in homes. Destruction of infected dogs and rodents will control the reservoir.

Early diagnosis and treatment can also control the spread of the disease from man to man.

7. Action

In endemic areas, exclude kala azar in patients who present with wasting, fever, anaemia, and enlarged spleen and liver. Transfer to hospital for definitive diagnosis and treatment.

Educate the population about the disease and the need for early treatment.

8. Summary

Visceral leishmaniasis is a serious disease characterized by chronic splenomegaly, wasting, fever and anaemia and is transmitted from wild animals and dogs to man by the bites of sandflies which live within certain moist habitats in dry regions.

The disease can occur in epidemics. Early diagnosis and treatment in hospital is required.

CUTANEOUS LEISHMANIASIS

Synonym: Oriental sore.

1. This is a zoonotic infection which is characterized by a single or several chronic cutaneous sores which usually heal spontaneously.

2. Occurrence and importance

The disease affects parts of the Sahelian region of west Africa where it is caused by *L. tropica major*. Human cases are, however, uncommon and sporadic. The zoonotic reservoirs are desert gerbils and dogs and the vectors are *Phlebotomus papatsi* and *P. sergenti*.

In eastern Africa, the Ethiopian and Kenyan highlands are affected by the disease caused by *L. aethiopia*. The zoonotic reservoir is the rock hyrax and the vectors are the high-altitude sandflies *P. longipes* and *P. pedifer*.

3. Epidemiology

Cutaneous leishmaniasis is a rare disease of which there are three main forms, caused by different species and strains of leishmania. The rural form of the disease affects man in uninhabited areas and in villages situated on the edge of the desert. In these cases the disease is caused by *L. tropica* major, the desert gerbil is the main reservoir and it is transmitted by *P. papatsi*.

The urban form of cutaneous leishmaniasis is caused by *L. tropica* minor and is an infection of man and dogs in big cities and towns. It is transmitted by *P. sergenti*.

A third form of the disease is that found in the Ethiopian and Kenyan highlands. It is an infection of man and the rock hyrax caused by *L. aethiopia*. The vector *P. longipes* bites people in their houses at night.

4. Clinical picture

The incubation period is 2–8 weeks. The disease starts as a small itchy papule, usually on the face, which expands and grows over several weeks to form a single indolent ulcer or multiple ulcers. The ulcer(s) may resemble skin tuberculosis of the face (lupus vulgaris). There

may be local lymphadenopathy. Rarely, when the lesion infiltrates the skin diffusely, it may resemble lepromatous leprosy.

Spontaneous healing starts 2–12 months afterwards. The disease does not spread to involve the viscera.

5. Management of the individual patient

- Usually not necessary as spontaneous healing occurs;
- If the lesions are diffuse or extensive, use pentavalent antimonials (e.g., sodium stibogluconate) as in the treatment of visceral leishmaniasis. The response is, however, not as satisfactory;
- Surgical treatment may be considered in some cases.

6. Prevention and control

As for visceral leishmaniasis.

7. Action

- Exclude TB and leprosy in patients with cutaneous leishmaniasis.
- Refer to hospital for diagnosis and treatment.
- Institute control measures.

8. Summary

Cutaneous leishmaniasis is a zoonosis caused by leishmania parasites and spread from zoonotic hosts to man by sandflies. It is characterized by single or multiple chronic sores on the face which usually heal spontaneously.

Chapter five

Diseases Caused by Faecal Contamination

INTRODUCTION

The causative organisms of the diseases in this group are excreted in the stools of infected persons (or, less commonly, animals).

The portal of entry for these diseases is the mouth. Therefore, the causative organisms have to pass through the environment from the faeces of an infected person to the gastro-intestinal tract of a susceptible person. This is known as the faeco-oral transmission route.

Faeco-oral transmission occurs mostly through inapparent faecal contamination of food, water, and hands. Very small amounts of faeces can carry enough organisms to establish infection. Sparkling clear water may be dangerously polluted. Contaminated food may smell, look, and taste normal and yet harbour disease. Apparently clean hands may carry and transmit enough micro-organisms to spread disease. As shown in Figure 5.1, food plays a central role because it can be directly or indirectly contaminated via polluted water, dirty hands, contaminated soil, flies, and animals or animal products.

Water can be polluted directly by faeces, or faecal material may be washed in from polluted soil from the river banks.

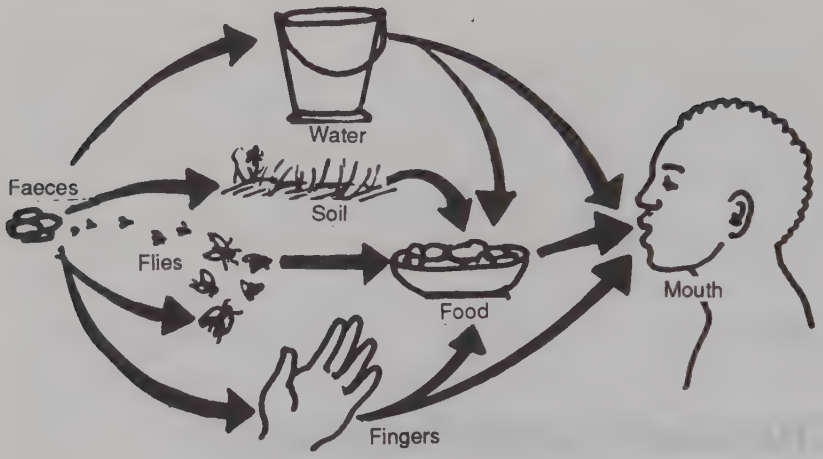


Figure 5.1 **The faeco-oral transmission route**

Hands are contaminated after defecation or by touching contaminated objects.

The house fly is very likely to carry faecal material because of its habits of starting a meal on faeces and finishing off on human food. The fly can transfer the germs from faeces to food by carrying them on its body, by vomiting on solid food in order to liquefy the food, and by defecating on food. The faeces and vomitus of the fly may contain viable pathogenic organisms from human faeces.

There are many diseases which are transmissible through faeces, but not all of them are strictly faeco-oral in transmission. For instance, the polio and infectious hepatitis A viruses are detectable in the faeces of infected persons by using special techniques, but they are now also known to be transmitted by other routes besides the faeco-oral route.

The eggs of the intestinal worms and flukes (e.g., *Schistosoma mansoni*) are excreted in faeces, but the portal of entry for infestation with the worms or flukes is not always the mouth. Because of their related life cycles, the intestinal worms are dealt with in a separate chapter. Schistosomiasis has already been considered as a vector-borne disease.

Food poisoning is not always due to faecal contamination but certainly belongs to the food-borne group of diseases and is therefore included in this chapter. Many types of salmonella other than *S. typhi* come from animals and can cause diarrhoea in man. Campylobacter from poultry can also be transmitted to man. In these diseases, which

Table 5.1 Diseases in which the gastro-intestinal tract is the portal of entry

Viral	Bacterial	Protozoal	Toxins	Worms
Polio Hepatitis Viral diarrhoeas (entero-viruses e.g. rota virus)	Typhoid and paratyphoid Other salmonelloses Cholera Bacillary dysentery* Anthrax Specific types of *E. coli*	Amoebiasis Giardiasis	Staphylo-coccal food poisoning Botulism	Ascariasis Enterobiasis Trichuriasis Taeniasis Hydatidosis
Unspecified diarrhoeal diseases				

* Dysentery means infective diarrhoea with blood, and it is a clinical syndrome with different causes: *Shigella* spp. causes bacillary dysentery; *Entamoeba histolytica* causes amoebic dysentery; and *Schistosoma mansoni* causes schistosomal dysentery.

are also dealt with in a separate chapter, infection is acquired by eating infected meat or eggs, or from contaminated kitchen utensils and surfaces.

Some of these diseases are shown in Table 5.1.

The mode of transmission of some of the viral gastro-intestinal infections is not firmly established. For some other infections, the number of organisms ingested determines whether infection occurs or not. These numbers have been calculated for some of these germs, as shown in Table 5.2.

Table 5.2 The number of germs necessary to cause disease

Disease	Organism	Number required	Main route of transmission
Cholera	*Vibrio cholerae*	1,000,000,000	Water
Typhoid	*Salmonella typhi*	100,000	Water
Paratyphoid	*Salmonella paratyphi*	1,000	Food/Water
Dysentery	*Shigella* spp.	100	Food/Water

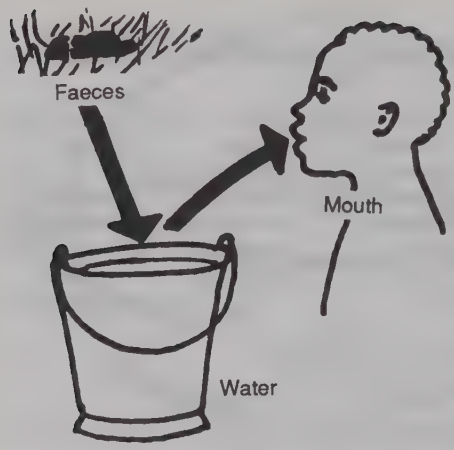


Figure 5.2 **Main way of transmission of typhoid and cholera**

The explanation for the marked differences in the infectivity of these organisms is their susceptibility to lysis by gastric acid in the stomach. Gastric acid is capable of killing many organisms by lysis, thus preventing infection in some cases.

This natural defence mechanism is most effective against organisms such as cholera vibrios and typhoid bacilli (see Table 5.2). As a result, very large doses of vibrios or *Salmonella typhi* have to be ingested in order to cause disease. This makes accidental infection with typhoid or cholera by contact or contamination of food by flies very unlikely. Thus, both diseases are almost exclusively water and food-borne.

When only a very small number of organisms is required to cause infection, as in bacillary or amoebic dysentery, this is very easily done accidentally by the fingers, or by contamination of food and eating utensils by flies. Indeed, many dysentery cases are usually found during the season when flies are plentiful. Of course, infection through contaminated water occurs as well. Contamination of fingers and eating utensils is most likely to occur when water for hand-washing and cleaning is in short supply.

Diarrhoeal diseases occur more often during the dry season. These diseases are associated more with the inadequate supply of water than the contamination of water, and will usually disappear when the amount of available water is increased. It is the quantity of water rather than its quality which appears to be important in relation to diarrhoeal disease transmission.

Natural Defences

1. Gastric acid, as mentioned above.
2. *Normal intestinal bacterial flora.* These bacteria form an important protective barrier against pathogenic bacteria. As long as they live and multiply in the bowel, pathogenic organisms have less chance of thriving. This is called bacterial antagonism. One way to predispose animals to bowel infection is to treat them with broad-spectrum antibiotics, which kill their intestinal flora.
3. *Normal bowel motility.* This acts as a cleansing mechanism. Diarrhoea is usually accompanied by increased bowel motility, and it can be regarded as a normal body response for flushing out harmful substances from the body.

   Thus, it should not be surprising that wilful suppression of bowel motility may be harmful. Studies have shown that treatment of shigellosis patients with opiates (or Lomotil), even when antibiotics are given, will delay recovery. Stool solidifiers do not decrease the amount of water lost in diarrhoea: they only make the stools look more solid. These drugs are not as harmful as drugs which cause bowel paralysis, but there is a danger that they may be given instead of proper treatment, such as oral rehydration.

Death from diarrhoeal disease is largely due to dehydration and disturbances of electrolyte balance. The bar diagram in Figure 5.3, from hospital data in Tanzania, shows 1,400 deaths caused by gastro-enteritis alone in 1 year and a total of 200 other deaths from dysentery and food poisoning. Typhoid fever and hepatitis did not feature very much, with only 160 deaths.

Prevention and early treatment of dehydration and electrolyte imbalance would have avoided most of these deaths.

Other facts

From Figure 5.3, page 162, it can be seen that acute gastro-enteritis was the second commonest cause of admission to hospital, second only to pneumonia. It was followed by malaria and measles.

Acute diarrhoea and vomiting, or gastro-enteritis, is the most frequent form of diarrhoea. In the data shown in Figure 5.3, it accounted for eight times more hospital admissions than bacillary dysentery. (Note that these figures show hospital data only.)

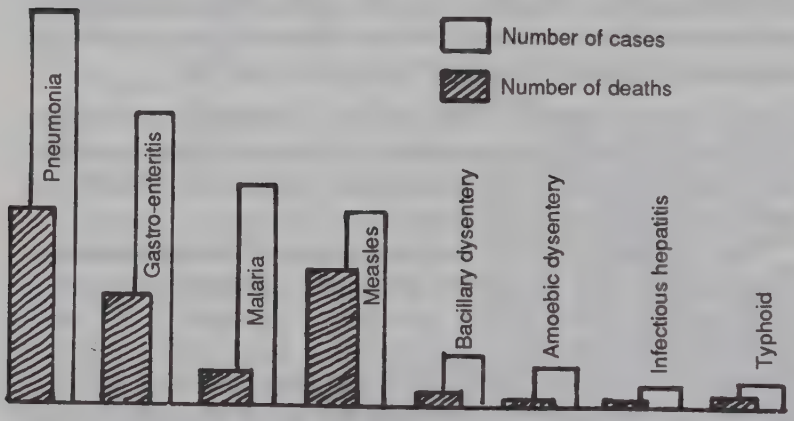


Figure 5.3 **Common causes of hospital admission and deaths**

Rehydration is the first priority

Treatment

In mild cases oral rehydration with home-made fluids is best. If available, ORS solution is ideal for preventing or treating mild forms of dehydration.

There are several ways of making emergency rehydration solutions at home. Mothers have to be very carefully taught how to make these solutions by practical "hands-on" demonstrations. The solution should be tasted, to make sure it is not too salty, before giving it to the baby. Too much salt and the wrong amount of water will produce a fluid that can be dangerous. Because it is difficult to prepare these solutions,



Figure 5.4 Preparation of home-made oral rehydration solution: 500 ml of boiled water (one beer bottle), two thumb-and-two-finger pinches of salt, one four-finger handful of sugar and some fruit juice.

mothers who are not familiar with their preparation should be advised to give plenty of any readily available fluids, such as milk, tea, coconut water, thin cereal gruel, rice water etc., at the first sign of diarrhoea. They must, of course, continue breastfeeding, and also give other foods if the baby has already started on them.

They must give the child as much rehydration fluid as possible, and continue even when the child is vomiting. If the diarrhoea and vomiting do not stop soon, or the dehydration is more severe, the child should be taken to a health unit immediately for rehydration. When the dehydration is severe, blood circulation is poor and fluids given subcutaneously or intraperitoneally are not absorbed rapidly. Therefore, intravenous rehydration is best for severe forms of diarrhoea and dehydration.

PREVENTION

Prevention depends on breaking through the faeco-oral transmission cycle.

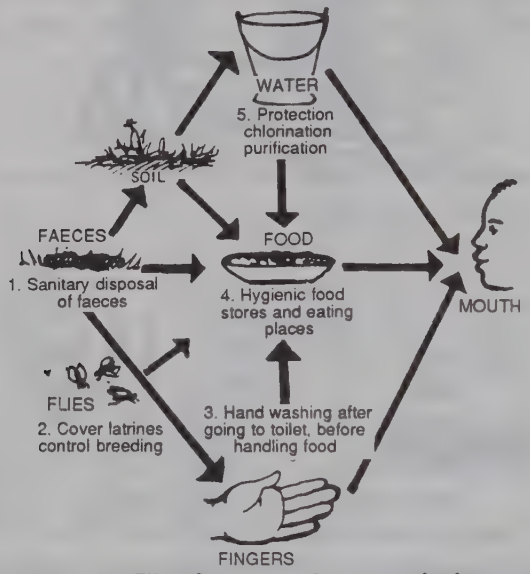


Figure 5.5 The faeco-oral transmission cycle

1. Faeces

Control of diarrhoeal diseases (including dysentery) is only possible when the methods of stool disposal are improved (use of pit latrines in rural areas). Hand-washing facilities should be provided in toilets.

2. Fingers

Provide hand-washing facilities in toilets. Wash hands after going to the toilet, and before cooking or eating.

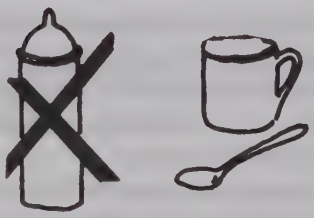
3. Flies

Control flies by proper refuse and faeces disposal. This can be done by:

- Ventilated and improved pit latrines;
- Screening kitchens and food stores;
- Storing left-over food where flies cannot reach it;
- Spraying with insecticides.

4. Food

Food should always be properly cooked. Raw vegetables and fresh fruits without intact skins are unsafe. Green salad leaves can be made safe by immersion for 15 minutes in sodium hypochlorite solution 1% (Milton). Fresh intact fruits should be washed and then peeled at the time of eating.* Milk should be boiled or pasteurized. Eating utensils should be properly cleaned and dried after use. Public eating places should be inspected to ensure that the processing, preparation and serving of food is done in a hygienic manner.



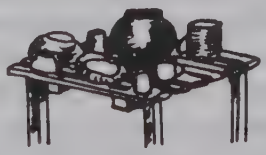
Feed with a cup and spoon



Wash hands and utensils

5. Water

The protection, purification, and chlorination of public water supplies is very important. Water from wells can be made safe with chloride of lime. Water from deep wells is usually uncontaminated if the well-head is protected and if there are no pit latrines nearby. Drinking water from other sources should be boiled when there is an outbreak of any water-borne disease.



Dry in the sun

Figure 5.6 **Long-term control of diarrhoeal diseases**

Piped water near each house would contribute greatly to general and personal cleanliness, and diminish the risk of infection from contaminated water.

*Green salad leaves can be made safe by immersion for 15 minutes in sodium hypochlorite solution 1% (Milton/Jik). Fresh intact fruits should be washed and then peeled at the time of eating.

6. Health education

- Educate people about the dangers of bottle-feeding, and encourage prolonged breastfeeding.
- Explain sources of infectious diarrhoea, encourage the use of latrines (also for children); explain importance of hand-washing, and cleaning utensils.
- Demonstrate prevention of dehydration by home-made sugar/salt solutions, and other suitable fluids.
- Encourage antenatal attendance, and immunization of infants.
- Educate mothers about growth monitoring and preparation of good weaning foods and extra meals for a child after any illness in order to improve nutrition and "catch-up growth".

CHECK-POINTS FOR SOURCES OF WATER-BORNE DISEASES

1. Primary school

- Drinking water sources: how and where is the water stored?
- Latrines: sufficient for number of children; cleanliness, etc.
- Facilities for washing hands.
- If the school provides food, check on the cleanliness of the cooks, kitchen, equipment and utensils; also check the place where children eat food brought from home.

2. Kiosks and food shops

- Source of water: possible contamination, and whether water is stored in clean vessels.
- Preparation process: cleanliness of equipment, hygiene of the process and disposal of waste materials.
- Staff: personal hygiene, septic sores.
- Consumption: care of the drinking cups, etc.
- Check for rats.

3. Hotels

- Food; water source; storage of water. How is the food stored and for how long? Rats and mice around?

- Food preparation: kitchen and cleanliness of utensils. Where is waste food disposed of?
- Staff personal hygiene, septic sores.
- Customer's facilities: latrine, washing facilities, including the cleanliness of towels.

4. Private homes

- *Water supply:* from what distance must the water be collected? Is the supply enough throughout the year? Is water for animals drawn from a separate source? Is there a slab for washing clothes?
- *Water collection:* private or common buckets? Who takes care of the cleanliness of the bucket? Pump? Tap?
- *Water storage in the house*: tins or pots; covered and cleansed regularly; emptied before refilling; protected against animals; advise the family on the three-pot system, boiling of drinking water.
- *Latrines:* each private home should have a latrine; look for the care of each latrine and whether it is used by each member of the family.
- Are there washing and bathing facilities?

5. Irrigation furrows

- *Site:* chance of contamination; possibility of protection along its course.
- *Use:* speed and volume of water; fluctuations of amount of water during various periods of the year; use for man and/or animals; maintenance.
- *Quality of water:* Is water used for drinking, washing?

6. Fields or gardens

- Where do people working in the fields get water for drinking?
- Where do people working in the fields go to relieve themselves?
- Where is waste and refuse from the garden put?

DIARRHOEAL DISEASES UNSPECIFIED

1. Acute diarrhoeal disease is a clinical syndrome of diarrhoea (more than three liquid stools in 24 hours), nausea and/or vomiting, and often fever.

Synonym: Gastro-enteritis.

2. Occurrence and importance

Diarrhoeal diseases affect all the population, but severity varies in different age groups. Dehydration occurs rapidly in children and is a common cause of death (see the *Child Health* manual).

Especially at risk are:

Infants

Low-birth-weight children and premature children easily get *E. coli* infections. In the prematures, the case fatality rate may be high.

Weanlings

In the weaning period new types of food are introduced to children. They are then exposed to a variety of micro-organisms, pathogenic and non-pathogenic. Malnutrition is common during this period, and malnourished children have a lower resistance to infection. The incidence of diarrhoea in this period may be as high as three attacks per child per year. Diarrhoea is often more frequent in hot, dry periods — clearly associated with the shortage of water.

Bottle-fed children

Bottle-feeding is particularly dangerous in rural communities and among the urban poor. The mothers are generally poor and of low educational standard. They often do not have the facilities to clean the bottle properly, and they do not have enough money to buy adequate amounts of milk and other artificial feeds. The child gets fed with poorly prepared diluted feeds, and inevitably gets diarrhoea. Bottle-feeding is associated with a high mortality rate due to diarrhoea, and should be firmly discouraged in favour of breast-feeding.

Travellers

Traveller's diarrhoea occurs in people who are exposed to a new environment. This situation can arise from natural disasters, man-made disasters (war refugees) or among migrants (seasonal labourers) and holiday-makers. It is thought to be the gut's response to new intestinal flora acquired through faeco-oral contact, but other factors such as changes in food may also contribute. This diarrhoea is self-limiting.

Acute gastro-enteritis is endemic in all areas where sanitation is poor. From time to time the number of people with diarrhoea increases and so epidemics occur. The diarrhoeal diseases of children are closely related to this endemic diarrhoea in the general population.

Diarrhoeal diseases are important for the following reasons:

- They are a leading cause of infant mortality through dehydration;
- Numerous episodes interfere with nutrition and so they are an important cause of malnutrition (protein-energy malnutrition and vitamin A deficiency);
- Diarrhoea often accompanies measles and so contributes to measles deaths.

3. Epidemiology

Many organisms can cause diarrhoea but it is difficult to prove that any particular organism is responsible. Even when sophisticated techniques are used, in some of the cases no organism can be found. Bacteria and rota-viruses are common causes.

As will be described in the following sections, bacillary dysentery, paratyphoid fever, food poisoning, cholera, campylobacter infections and giardiasis, may all present as gastro-enteritis. In children, diseases like malaria and otitis media may cause what is known as parenteral diarrhoea. Other diarrhoeal conditions are caused by enteroviruses. In infants diarrhoea may be caused by enteropathic *Escherichia coli*, a bacterium of the bowel. All these organisms are transmitted by the faeco-oral route.

4. Clinical picture

There are various clinical presentations, depending on the cause of the diarrhoea. *E.coli* infection of babies causes profuse watery diarrhoea

with mucus but no blood. Fever is often absent. The onset is acute and it may progress rapidly to severe diarrhoea with as many as 20 motions a day (see also the *Child Health* manual.)

Protein-calorie malnutrition is commonly associated with weanling diarrhoea, and frank kwashiorkor may follow an attack.

The fluid balance in children is more easily disturbed than in adults because of rapid fluid turnover. Dehydration readily occurs and may be severe.

The clinical picture of gastro-enteritis in adults may vary from a mild abdominal disturbance to a dysentery-like disease.

5. Management of the individual patient

The treatment of choice in the management of diarrhoea is rehydration fluid. (For the management of diarrhoea in children see *Child Health* manual.)

The degree of dehydration should be carefully assessed, and the plan shown in Figure 5.7 followed. Important points to remember are:

- Continue feeding and give extra meals on recovery to improve catch-up growth;
- Continue and increase breast-feeding;
- Give an antibiotic only if there is blood in the stool;
- If there is fever, look for the cause and treat;
- Do not give other medicines such as kaolin;
- Teach the mother how to make oral rehydration fluid and how to feed it to the child. Give her enough packets of ORS to take home if you are using them for oral rehydration;
- If the child has had several episodes of diarrhoea before, give vitamin A also;
- Educate the mother on prevention.

6. Prevention and control

Infants

Prevent low birth weight and prematurity by improved antenatal care.

Weanlings

Prevent malnutrition in the weaning period by nutrition education and improved care. Continue breast feeding in the second year.

Bottles: Discourage bottle-feeding (see page 168).

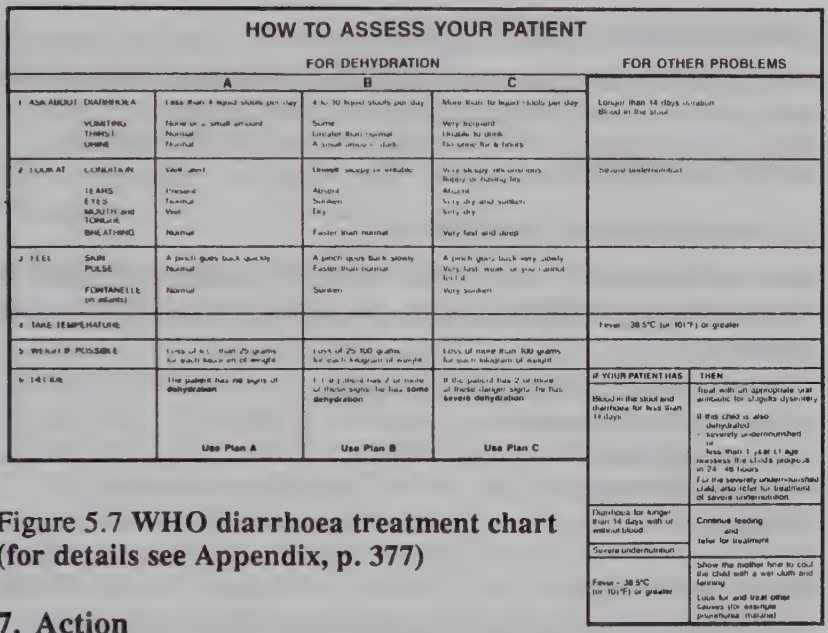


Figure 5.7 WHO diarrhoea treatment chart (for details see Appendix, p. 377)

7. Action

Control of Diarrhoeal Diseases (CDD) programmes are now an active part of the child survival programmes in the health ministries of several countries. The activities included in these programmes are:

- Oral rehydration therapy to cut down diarrhoeal deaths;
- Improvement of water and sanitation to reduce transmission and the number of episodes of diarrhoea;
- Improved weaning practices and nutrition of children;
- Investigation of diarrhoeal outbreaks;
- Immunization against measles;
- Sustained breast-feeding;
- Antenatal care to reduce low birth weight.

8. Summary

Diarrhoeal diseases are very common in Africa. Children are especially at risk. Causative organisms are difficult to identify without very good laboratory facilities. Spread is by the faeco-oral transmission route. Correction of dehydration is the main priority in treatment. Control: improvement of sanitation, nutrition, and health education.

BACILLARY DYSENTERY

1. Bacillary dysentery is an acute diarrhoeal disease characterized by bloody stools, fever, vomiting, and abdominal cramps.

Synonym: Shigellosis.

2. Occurrence and importance

Bacillary dysentery is common throughout Africa, especially in areas where sanitary conditions are poor. The main factors influencing its occurrence are:

- Methods used in the disposal of faeces;
- Availability of water;
- Fly population;
- Seasonal changes;
- Nutrition.

Water availability

In the dry season the amount of water available for cleaning purposes decreases. Cleaning of eating utensils (and washing of hands) gets a low priority. The available water sources may be contaminated. Thus an increase in dysentery cases can be expected.

Flies

The number of house flies is dependent on seasonal influences. House flies are very likely to transmit dysentery. When the number of flies is high, the incidence of dysentery also increases.

Seasonal changes

During the rainy season a lot of faecal material outside latrines will be washed into ponds and rivers. This results in heavy contamination of water sources, and increased incidence of diarrhoeal diseases.

Nutrition

Malnutrition lowers general resistance against dysentery. Shigella infections will occur more readily, resulting in attacks of frank dysentery. Poorly nourished children and old people are therefore

especially at risk. Other undernourished groups living in poor and overcrowded conditions, such as prisoners of war and refugees, are also at risk.

In children, diarrhoea or dysentery easily causes dehydration and this accounts for the high mortality rate of diarrhoeal diseases in this age group.

3. Epidemiology

Bacillary dysentery is caused by non-motile Gram-negative bacilli of the *Shigella* spp. Those most frequently responsible for outbreaks are *Sh. sonnei*, *Sh. dysenteriae* and *Sh. flexneri*. Man is the only reservoir for outbreaks. Following infection, people may be symptomless carriers for up to 3 months. Transmission is by faeco-oral contact.

Shigellae multiply in food. The food may be contaminated directly by flies or unwashed hands carrying the bacteria, or indirectly through dishes which are not washed properly. The shigellae start to multiply and if such food is ingested, dysentery may result. Children form the main reservoir of infection because they often defecate in and around houses. As the bacilli are present in the stools of children, even after an attack, contamination of food and/or water can easily occur.

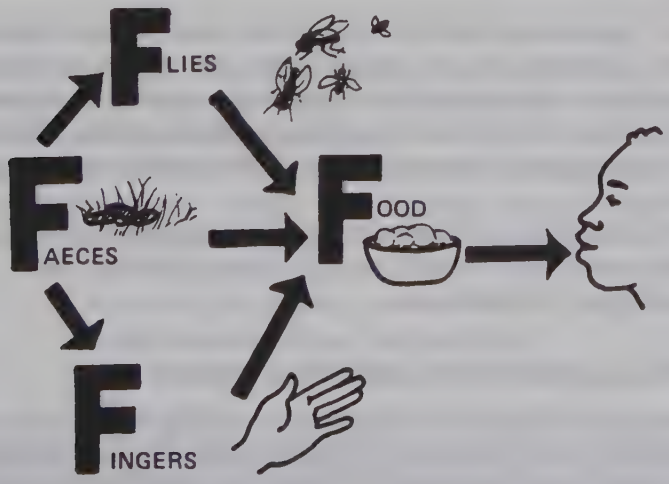


Figure 5.8 The rule of F

4. Clinical picture

The incubation period is short (1–4 days). In the well-nourished adult, infection with shigella may result only in an attack of mild diarrhoea. In the undernourished child it may result in a fulminating toxic and fatal disease.

Mild cases are often not recognized and are regarded as non-specific gastro-enteritis.

In typical cases, the onset is sudden with fever, colicky abdominal pains, and faecal diarrhoea.

After a few motions the diarrhoea stops and the dysenteric syndrome sets in. This is characterized by abdominal cramps (colicky pains) and tenesmus. Tenesmus is painful contractions of the sphincter ani, producing an almost continuous and irresistible urge to defecate. However, no faecal matter is produced, only small quantities of purulent mucus and blood. There may be associated vomiting.

Toxaemia is present due to absorption of an exotoxin secreted by the bacilli. There is a high fever, and the pulse is rapid. Convulsions may occur in children. Dehydration is common and dangerous: it may cause muscular cramps, oliguria and shock.

Rectal prolapse may occur in infants.

Diagnosis

Stools: Appearance dark red (blood) with much mucus. Microscopy shows numerous white blood cells and many erythrocytes. Macrophages which contain red blood cells (after phagocytosis) are easily mistaken for trophozoites of *Entamoeba histolytica*. The nucleus of a macrophage is, however, more clearly visible and is irregularly shaped.

The diagnosis is confirmed by a positive stool culture for *Shigella*.
Blood examination: There is usually a leucocytosis of about 15,000, but anaemia is not common.

5. Management of the individual patient

Prevention or treatment of dehydration is all that is necessary in mild infections. In severe infection, rehydration must be be combined with appropriate antibiotics because death is usually due to a combination of dehydration and toxaemia.

Table 5.3 Distinguishing between bacillary and amoebic dysentery

	Bacillary dysentery	Amoebic dysentery
Incubation period	Short: less than 1 week	Long: 3 weeks or more
Onset	Acute	Insidious
Occurrence	Epidemic	Endemic
Fever	Common	Only in complications
Clinical picture	"Lying-down dysentery"	"Walking dysentery"
Tenderness	Whole abdomen	More localized (sigmoid)
Tenesmus	Very severe	Not usual
Stools		
Macroscopic	Mucus and blood only	Stools with blood and mucus
Microscopic	Numerous red cells	Numerous red cells in clumps
	Numerous polymorphs	Polymorphs scanty
	Few bacteria	Many bacteria
	Macrophages	*E. histolytica* trophozoites with ingested red cells

Oral rehydration can be started as soon as possible. Oral rehydration is always useful as an aid to parenteral rehydration and it carries less danger of disturbing electrolyte balance.

Morphia and other narcotics are not indicated, although they may be used for the painful cramps and tenesmus. Spasmolytics like belladonna will relieve most pain.

Rehydration is the first priority

Antibiotics are indicated in case of severe systemic symptoms.

Tetracycline, ampicillin or cotrimoxazole may be used, but most shigella strains are now resistant to these and other drugs. Only culture and sensitivity tests can identify the most appropriate antibiotic.

Unfortunately, this investigation is only possible in big hospital laboratories.

Faeces of the patients are highly infectious, and should be handled with care. Required measures are:

- Rigid personal precautions by attendants;
- Disinfection and proper disposal of faeces;
- Elimination of fly contact with faeces.

Patients and contacts should not be employed as food handlers until any danger of spread is no longer present.

6. Prevention and control

Prevention of bacillary dysentery depends on stopping the faeco-oral transmission.

7. Action

Patient

- Rehydrate (oral and parenteral);
- Treat with antibiotics (determine sensitivity if possible);
- Concurrent disinfection of faeces;
- Terminal cleaning;
- Exclude from food handling.

Outbreaks

Whenever there is an outbreak of bacillary dysentery (or other diarrhoeal disease):

- Check the water supply;
- Find out if there is a localized pattern in the spread of the disease: families/village; is vehicle of transmission water? Food? Flies? Examine all points on the checklist in the Introduction to this chapter (p. 166);
- Direct your actions to possible causes;
- Give health education on preparation of rehydration fluids; use of safe water (boiling); use of safe food (preparation and storage); washing of hands with soap and water; proper refuse disposal; and proper faeces disposal.

Endemic bacillary dysentery

- Give health education on use of latrines, safe water, safe food, refuse disposal, and personal hygiene.
- Stress the importance of prolonged breast-feeding.
- Stress in village development committee meetings the importance of improvement of water supply, either with protected wells or piped water.
- Stress in the same committees the importance of latrines.
- Inspect, with your health assistant, public eating houses, markets, boarding institutions (schools, camps); see checklist p. 166.

8. Summary

Bacillary dysentery is an acute diarrhoeal condition which may vary from a mild diarrhoea to a severe toxic and fatal disease. It is widely spread in Africa, and is common and severe, especially in malnourished children. Rehydration is the mainstay of management. Antibiotic resistance of various *Shigella* strains is now common. Control depends on proper disposal of faeces and improvement of water supply.

CAMPYLOBACTER JEJUNI INFECTIONS

1. This infection is due to an enteric Gram-negative micro-aerophilic bacterium known as *Campylobacter jejuni*. It presents with various symptoms ranging from mild gastro-enteritis to severe dysentery. Asymptomatic carriers may serve as a significant reservoir for the infection. In many countries this organism is becoming recognized as an important cause of bloody diarrhoea.

2. Occurrence and importance

Campylobacter exists as a non-pathogen in cattle, sheep, pigs, fowl, dogs and cats. Humans may also carry it as a commensal organism (asymptomatic carriers may range from 5–15% of the population), and it is particularly common in children under 2 years of age. In many parts of Africa where investigations have been made poultry is a common source. Chickens living in houses may thus pose a danger to small crawling infants.

3. Epidemiology

Ingestion of less than 500 bacteria will cause clinical illness. Symptoms and signs are related to dose and bacterial virulence, as well as host factors. Low gastric pH (high acidity) kills many of the bacteria. Once through the stomach, the bacteria colonize the distal small intestine and colon. The incubation period ranges from 1 to 7 days with a mean of 2– 4 days. The bacteria may invade the intestinal wall, making small ulcerations in the mucosa. Transient blood invasion may occur. During this period large numbers of bacteria are shed into the stool. Symptoms are related to the time taken for the bacteria to be cleared from the intestines, and usually last 1–3 weeks, coinciding with the onset of increasing antibody titres.

4. Clinical picture

Three cardinal signs and symptoms are abdominal pain, diarrhoea and fever. Bloody stools are not common. Symptoms may range from a mild diarrhoeal illness to a fulminant dysentery.

Diagnosis

Because *Campylobacter jejuni* grows best in an environment of 5% oxygen at a temperature of 42°C and is slow growing, special techniques and media are necessary to isolate it. Stool culture is the only way to confirm the diagnosis. Presumptive evidence is seeing motile bacteria under dark-field microscopy.

5. Management of the individual patient

Studies suggest that antibiotics significantly decrease bacterial shedding time but do not significantly alter the patient's symptoms nor the length of illness. If a patient has mild symptoms, treatment may not be necessary. For more severe illness, erythromycin, in doses of 500 mg orally four times a day (or 50 mg/kg/day for children) for 7 days, is effective. If relapse occurs, treat for 3 weeks. An alternate drug is tetracycline, 500 mg orally four times a day.

6. Prevention and control

Prevention largely depends on reducing household (especially

kitchens) contamination by domestic animals. Improved food hygiene techniques are also important.

7. Action

Patient

- Stool culture but specify possibility of campylobacter. If seriously ill treat with erythromycin.

Environment

- Reduce the number of animals and poultry coming into houses and yards.
- Cook all poultry very well and wash utensils after preparation and expose to sun on drying table.
- Regard all purchased chicken as potentially infected and observe good hygienic preparation and cooking methods.

8. Summary

Campylobacter infections are now a more common cause of diarrhoea and dysentery and the source is often domestic animals especially poultry.

GIARDIASIS

1. This is an infection of the small intestine caused by a flagellated protozoal parasite, *Giardia lamblia*. When symptomatic, it causes diarrhoea, malabsorption and weight loss.

2. Occurrence and importance

This is an infection of the small intestine caused by a flagellated protozoal parasite, *Giardia lamblia*. When symptomatic, it causes diarrhoea, malabsorption and weight loss.

3. Epidemiology

Giardiasis is endemic throughout the world, but commoner in developing countries where water supplies may be contaminated by human sewage. Several rodents and dogs are thought to be reservoirs

for the disease, but it is not yet clear whether they harbour strains pathogenic to man. Asymptomatic carriers among people are common, and infection can spread from person to person. Cysts are excreted in the stool, and are infectious for up to 3 months in cold water (but only 4 days if the temperature is 37°C). Ingestion of the flagellated organism (trophozoite) does not cause infection. Ingestion of the cyst, however, does.

Cysts are activated by the acid of the stomach when eaten by man. Emerging trophozoites adhere to the mucosal wall of the upper portion of the small intestine and begin to divide. The incubation period ranges from 5 to 15 days. Increased susceptibility to the illness occurs in the elderly, the debilitated, poorly nourished children, and those with reduced immunity.

4. Clinical picture

The majority of infected persons are asymptomatic. In those who are ill, the symptoms range from mild diarrhoea to debilitating malabsorption and weight loss. Lactose intolerance due to reversible lactase deficiency and malabsorption of fat and vitamin B12 may occur as complications.

Most of the symptoms are a result of the malabsorption, and include abdominal discomfort, cramps, nausea, bloating sensation, frequent loose, bulky, foul and urgent stools, malaise and/or weight loss. The disease may be self-limiting or prolonged. The acute stage lasts for approximately 4 days on average, but may persist for months or years. The mean duration of symptoms is 6–7 weeks.

Diagnosis

This is often difficult to establish. Stool examination rarely reveals motile trophozoites, but approximately 60% of samples will show cysts. Three separate stool specimens can bring this sensitivity to 90%. If symptoms persist for weeks, and there is a strong suspicion of giardiasis in spite of negative stool examination, a treatment trial may be warranted.

5. Management of the individual patient

Metronidazole 250 mg orally three times a day for 7 days (5 mg/kg three times a day for 7 days); reported effectiveness 85%. Single-dose

treatment is not very effective.

OR

Tinidazole 2 g orally as a single dose; reported effectiveness is around 90%.

A second course of treatment may be necessary if the initial one is unsuccessful.

6. Prevention and control

The cysts of *Giardia lamblia* are highly susceptible to heat. Cooking food or boiling drinking water kills the cysts rapidly. The cysts are not affected by chlorine treatment of water or by iodine.

7. Action

- Improve food hygiene.
- Avoid eating raw vegetables in endemic areas.
- Improve water and sanitation.

AMOEBIASIS

1. Amoebiasis is an infection caused by a pathogenic amoeba *Entamoeba histolytica*. Infection with amoebae is in most cases symptomless. Under certain circumstances the amoebae may change into tissue parasites and invade the bowel wall causing amoebic dysentery. The infection can be spread to other organs, especially the liver, where it causes an abscess.

2. Occurrence and importance

Clinical amoebiasis is endemic in areas where sanitary conditions are poor. In eastern Africa, liver amoebiasis is especially common in the Arusha and Kilimanjaro region of Tanzania. Immunity seems an important factor in the spread of the disease.

3. Epidemiology

Entamoeba histolytica is the only pathogenic one of several *Entamoeba* species which can parasitize man. It is distinguished from the others by size and the number of nuclei (four) seen in cyst form. It is acquired

when cysts are ingested. The emerging trophozoite takes up residence in the ascending large bowel where it can live as a commensal (asymptomatic carrier) or begin penetrating the intestinal wall causing small mucosal ulcerations which result in dysentery. The parasite penetrates through the submucosa and into the muscularis layer of the colon. If the muscularis layer becomes extensively involved, scarring can result (amoeboma). Invasion of the bloodstream by the trophozoites may lead to infection of the liver, especially the right lobe. After a variable incubation period, a liver abscess may form. Other sites of metastasis may occur but are rare (see Figure 5.9).

The only infective form of *E. histolytica* is the cyst. A patient with amoebic dysentery is unlikely to spread the disease because he is only passing out trophozoites, which if ingested are destroyed in the stomach. It is the asymptomatic cyst-passer who forms the main reservoir for the spread of amoebiasis. Cysts are passed from person to person by the faeco-oral route, by fingers soiled with faeces, either directly into the mouth or via food. Amoebiasis can occur in families or spread through institutions, but usually does not occur in epidemics, unlike bacillary dysentery. Although infections may occur from drinking water, amoebiasis is not really a water-borne disease. It can be endemic in a population in which many individuals are asymptomatic cyst-passers with only a few getting the disease.

4. Clinical picture

Infection is usually asymptomatic

When amoebae penetrate the intestinal wall they multiply in the submucosa, causing bottle-shaped ulcers. From these ulcers the amoebae may be transported to the liver. An amoeboma, or amoebic granuloma, may result from repeated invasion in the colon. An amoeboma may become very large, forming a hard swelling which is very difficult to differentiate clinically from carcinoma (try treatment for amoebae first in case of suspected colon cancer). Usually the onset of amoebic dysentery is insidious and associated with abdominal discomfort. There may be mildly loose stools or frank diarrhoea with

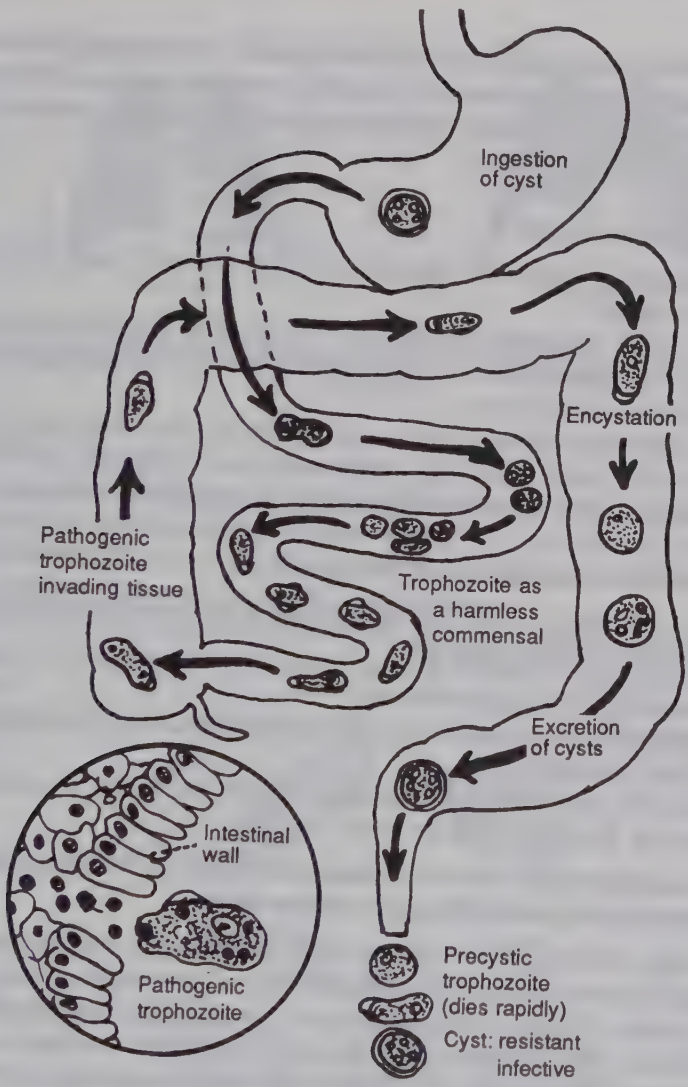


Figure 5.9 **The life cycle of *Entamoeba histolytica***

or without blood and mucus. Tenderness may develop over the caecum or sigmoid.

In severe cases the onset is more sudden, the patient is ill and toxic with fever and signs of dehydration. Hiccup is a bad sign. The faeces contain a lot of dark, altered blood and blood-streaked mucus. Trophozoites are present in large numbers.

Chronic amoebiasis is difficult to diagnose. Alternating diarrhoea and constipation are often seen. The colon may be distended. Chronic amoebiasis may resemble duodenal ulcer, gall bladder disease, or carcinoma of the colon.

Amoebic liver abscess

This condition should be suspected in people who have lived in endemic areas.

The most common findings are: high ESR and leucocytosis; enlarged and tender liver; elevation and fixation of the right diaphragm (X-ray). Fever may occur.

An amoebic liver abscess may perforate into the chest cavity (see Figure 5.10, page 185), the peritoneal cavity (causing an acute abdomen), or through the skin (causing cutaneous amoebiasis).

Other extra-intestinal manifestations

Skin: Amoebiasis of the skin may occur where amoebae come into contact with the skin, that is:

- around the anus and perineum;
- around incision wounds, e.g., after appendicectomy or drainage of a liver abscess;
- around a fistula from a liver abscess which perforated; this opening may be far away from the liver.

An amoebic skin ulcer is irregular and painful. The ulcer enlarges continuously because of necrosis of the edges.

Lungs: When a liver abscess breaks through into the chest cavity, a lung abscess (which may perforate into a bronchus) or empyema may occur. When a liver abscess is close to the diaphragm, the diaphragm will not move and the right lower lobe will not be well ventilated. As a result, liver abscess is often accompanied or masked by bronchopneumonia in the right lower lobe. Pleurisy, dry or with effusion, may occur if the pleura covering the diaphragm is inflamed.

Brain: Rarely, an amoebic brain abscess may complicate severe amoebiasis.

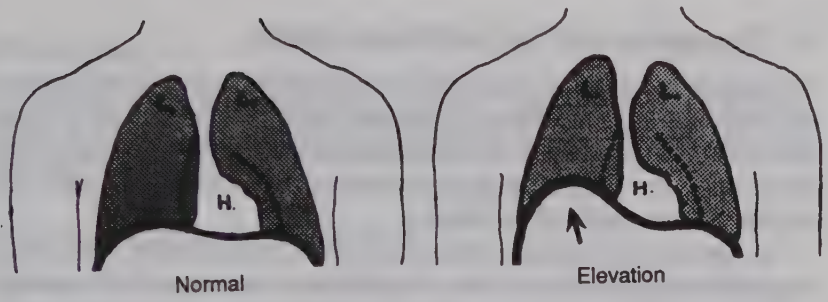


Figure 5.10 The elevation of the diaphragm in amoebic liver abscess

Diagnosis

The presence of cysts in stools does not prove that symptoms are caused by entamoebae. Trophozoites of *E. histolytica* should be differentiated from those of *E. coli*. The main difference is the presence of ingested erythrocytes in the *E. histolytica*.

Cysts can be differentiated when a stool specimen is stained with Lugol's solution to demonstrate the nuclei. *Histolytica* cysts have four nuclei.

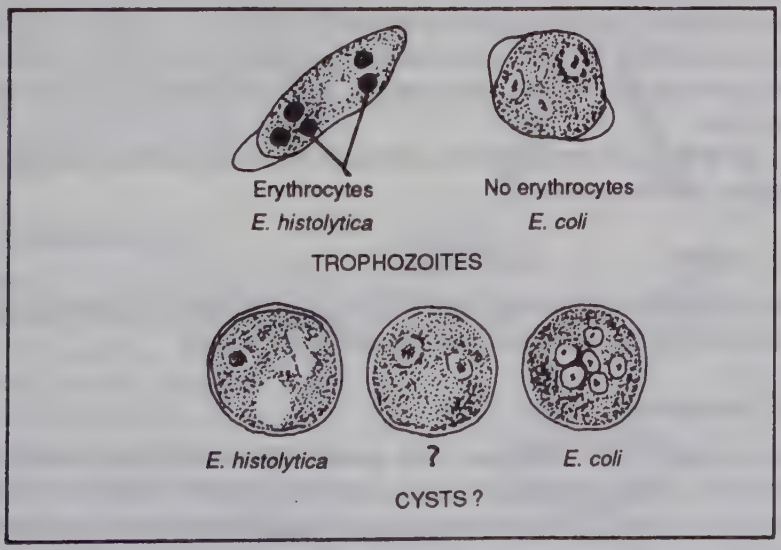


Figure 5.11 Differentiation of *E. histolytica* and *E. coli*

5. Management of the individual patient

Asymptomatic patients normally do not need treatment because in time they clear the infection. (However, some clinicians like to clear the cysts in the pre-invasive phase. Metronidazole 800 mg orally three times a day for 5 days can be combined with diloxanide furoate 500 mg three times daily for 10 days.)

In invasive amoebiasis where the active trophozoites are found, metronidazole is used as above. For children the dosage is 25 mg/kg/day. If the disease is severe, diloxanide furoate (as above) can be added as well as tetracycline 250 mg four times a day.

In hepatic amoebiasis, Metronidazole is the drug of choice. Aspiration may be needed for large abscesses. This should only be carried out in hospital. If response is poor, chloroquine 600 mg base initially, followed by 150 mg twice a day for a month may be tried. Emetine is very occasionally still used for hospitalized cases which do not respond to the above (1 mg/kg intramuscularly daily for 5 days).

6. Prevention and control

The cyst-passers are responsible for the spread of amoebic dysentery in the community. They are usually asymptomatic and will not report for treatment. People employed as food handlers, i.e., cooks, waiters, etc., should be screened before and during employment. Water cannot be made safe by ordinary chlorination. Boiling of drinking water kills the cysts.

As with bacillary dysentery, the most important control method is proper faeces disposal.

7. Action

Search for carriers among food handlers. Commence a campaign for more latrines in any area with endemic amoebiasis.

8. Summary

Amoebiasis is an infection with potentially pathogenic amoebae. Infection occurs through the faeco-oral transmission route. Infection is usually symptomless but may result in attacks of dysentery and/or liver abscess. Treatment covers both tissue parasites and parasites in the bowel lumen. Control depends on proper disposal of faeces. Asymptomatic carriers are difficult to trace.

CHOLERA

1. Cholera is an acute intestinal disease characterized by sudden onset of profuse watery stools, vomiting, rapid dehydration and circulatory collapse.

2. Occurrence and importance

Cholera epidemics occurred in Egypt and North Africa in the last century. The pandemic of El Tor cholera started spreading in eastern Africa in the early 1970s, and then also spread down the west coast of Africa.

Even in severe epidemics seldom more than 1–2% of the persons infected develop clinical signs of cholera. For every clinical case of cholera there may be 50–100 more asymptomatic carriers. Fatality rates of untreated classical cholera exceed 50%. But with adequate rehydration this can be brought below 1%.

Clinical cholera is usually seen in the lowest socio-economic groups. Children (but not infants) are more susceptible. A rapidly rising incidence of acute diarrhoea with dehydration in young people and adults should lead to a suspicion of cholera.

3. Epidemiology

Cholera is caused by *Vibrio cholerae*, the comma bacillus, a Gram-negative, very small, curved, motile organism. The current pandemic is caused by substrain El Tor vibrio. Because cholera vibrios are very sensitive to gastric acid a large number of vibrios have to be ingested in order to cause disease. For this reason cholera is not a very

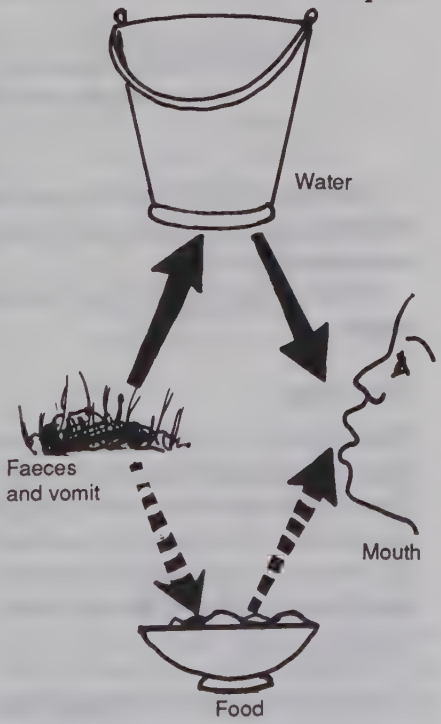


Figure 5.12 Transmission is by the faeco-oral route

infectious disease. Transmission is through the faeco-oral route but almost all cholera infections are water-borne. Vibrios can live in water for 2 weeks. They prefer brackish (salty) water. In sea water they may survive for longer periods. Vibrios readily multiply in certain foods such as milk and boiled rice. The reservoir of infection is formed by the carriers. These carriers excrete vibrios in smaller numbers than the patients but, because of their freedom of movement and the fact that they far outnumber the patients, they form the greatest danger to the community. The present El Tor strain is generally clinically mild and therefore more easily spread by convalescents and carriers.

4. Clinical picture

The incubation period is usually 2–3 days.

Most infections are asymptomatic or cause only a simple self-limiting diarrhoea

Cholera is not a systemic infection. The vibrios are confined to the intestinal canal. The clinical syndrome of cholera is caused by water and electrolyte loss.

In the typical case of classical cholera the disease develops in three stages.

First stage

This lasts for 3–12 hours. Profuse watery stools pour from the patient. Soon faecal matter disappears from the stools, which become almost clear fluid with flakes of mucus giving them the classical rice-water appearance. Vomiting follows the diarrhoea. At first food is vomited but soon only rice-water is vomited. Severe cramps in the abdomen and limbs develop from salt loss.

Second stage

This is the stage of collapse from dehydration. The body becomes cold, the skin dry and inelastic. The blood pressure is low or unrecordable, the pulse rapid and feeble. Urine production stops and finally the patient may die of shock.

Third stage

This is the stage of recovery, either spontaneously or with treatment. The diarrhoea decreases, the patient is able to take fluids, and the general condition rapidly improves.

Table 5.4 Distinguishing between cholera and food poisoning

	Cholera	Food poisoning
Diarrhoea	Precedes vomiting	Follows vomiting
Vomiting	Watery and projectile; causes no distress	Violent and distressing; vomit consists of food, is never watery
Nausea	Absent	Common
Abdominal pain	Not usually severe	Constant
Tenesmus	Absent	Common
Stools	Rice-water Not offensive	Liquid, faecal, offensive, never colourless
Urination	May be completely suppressed	Usually not suppressed
Muscular cramps	Constant, severe	In very severe cases, extremities only
Headache	Absent	Frequent
Fever	Absent	Only present if a salmonella infection

Diagnosis

Cholera should be suspected in any outbreak of diarrhoeal disease. The diagnosis is made on clinical grounds. Do not refer patients to confirm diagnosis but send a rectal swab or stool specimen in transport medium if available, to the district laboratory — if transport medium is not available soak a piece of blotting paper in stool and seal in a plastic bag and send to laboratory.

Do not refer a suspected cholera case but inform your district medical officer

5. Management of the individual patient

Patients can be admitted to a temporary hospital (school, church). Strict isolation is not necessary as only the vomitus and stools are infective. These should be disinfected. Carrry out concurrent disinfection and terminal cleaning.

The patients are treated on "cholera beds", beds with a central hole through which the continuous stools can pass into a bucket and be measured.

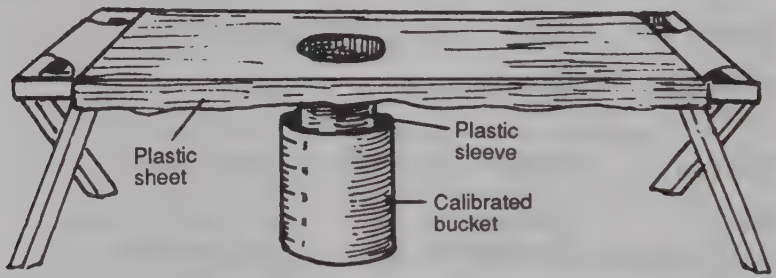


Figure 5.13 **Cholera bed**

The essential cure of cholera is rehydration; this, even without any drug, will save many cholera cases if started in time.

Oral rehydration

Patients of all ages who are strong enough to drink will voluntarily ingest the volume of glucose/electrolyte solution needed for rehydration and maintenance.

Patients in shock or too weak to drink require intravenous fluids for a few hours only and can then be given oral fluids. Vomiting is caused by acidosis and fluid loss. It may last for a few hours but the volume is usually small and continuous drinking should be able to replace the loss.

Oral rehydration should be given in frequent small amounts or by gastric tube (children). Disturbances of electrolyte balance are less

common with oral rehydration, and intravenous fluids can be reserved for those with severe dehydration who really need it.

A mother can give a child a teaspoon of fluid per minute and so 200–300 ml per hour.

A suitable oral rehydration fluid is the standard WHO/UNICEF ORS sachet,which is made up of the following:

NaCl 3.5 g
$NaHCO_3$ 2.5 g or citrate
KCl 1.5 g
Glucose 20 g
Water to 1,000 ml.

The volume given should be about 1.5 times the volume of the stool. The absorption of glucose and sodium takes place in spite of the massive secretion of fluid by the intestine. The fluids can be administered by family members and non-professionals as well as by health workers.

Glucose has been shown to speed up the absorption of sodium and water in the small intestine. If glucose is not available it can be replaced by 40 g/litre sucrose (sugar).

Rehydration will save almost all cases

Tetracycline will speed up the cure and prevent the convalescent carrier stage, but multiple resistance strains of El Tor vibrios are now being identified (e.g., in Tanzania). It is given in a dose of 500 mg four times a day. Cotrimoxazole two tablets twice daily for 3 days for adults can also be used effectively.

Note
A team from the nearest large health centre or the district centre should assist as soon as possible because if cases are brought in late they may already be in shock requiring urgent intravenous drips which may be in short supply in a small unit.

6. Prevention and control

Surveillance is the key to a successful cholera control programme.

Surveillance is the continuous watching of all aspects of a disease; it includes collection of morbidity and mortality reports, field

investigations of epidemics or individual cases, laboratory investigations such as culturing.

Once an outbreak of a disease under surveillance is noted in a certain area immediate action must be taken in that area. Surveillance depends on reporting suspected cases of cholera. It is obligatory to report any case immediately to the DMO by telegraph, telephone or any other rapid means.

Cholera is an internationally notifiable disease

Three factors influence reporting of new cases by the public:

(a) Reporting is greatly inhibited when repressive measures are taken once a case is identified. Repressive measures can be: quarantining a family, hospital, community or an area; imposing a military cordon or restricting movements of people or goods into and out of infected areas. Measures that limit the movements of people or restrict the shipment of goods and food are never necessary. Such measures will increase the hysteria among the people and will hinder surveillance, as people will not come forward to report cases.

(b) Saving of lives should be the top priority in the control of cholera. Fear and panic occur in the community when deaths occur. If cases are recognized and treatment given without delay, fear will disappear and families will report their cases.

(c) Treatment centres should not be quarantined. Reluctance to report cases and fear of the disease can be overcome by allowing visits to the patients. A parent should stay with a paediatric case to assist in oral fluid therapy and nursing.

These measures are important because they stress the relatively benign nature of the disease and show it is not very infectious.

Water

As cholera is mainly a water-borne disease it cannot spread when water is made safe. This can be done by chlorination of public water supply or by boiling or treating supplies for individuals. In case of an emergency large quantities of water can be treated with bleaching

powder (chloride of lime): 4 lb of bleaching powder (= 1 lb chlorine) in 100,000 gallons of water (or 2 kg powder in 500,000 litres) will give the required concentration of one part per million (1 PPM).

Food

Only certain foods can transmit cholera, under special circumstances and for a limited period of time. Milk products should be pasteurized. Uncooked food should be avoided or washed in safe water. Left-overs should be protected against contamination by flies. Markets must be inspected.

Sanitation

Improvement of sanitation facilities will result in a lower incidence of all diarrhoeal diseases including cholera. Emergency measures in case of epidemics are impracticable and not the first priority.

Chemoprophylaxis

Tetracycline can prevent cholera in households where there is a cholera case. But administration of tetracycline to the entire population is impracticable and inadvisable. Mass chemoprophylaxis would only result in indiscriminate use of drugs and increase the danger of drug-resistance or drug reactions developing.

Quarantine

This can never be an efficient protection against the introduction of cholera into the community since convalescent patients and many healthy carriers pass vibrios in their stools.

Immunization

Cholera vaccine is of low potency. It gives 50% protection for 3–6 months only. The vaccine will give some individual protection against clinical cholera, but mass immunization will increase the number of asymptomatic carriers. As a general measure to stop an epidemic from spreading, immunization is of no value.

The public should be informed of the limited value of immunization because in an epidemic they will demand protection and they may panic when they find that vaccine is not available. On the other hand,

pressure may make the authorities start an immunization programme, and so neglect the more important purification of the water supply. People who are immunized may have a false feeling of security and may be encouraged to consume unsafe water or food.

Prevent cholera with clean water supply

7. Action

If you suspect an outbreak of cholera in the catchment area of your health centre do the following:

- Open a temporary hospital (in the school, church);
- Notify your DMO and plan to inform the public;
- Treat the main water supply with bleaching powder or chlorine;
- Take stool specimens for culture;
- Do not refer suspected cases;
- Have large amounts of rehydration fluid prepared;
- Rehydrate as many patients as possible with oral fluids only;
- Give intravenous fluids for a short period, only to patients in shock;
- Have cholera beds prepared;
- Give health education on how cholera is spread; how water can be made safe (boiling).

Some funeral ceremonies may be conducive to the spread of the disease. If this is the case, communities need further education about burials.

8. Summary

Cholera is an acute intestinal disease characterized by rice-water stools, vomiting, rapid dehydration with shock. It is spread through contaminated water. Most cases are subclinical infections resulting in the carrier state. Control depends on surveillance (international). In an outbreak the first priority is to improve the water supply.

THE ENTERIC FEVERS

1. The enteric fevers include typhoid fever and paratyphoid A and B fevers. Typhoid fever is a systemic infectious disease characterized by high continuous fever, malaise, and involvement of lymphoid tissues and spleen.

Diarrhoea is not a common symptom in typhoid fever.

Paratyphoid fever may present like typhoid fever, but most cases present as gastro-enteritis or transient diarrhoea. See salmonellosis under Food Poisoning, page 199.

2. Occurrence and importance

The enteric fevers are endemic in many regions of Africa. Epidemic outbreaks can occur when a source of water or a food used by many people is contaminated.

Fatality rate can be 10% but with adequate antibiotic treatment this should be reduced to less than 3%. Paratyphoid is not often diagnosed in eastern Africa.

3. Epidemiology

The enteric fevers are caused by salmonellae. *S. typhi* causes typhoid in man, and man is the only source and reservoir. Transmission is by the faeco-oral route. Salmonellae are passed out in faeces and urine of cases or carriers. The main ways of spread are through contaminated water and food. It is usually most common at the end of the dry season and at the start of the rains. Contamination of food usually occurs from the hands of carriers or undiagnosed patients. It is common in areas where there is insufficient water for washing hands.

Reservoir

Man is the only reservoir: patients and carriers. During convalescence typhoid bacilli often settle in the gall bladder, and are excreted in the faeces for a very long time if the patient is not treated with ampicillin to eradicate the *S. typhi*. This is especially common in women.

Urinary carriers are seen particularly in areas where *Schistosoma haematobium* infections occur.

4. Clinical picture

The incubation period is 2–3 weeks. The onset is gradual. Early symptoms and prodrome are severe headache, malaise, anorexia, body pains, and epistaxis. In the first week of the disease the temperature rises in steps. Most patients cough due to bronchitis. Usually the patient is constipated.

The pulse rate is relatively slow — that is, slower than would be expected from the level of temperature. The spleen is usually enlarged and tender but this is of little diagnostic significance in malarious areas. During the first week of the disease the patient feels sick and feverish. He may complain of cough and constipation.

In the second week the temperature is continuously high. Half of the patients may develop diarrhoea. Typhoid patients may become confused and disoriented with hallucinations. The abdomen becomes distended and tender due to ulcers in the lymphatic tissues of the intestine.

Table 5.5 **Clinical picture of enteric fevers**

	Prodrome	First week	Second week	Third week
General	Headache Anorexia Malaise	Apathy Meningism	Mental confusion Disorientation	
Temperature	Normal	Rising stepwise	Continuously high	Decreasing stepwise
Chest		Bronchitis Scattered crepitations	Eventually broncho-pneumonia	
Abdomen		Spleen enlargement	Abdominal distension and tenderness	Complications: bleeding perforation
		(Ileum may perforate in first or second week.)		
Stools		Constipation	"Pea soup" diarrhoea	

These ulcers may cause bleeding and perforation in the third week of the disease.

Diagnosis

The best way to prove typhoid fever is to make a blood culture. This may be positive during the first week and for a variable period after this. Stool and urine cultures are positive after the first week. The Widal test becomes positive by the end of the first week, and a rising titre shown by two tests performed 4–5 days apart may indicate active typhoid. The interpretation of Widal tests is, however, full of difficulties and in many cases it is of little use in the diagnosis of typhoid, especially in endemic areas and in people who have had the vaccine.

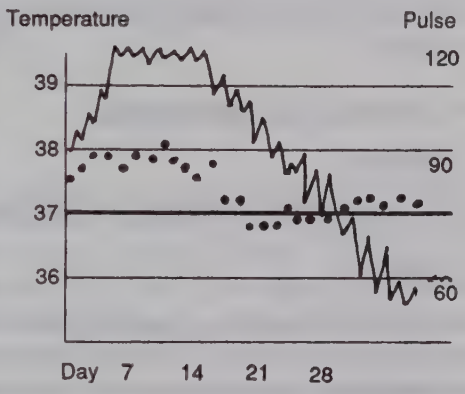


Figure 5.14 **Temperature and pulse rate in typhoid fever**

Helpful laboratory tests are:

- WBC count: low (leukopenia) with a relative lymphocytosis;
- Stools: in 100% of the cases occult blood is present in the stools.

5. Management of the individual patient

Chloramphenicol is the drug of choice. Do not give a loading dose as it may cause a toxic crisis with circulatory failure (Herxheimer reaction). The dose is 500 mg 6-hourly for 14 days (if given for less than 10 days there may be a relapse). Chloramphenicol will not bring down the fever immediately: usually it takes at least 3 days before the effect is seen. Alternatives to chloramphenicol are: amoxycillin, ampicillin and cotrimoxazole.

Patients should be isolated in a fly-proof room. Rigid personal precautions should be taken by attendants. Concurrent disinfection of faeces and urine and all contaminated articles is required before they can be disposed of directly into sewers, if they exist. Terminal cleaning of all contaminated articles must also be undertaken.

Perforation of the bowel needs surgical treatment. Patients with signs of peritonitis should be referred as they may be about to perforate. Bleeding should be treated with blood transfusions. When treatment is started early it is usually not necessary to refer typhoid patients.

6. Prevention and control

General

As for diarrhoeal diseases (see bacillary dysentery).

Carriers

Search for carriers is impracticable in endemic areas of Africa. Repeated cultures are necessary and/or sophisticated immunological techniques. Carriers who work as food handlers are especially likely to transmit enteric fever. Patients and family contacts should not be employed as food handlers until the danger of transmission is over.

Immunization

A combined vaccine against typhoid and para-typhoid was available as TAB vaccine; but now a single-strain *S. typhi* vaccine is used.

The vaccine does not give complete protection, but the number of infecting organisms necessary to cause typhoid fever is higher in immunized than in non-immunized persons. Immunization should be repeated yearly. In the control of typhoid the vaccine is of little value but as individual prophylaxis, especially for health workers in endemic areas, it is useful. It has been most used by the armed forces. However a good water supply and improved food hygiene are the best control measures.

7. Action

See Introduction to this chapter.

8. Summary

The enteric fevers are systemic diseases. Typhoid fever is characterized by high continuous fever and malaise. Paratyphoid fever may resemble typhoid fever or gastro-enteritis. Both diseases are spread by infected

water and contaminated food, through the faeco-oral transmission route.

Chloramphenicol is the drug of choice.

Control depends mainly on sanitary disposal of faeces and improvement of water supply.

FOOD POISONING

1. Food poisoning is a term applied to an acute intestinal disease acquired by the consumption of food or water. The cause may be an intoxication with chemicals (heavy metals, fluoride, and others), toxins produced by bacterial growth, and a variety of organic substances that may be present in natural food, e.g., mushrooms. Acute salmonellosis and intestinal anthrax are often regarded as food poisoning although these are more of acute enteric infections than intoxications. See also Chapter 7.

2. Occurrence and importance

Food poisoning occurs in small outbreaks; mortality is very low. Family members or institutions are usually affected. Figures for Africa are not available. At least some of the cases reported as gastro-enteritis may well be due to one or other form of food poisoning. Food poisoning is usually recognized when all members sharing the same food, members of one household, those eating in one restaurant or travelling on an aeroplane, fall sick within a short time.



Figure 5.15 Food poisoning occurs among people who share the same meal

3. Epidemiology

Food poisoning may be a real intoxication or an infection. Intoxication may be caused by ingestion of food contaminated with toxin-producing staphylococci from purulent discharges of an infected person, e.g., a septic finger. The staphylococci multiply when food is allowed to stand for several hours before serving. Infection may be caused by ingestion of salmonellae in food contaminated by infected faeces of man or animals, or ingestion of meat containing anthrax bacilli.

Reservoir

- *Staphylococci:* man only;
- *Salmonellae:* domestic and wild animals (especially rats and poultry);
- *Anthrax:* cattle and game.

4. Clinical picture

Incubation period is short:

- Staphylococcal food poisoning: 1–6 hours.
- Salmonella food poisoning: 12–14 hours.

Acute onset of vomiting and diarrhoea after ingestion of made-up food; usually a number of cases occur together. There may be moderate fever, seldom over 38°C (see also page 189, Cholera, differential diagnosis). Complications are rare but children with sickle cell disease may develop a salmonella osteomyelitis after salmonella food poisoning.

Gastro-intestinal anthrax has an incubation period of 2–5 days when vomiting, abdominal pain, haematemesis, bloody diarrhoea and toxaemia develop. Mortality rates can be high. Treatment is with large doses of benzyl penicillin together with streptomycin.

5. Management of the individual patient

Symptomatic treatment; correct dehydration; antibiotics are not indicated except for anthrax.

6. Prevention and control

- Serve meals immediately after preparing to avoid growth of staphylococci accidentally introduced.

- Exclude people with pyogenic (staphylococcal) skin infections from food handling. Search for them in case of an outbreak.
- Cook foodstuffs derived from animals thoroughly. Avoid the use of raw eggs.
- Ratproof all food stores.
- Give health education to food handlers (housewives and shopkeepers) about the necessity of refrigerating foods, washing hands, and maintaining a clean kitchen.
- Educate communities about not eating animals that have died after illness.

Note
The toxin produced by staphylococci is heat-stable, so cooking of already prepared food will kill the staphylococci but will not break down the toxin unless the food is heated to over 140°C. Thorough cooking of food will prevent all cases of *Salmonella* food poisoning.

7. Action

- Trace all persons who ate the infected meal and treat them if necessary.
- Try to establish the cause: staphylococci, salmonellae, or anthrax? Watch out for neurological symptoms suggesting insecticide poisoning, mushrooms. Rehydrate all patients, reassure or sedate the patients who panic.

8. Summary

Food poisoning occurs among people who share the same meal. Causes are intoxication or infection. Rehydration is the first priority. Reassure or sedate when necessary.

POLIOMYELITIS

1. Poliomyelitis is an acute viral disease of the nervous system with a wide range of clinical presentations, from symptomless infection to paralytic disease.

Synonym: Infantile paralysis; abbreviated, polio.

2. Occurrence and importance

Infections with the polio virus are very common in Africa. Almost all adults will have had it at some time in their childhood, just as every adult will have had measles as a child. Like measles, polio infection gives a life-long immunity. Infection below the age of 3 years rarely results in paralysis, and because most people are infected at a very young age only a few develop paralytic complications with permanent disability. In Africa many children are exposed to the infection at a young age because of:

- Poor hygiene;
- Large families and overcrowding;
- Lack of immunization.

The risk of paralysis increases with age in unimmunized persons. People who are infected after the age of 20 years have a much greater chance of getting paralysis (one in fifty, 1:50). As standards of living rise, polio infection will occur at a later age and an increased number of paralytic cases can be expected if there is not adequate immunization. Paralysis is caused by the polio virus invading the CNS. This invasion is easier when nerve endings are damaged, e.g., by intramuscular injections, muscular exhaustion (sports, long walks), and operations like tonsillectomy.

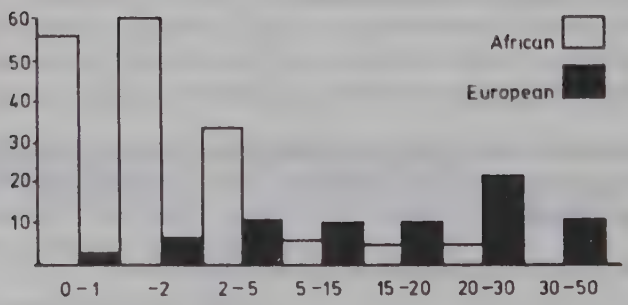


Figure 5.16 **The increasing risk of paralysis with age**

3. Epidemiology

There are three types of polio virus. The virus is detectable both in faeces and pharyngeal secretions but it is still not sure whether the faeco-oral route or the oral-oral route is most important.

Reservoir of infection

Persons with symptomless infections, mostly children.

4. Clinical picture

Infection with polio virus usually results in a mild general reaction; this is known as the 'minor illness'. There is fever, headache, malaise, sore throat, and gastro-intestinal disturbance lasting 1 or 2 days. The minor illness cannot be differentiated from other mild viral infections and is only recognizable during an epidemic of polio.

After the minor illness, the disease sometimes progresses into the "major illness" with a recurrence of fever. The major illness can be divided into three stages.

Pre-paralytic stage

The temperature rises rapidly to 39–40°C with headache and signs of meningeal irritation. The general symptoms return: malaise, vomiting, anorexia, diarrhoea. After 1 or 2 days the symptoms disappear. The patient may recover or may go on to the next, paralytic stage. Physical activity at the time of temporary improvement may aggravate the degree of subsequent paralysis.

Paralytic stage

As the temperature goes down the paralysis appears. Paralysis can appear at any site of the body and is asymmetrical. The lower limbs are more often affected than the upper. Spread of paralysis is usually completed in 24 hours. The paralysed muscles are painful.

Post-paralytic stage

(Stage of residual disability.) Paralysis or weakness of muscles will lead to deformity and contractures. A severely affected limb will show effects of abnormalities in blood circulation such as coldness

and cyanosis. There may be retardation of bone growth, resulting in shortening of the affected limb.

5. Management of the individual patient

No specific drug is available as poliomyelitis is a viral infection. Absolute bed rest is necessary in the pre-paralytic stage until it is certain that paralysis will not develop. All injections should be avoided for fear of precipitating paralysis.In the paralytic stage mobility by passive movements of the affected limb must be maintained to prevent contractures. In the post-paralytic stage of the disease active physiotherapy should be started. Physiotherapists can help in assessment of disability and deformity, with active exercises and passive stretching of contractures, and by measuring for braces (calipers) and gait training. Patients may be isolated. It is essential to immunize all contacts and ensure high coverage in the community.

In many rural communities, there are, however, unfortunate cases that have occurred before immunization was available or before mothers became aware of the advantages of immunization. Unless immunization is started at birth, there will be a proportion of cases that will occur in the first 5 months of life. For those patients that contact poliomyelitis the health workers in a community have a responsibility to help mothers with the rehabilitation of crippled children. A disability should not be allowed to progress into a social handicap. Every effort must be made to bring up the child in as normal a way as possible. This includes play with others, walking upright and going to school. Health workers need to teach families how to construct low-cost aids such as wooden parallel bars for practising walking, supporting seats and crutches. The Ministry of Health can send physiotherapists and occupational therapists to start local community-based rehabilitation schemes. Assessment of individual paralysed children could also be facilitated through referral from the health centre. Some children need special exercises, calipers, boots and surgical correction of deformities such as contractures. These specialized services are only found in large hospitals — hence the justification for referral.

6. Prevention and control

Immunization

The vaccine available is the Sabin vaccine, a live attenuated vaccine

given orally. The disadvantage of the Sabin vaccine is that when another enterovirus is present in the bowel it can compete with vaccine. For this reason it should be given when there is diarrhoea, and then repeated when the child is well.

Polio vaccine contains three types of virus. When it is given, usually one strain becomes dominant and the antibody response will be mainly against that type. Therefore the vaccine has to be given four or more times, at intervals of at least a month.

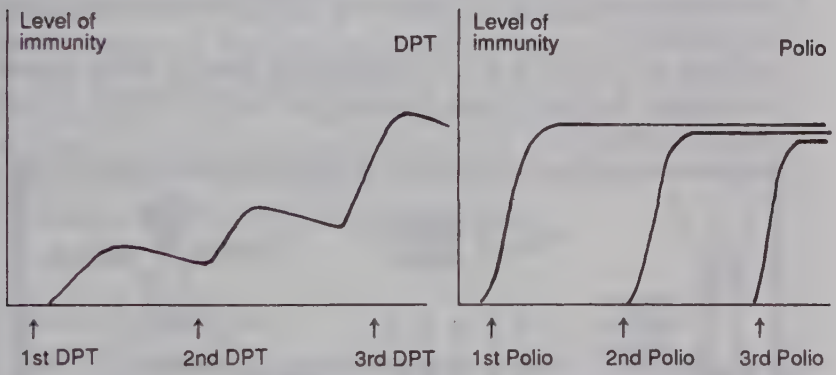


Figure 5.17 Levels of immunity from DPT and polio immunization

Note

The second and/or third injections of DPT are needed to boost the response to the vaccine as a whole. The second and subsequent polio feedings are to make sure all three strains get a good chance to 'take' as probably only one will 'take' at each feeding, and quite often one will not take.

Polio vaccine is given routinely to all under-fives, starting the immunization at birth and then at 6 weeks (together with DPT 1), again at 10 weeks (with DPT 2), and at 14 weeks (with DPT 3). It can also be given to schoolchildren and even to adults in case of an epidemic.

Since 1969, the incidence of polio has continued to fall in many parts of Africa as a result of immunization programmes. There is a danger, however, if not all children are routinely immunized against polio, that an epidemic can break out again. This will not happen if 80% of all individuals have immunity (high herd immunity). This can be achieved by continuous routine immunization of all the under-one-year olds.

Notification

Poliomyelitis is a notifiable disease. Inform your DMO when you suspect an outbreak or when you are confronted with a new paralytic case.

Other measures

In case of an outbreak of poliomyelitis:

- Start mass immunization with oral vaccine immediately;
- Minimize prescriptions for injections. DPT immunizations should be postponed;
- Individuals should not tire themselves: should avoid games, swimming, and heavy work, especially if they feel a little un-well ("minor illness").

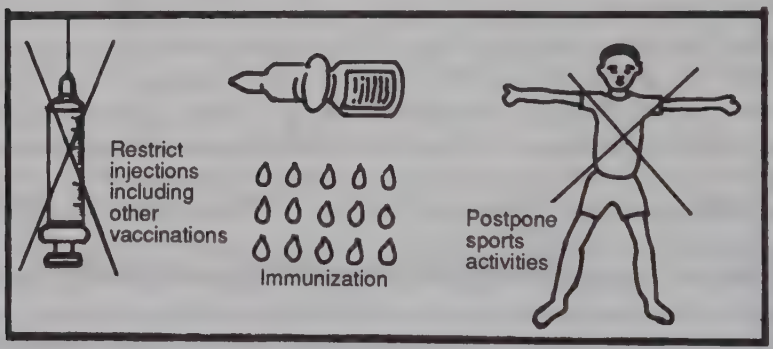


Figure 5.18 **Measures to be taken in case of an outbreak of poliomyelitis**

7. Action

General

- Check your MCH clinic. Are polio immunizations carried out routinely? Is the refrigerator working as it should? Are there wasted opportunities to complete each infant's full immunization?

Outbreak of polio

- Inform your DMO;
- Start mass immunization of all under-fives;
- Postpone other immunizations (injections);

- Restrict use of all injections;
- Advise people, especially teachers, to postpone sports activities and other exercises;
- Refer paralytic patients for physiotherapy as soon as the acute stage (pain and fever) is over.

8. Summary

Poliomyelitis is an acute viral disease which can be complicated by CNS involvement. Transmission is by close contact. Previously most children have been infected at a young age resulting in relatively few cases of paralysis. Now almost all children should be immunized.

Paralytic cases need physiotherapy. Polio can be prevented by immunization, and the World Health Organization Assembly has resolved to eradicate poliomyelitis from the world by the year 2000. Each suspected case must be referred to hospital for detailed investigation and laboratory confirmation.

VIRAL HEPATITIS

1. Viral hepatitis is a systemic disease predominantly affecting the liver. It is characterized by a constitutional upset followed by jaundice, although there are many asymptomatic cases. There are now four recognizable types of viral hepatitis:

- Hepatitis A
- Hepatitis B
- Delta hepatitis
- Non-A, non-B hepatitis.

All four types produce similar clinical disease. This may range from asymptomatic infection to a fulminating, fatal acute infection. Delta hepatitis has been discovered recently.

2. Occurrence and importance

Occurrence and importance of each of the diseases is difficult to estimate because it is usually impossible to tell the difference between the types on clinical grounds. In general the case fatality rate in hepatitis A and B is very low (about 0.1%), but this increases with

advanced age and debilitating disorders. In patients ill enough to be hospitalized with acute hepatitis B, the fatality rate is 1%. Case fatality rates for non-A, non-B are not precisely known, but are also very low, except in epidemics of water-borne non-A, non-B hepatitis where the case fatality rate may reach as high as 10%. The case fatality rate for simultaneous delta hepatitis and hepatitis B may range from 5 to 20%.

3. Epidemiology

Hepatitis A

Hepatitis A occurs in slowly spreading epidemics in families and institutions. Epidemics can be traced to contaminated food, water or milk. Poor personal hygiene, overcrowding and poor sanitation increase the transmission which is mainly via the faeco-oral route. Most children in African countries are infected at a young age. The disease is very mild in children, presenting as gastro-enteritis, without jaundice. The resulting immunity is long-lasting. When children escape infection at an early age, they are more likely to develop jaundice when they are infected later in life. Most cases of infectious hepatitis are therefore diagnosed in older children and young adults.

The infectious agent is excreted in faeces and urine. Most probably it is also present in nasal and pharyngeal discharges. The main way of transmission is, however, by faecal contamination of water or food.

Hepatitis B

The disease has a worldwide distribution, but is particularly common in sub-Saharan Africa, being maintained by the high perinatal transmission rate, and intimate contact between toddlers.

The hepatitis B virus is now known to be transmitted through several routes which can be broadly classified as percutaneous and non-percutaneous. The percutaneous route includes transmission via injections and blood and blood products. The non-percutaneous route includes transmission through close personal contact, as in kissing and sexual intercourse, and from mother to baby across the placenta or in the birth canal (perinatal transmission). The non-percutaneous

route of transmission is probably the most important in the spread of hepatitis B. Reservoirs of infection are the chronic carriers of the virus, sexually promiscuous individuals, spouses of acutely-infected persons, healthcare workers exposed to blood, family members of chronically-infected persons, toddlers, and persons who require repeated blood transfusions. Carrier rates are very high in Senegal and Somalia, and may reach 5– 20%. Carriers can be identified by the presence in blood of hepatitis B surface antigen (HBsAg) which used to be known as Australia antigen. Chronic active hepatitis occurs in 1–3% of cases and may be followed some 10 years later by liver cirrhosis in 10–20% of cases. In some cases cirrhosis progresses to frank hepatocellular carcinoma.

Delta hepatitis

This newly discovered type of hepatitis is caused by a defective RNA virus which requires the help of hepatitis B virus to cause successful infection. It has a worldwide distribution and will be common in areas where hepatitis B is common. It is transmitted through blood and blood products, and through close personal contact. Since hepatitis B has a carrier state, it is conceivable that delta hepatitis may also have a carrier state. Delta hepatitis can make asymptomatic or mild chronic active hepatitis B worse and lead to cirrhosis.

Non-A, Non-B hepatitis

Incubation period: 1–4 weeks.

This accounts for a large proportion of post-transfusion hepatitis, and so its major transmission route is via blood and, particularly, blood products. It is also transmissible through close personal contact, either with infected persons or blood and blood products. An epidemic form of non-A, non-B was identified in India in which the mode of transmission was the faeco-oral route.

Chronic active hepatitis may complicate up to 50% of cases of post-transfusion non-A, non-B hepatitis. Of these up to 20% may develop cirrhosis within 10 years.

Though the clinical features, management, control and action required for the different types of hepatitis have much in common, the two main types are described separately to emphasize the differences.

Hepatitis A

For general information on occurrence, importance and epidemiology, see Viral hepatitis above.

4. Clinical picture

Incubation period: 1–4 weeks.
Pre-icteric phase: presents like a gastro-enteritis with sudden onset of fever, malaise, anorexia, nausea, and abdominal discomfort. Children often do not go into the next phase.
Icteric phase: after a few days jaundice appears. The jaundice is partly of the hepato-cellular type and partly obstructive, because the swollen liver cells block the bile flow in the bile capillaries. Itching is not usually present.

Complete recovery in about 2 weeks is the rule, but a long period of extreme tiredness with depression may occur after the jaundice has disappeared.

Diagnosis

Stools are usually pale. The urine is dark and contains bilirubin. WBC total and differential are normal. There is no albuminuria. In blood, both direct and indirect bilirubin are raised. A blood slide should be taken to exclude other causes of jaundice such as malaria and relapsing fever.

Table 5.6 **Differential diagnosis of jaundice and fever**

	Blood slide	WBC total	Albuminuria
Viral hepatitis	Negative	Normal	No
Yellow fever	Negative	Low	Yes
Relapsing fever	Positive	Raised	Yes
Malaria	Positive	Low	No

5. Management of the individual patient

No specific treatment is available, therefore there is no strict reason for admission to hospital. Bed rest at home is best and will prevent spread of the disease. If admission for one or other reason is indicated, isolate the patient. Patients should be advised to eat a high calorie diet. Fat can be restricted when there is nausea. Vitamin B is often prescribed but has no proven value. Alcohol should be avoided for at least 6 months. Corticosteroids are not of any proven value even in fulminant cases.

6. Prevention and control

Improvement of sanitation will diminish transmission of hepatitis A. Injection of pooled gamma-globulin can protect travellers for periods of about 3 months.

7. Action

- Isolate admitted patients with hepatitis.
- Improve food hygiene and water supplies.
- Health education on hand washing.

8. Summary

Viral hepatitis A is characterized by abdominal discomfort and malaise, most often followed by jaundice. No specific treatment is available. Control is difficult since epidemiology is not fully understood but environmental sanitation, personal hygiene and food hygiene are important.

Hepatitis B

For information on occurrence, importance and epidemiology see Viral hepatitis above.

4. Clinical picture

Incubation period: 12 weeks or longer.

Most infections in infants and young children go unnoticed. Many carrier women do not know they can pass the infection on to their children except in rare cases of acute hepatitis during pregnancy.

When hepatitis B follows blood transfusion or parenteral infection in an adult, it can be severe, can occur after a long incubation period, of about 3 months, and can progress to liver failure.

The more common problem is chronic hepatitis progressing to cirrhosis or hepatocellular carcinoma.

Diagnosis

The differential diagnosis of hepatitis B with jaundice would include hepatitis A, yellow fever, malaria and relapsing fever. Diagnosis is made on various immunological markers. The most important is the hepatitis B surface antigen (HBs Ag) which is present when the virus is in the blood in the acute stage and in the chronic carrier state.

5. Management of the individual patient

There is no specific treatment. Bed rest at home is sufficient in mild cases. Admission is required in severe cases, so refer. Alcohol is strictly forbidden because it increases the risk of cirrhosis.

6. Prevention and control

Blood for transfusion must be screened for hepatitis B antigen. Donors with a history of jaundice should be excluded. All instruments and needles and syringes should be heat-sterilized preferably by steam under pressure. In future in areas with a high carrier rate — greater than 10% — neonates will be given HBV vaccine. In wealthy countries mothers are screened and infants of HBsAg positive mothers are given hepatitis B immune globulin and HBV vaccine.

7. Action

- Take great care with sterilization of needles and syringes.
- Use screened blood for transfusion.
- Consider immunization in endemic areas with high carrier rates.

8. Summary

Hepatitis B virus is now considered the cause of hepatocellular carcinoma which follows a chronic carrier state often acquired in early childhood.

Chapter six

Helminthic Diseases

INTRODUCTION

The worms of medical importance in many parts of Africa can be divided into three groups according to their form, as in Table 6.1.

Table 6.1 Classification of worms of medical importance in Africa

Group	Name	Particulars
Nematodes or Roundworms	*Ascaris lumbricoides*	Lung passage
	Strongyloides stercoralis	
— cylindrical and elongated	*Ankylostoma duodenale*	Hookworms
	Necator americanus	
	Trichuris trichiura	Whipworm
	Enterobius vermicularis	Pinworm or threadworm
	The filarial worms	Insect vectors (see Chapter 4, page 115)
Cestodes or Tapeworms	*Taenia saginata*	Have an intermediate host (cattle)
—flat and segmented	*Taenia solium*	Pig intermediate host
	Echinococcus granulosus	Dog tapeworm (causing hydatid disease)
Trematodes or Flukes — leaf-like or cylindrical	*Schistosoma mansoni*	Snail intermediate host (see Chapter 4, page 140)
	Schistosoma haematobium	

Hookworm, ascaris, trichuris and strongyloides are all soil-transmitted helminths. Enterobius is transmitted directly by the faeco-oral route and taenia has an intermediate host.

Filarial diseases and schistosomiasis are dealt with in the chapter on vector-borne diseases. The final host of all worms in the table is man, except for the dog tapeworm for which man is an accidental intermediate host. The eggs of all the intestinal worms are excreted in stools. Sanitary disposal of human faeces is the preventive measure of choice because it will control these worm diseases (except hydatid disease, enterobius and schistosomiasis).

Intestinal worms are a problem of poor sanitation

Proper disposal of faeces is one of the most difficult preventive measures to achieve because the co-operation of every member of the community, including children, is necessary. When even one person does not dispose of his stools properly, transmission may continue. Building of latrines is of no use if they are not used properly. Health education has to be given to the people so that they can change their attitudes and behaviour about human waste disposal. Because of the lack of proper latrines and the attitudes of people, it will take some time before worm diseases are controlled in many parts of Africa.

As most worm infections are symptomless, or treated at health centres or dispensaries, there are few figures on their real prevalence in communities. It appears from available evidence in Kenya that helminths are as prevalent today as they were 60 years ago, in spite of limited efforts to control them.

Hookworm prevalence is dependent on climate because the larvae require moist warm soil for development. Therefore, hookworm is more frequent in tropical coastal regions in eastern Africa. Prevalence rates of up to 60% are often found in such areas.

Ascaris infestation does not appear to depend so much on the climate, although it is more perennial in the damp humid areas of the tropics. It is found in most areas where sanitation is poor. It is likely that on an average 25% of the rural population of any one country may be infected with ascaris. The prevalence may even be higher in some of the over-crowded cities of the third world than in the rural areas.

Taenia is also widely spread, with perhaps a higher prevalence in the cattle-keeping areas of the savannah from where meat comes.

Much of the transmission of helminths is through children who often do not use latrines to the same extent as their parents. Most of the helminthic diseases occur in children, except perhaps for hookworm anaemia and taeniasis which equally affect adults.

ASCARIASIS

1. Ascariasis is one of the commonest and most widespread nematode infections of the small intestine. Symptoms are usually vague or absent.

Synonym: Roundworm.

2. Occurrence and importance

Ascariasis is common in all areas of Africa. Children are more frequently and more heavily infected than adults because of their habit of putting all kinds of things in their mouths. Adults may develop some immunity.

3. Epidemiology

Ascariasis is caused by *Ascaris lumbricoides*, a large intestinal roundworm. The roundworm lives in the small intestine. A female may produce up to 200,000 eggs daily.

The eggs are passed out in the faeces. They have to be embryonated in soil before they are infective. This takes 8–50 days. The soil must be loose and not too dry. Oxygen must be available and the temperature over 15°C. But eggs can survive adverse circumstances for a long time, and embryonated eggs can be carried away from the contaminated place into houses by feet, footwear or in dust by wind. They can also pass through the gastro-intestinal tract of animals and remain infective.

Only when swallowed by a human being do the eggs hatch in the intestinal canal. The usual vehicle is fruit or other food eaten raw. Unwashed hands, and children picking up things from the floor or ground and putting them in their mouths, are common ways of acquiring ascaris.

To reach maturity, the larvae need to pass through the lungs. The

larvae penetrate the intestinal wall and reach the liver via the portal system. From the liver they are carried through the right side of the heart into the lungs. Here they penetrate into the airways and pass via the bronchioli, bronchi, and trachea to the pharynx. Then they are swallowed, return to the gastro-intestinal tract, and settle in the jejunum. They become mature in 2 months and can live for about a year.

First passage through gastro-intestinal tract

Liver, heart and lung passage

Second passage through gastro-intestinal tract

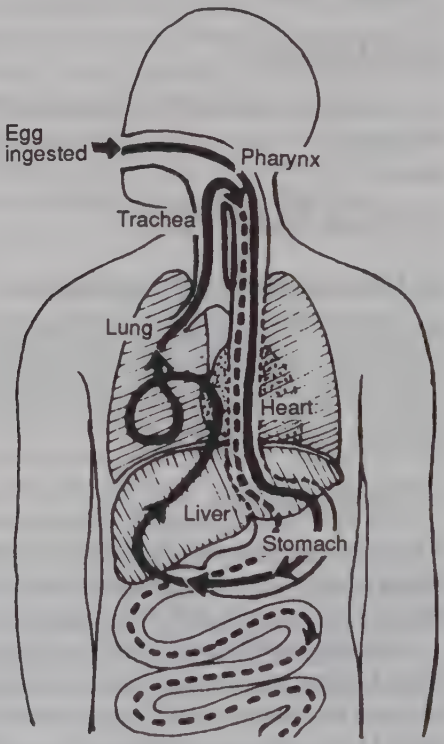


Figure 6.1 **Lung passage of ascaris larva**

During the lung passage eosinophilia develops. This eosinophilia is temporary if no new infection occurs. The migration phase may be associated with fever, cough, and allergic dermatitis. Migration may also cause pneumonitis.

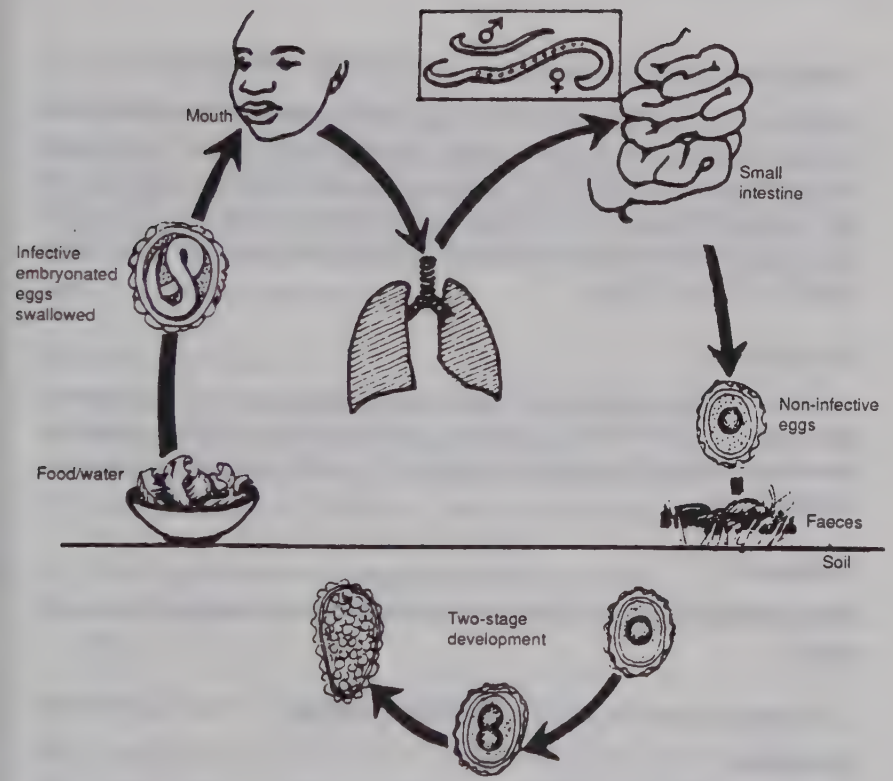


Figure 6.2 **Life cycle of *Ascaris lumbricoides***

4. Clinical picture

Except for the temporary symptoms during larval migration through the lungs, infection with a few ascaris is usually symptomless or, if symptoms are present, they are not characteristic.

There may be vague abdominal discomfort. Occasionally a worm may leave the body (in vomitus or stools) upsetting the patient and his family.

Complications may occur in very heavy infections or are due to wandering worms.

Intestinal obstruction

This may occur at the ileo-caecal junction by a tangled ball of worms. Obstruction may be partial or complete.

Wandering of the worms

This may be provoked by TCE (a drug used previously for hookworm treatment). Wandering ascaris may reach abnormal foci and cause acute symptoms: vomiting of worms may cause swelling of the glottis and larynx, resulting in difficulties in breathing; blockage of the bile ducts may cause obstructive jaundice; migration into liver tissue may result in a liver abscess.

Malnutrition

Ascariasis may contribute to malnutrition states such as kwashiorkor and vitamin A deficiency. After de-worming there is often catch-up growth showing the degree to which the worm load contributed to malnutrition.

Diagnosis

By stool microscopy, which should show the characteristic ascaris eggs.

5. Management of the individual patient

Treatment

(a) Mebendazole is now the drug most commonly supplied in drug kits of the Essential Drug Programme because it is a broad-spectrum anthelminthic. It is given in a dose of 100 mg twice daily for 3 days.
(b) Levamisole (Ketrax) three tablets as a single dose (5mg/kg).
(c) Piperazine (Antepar) syrup 150 mg/kg single dose to a maximum of 4g.

Intestinal obstruction

Refer all patients suspected of obstruction. Surgery may be necessary, but it is possible to try medical treatment before that. The symptoms of intestinal obstruction may resolve with anthelminthic treatment and intravenous fluids. If not, surgery is then indicated.

6. Prevention and control

Environmental measures

Provision of adequate and safe water supplies, facilities for safe faeces disposal, and prevention of faecal contamination of food.

Discourage use of fresh human faeces for manuring. Composting for 6 months will kill ascaris eggs. After this time the compost can safely be used as fertilizer.

Health education

- The need for and the proper use of latrines and toilets.
- Washing of hands before handling food.
- Avoidance of food which has fallen on the floor.
- Proper disposal of childrens' faeces into latrines.
- Training of children to use the latrine.
- Washing of fruits and vegetables before eating.
- Use of drying-racks for utensils so that they are above the soil and dust.

Mass treatment of infected persons

Mass treatment is not beneficial unless continued until transmission rates have been reduced to very low levels, and this exercise is combined with proper disposal of faeces. Periodic de-worming of children whose growth is not satisfactory is done in some clinics in areas where ascariasis is endemic.

7. Action

- Make sure that your health centre has a well maintained pit latrine, and that it is properly used.
- Give health education on the use of latrines.
- Inspect the latrines in schools and markets in your community; check on their use and cleanliness. Report your findings in the area development committee meeting.
- Stress to the local development committee the importance of latrines for individuals and families.
- Ask the committee to make materials available for constructing latrines.

- Build, in co-operation with the villagers, a demonstration VIP latrine in a suitable place as an example of proper construction.
- Try to find out why latrines are not being used. Find out about local taboos. Concentrate your health education on changing any unhygienic traditional behaviour.

8. Summary

Ascariasis is a common nematode infection. Its prevalence is related to poor sanitation and hygiene. Control is mainly by improving faeces disposal. A heavy infection in a child can contribute to undernutrition.

ENTEROBIASIS

1. Enterobiasis is a benign intestinal disease caused by *Enterobius vermicularis*.

2. Occurrence and importance

The disease has a worldwide distribution, but is most common in temperate zones, especially in school children. Whole families may be infected. It is common in boarding institutions.

3. Epidemiology

Enterobiasis is caused by *Enterobius vermicularis*, the thread or pinworm. Initial infection occurs by the faeco-oral route. Infection is maintained by direct transfer of infective eggs from the anus to the mouth (auto-infection), or indirect faeco-oral contact through clothing, bedding, food, and other articles. Airborne infection through inhalation of dust containing eggs and consequent swallowing is possible.

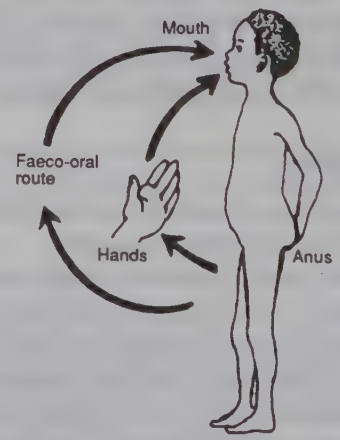


Figure 6.3 **Transmission of enterobiasis**

After ingestion, the eggs hatch in the stomach and small intestine. Young worms mature in the lower small intestine and upper colon. Gravid worms migrate to the rectum to discharge eggs on the peri-anal skin, especially during the night. This causes itching and consequent scratching. Auto-infection is then easily established. One life cycle takes 3–6 weeks.

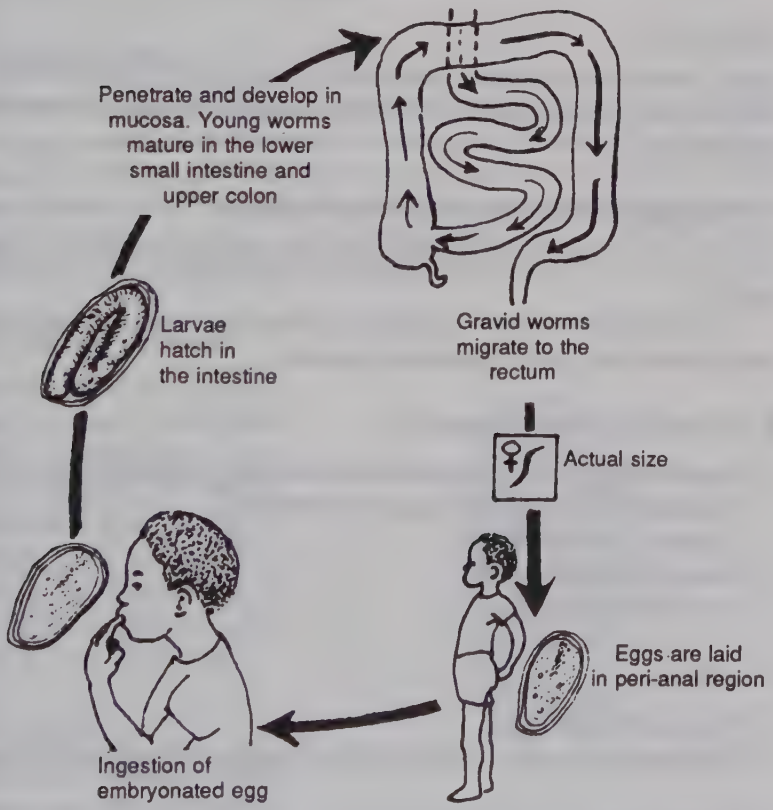


Figure 6.4 Life cycle of *Enterobius vermicularis*

4. Clinical picture

There is no lung passage, and no intensive tissue contact with the worm, therefore no eosinophilia. Pruritus ani is the main symptom, and this provokes intense scratching of the peri-anal region, resulting in excoriation and secondary bacterial infection. General symptoms include disturbance of sleep, restlessness, loss of appetite and weight loss. Vulvitis and appendicitis may occur.

Diagnosis

Apply transparent adhesive tape over the anus in the early morning, stick the tape onto a slide, and search for typical eggs under the microscope.

5. Management of the individual patient

Mebendazole: 100 mg as a single dose for all ages. Treat the whole family.

During treatment strict precautions should be taken to avoid auto-reinfection (see below: personal hygiene).

6. Prevention and control

- Personal hygiene: bathing and handwashing. Cut nails short; wash underclothes, night clothes and bedclothes.
- Correct overcrowding.
- Proper faeces disposal.
- Treat whole family.

7. Action

- Treat whole families.
- Give health education to infected individuals to prevent re-infection.

8. Summary

Enterobiasis is a benign intestinal nematode disease. Auto-reinfection is common. Control is by personal hygiene and treatment.

TRICHURIASIS

1. Trichuriasis is a nematode infection of the large intestine. It is usually asymptomatic.

Synonym: Whipworm.

2. Occurrence and importance

Common in hot, humid climates.

3. Epidemiology

Trichuriasis is caused by *Trichuris trichiura*. Transmission is indirect, as eggs passed in the faeces require embryonation in soil (as in ascaris). Therefore, auto-infection is not possible. Infection occurs in a similar way to ascariasis. When embryonated eggs are ingested, they hatch. The mature worms attach themselves to the mucosa of caecum and colon. There is no lung passage.

4. Clinical picture

Eosinophilia is present only in heavy infections. Mild infections are symptomless, but heavy infections may result in abdominal discomfort, bloody diarrhoea, loss of weight, anaemia, and prolapse of the rectum.

Diagnosis

By stool examination. More than 200 eggs in an ordinary faecal smear indicates heavy infection.

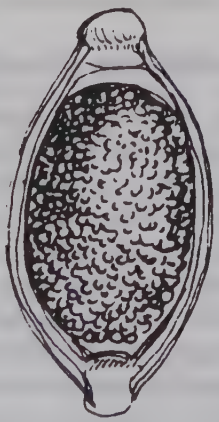


Figure 6.5 Egg of *Trichuris trichiura* as seen in a faecal smear

5. Management of the individual patient

Mebendazole 100 mg twice or three times daily for 3 days. Mild infections do not need treatment.

6. Prevention and control

As for ascariasis: sanitary disposal of faeces, personal hygiene.

7. Action

As for prevention and control.

8. Summary

Trichuriasis is a mild nematode infection transmitted by the faeco-oral route. It can be controlled by personal hygiene and sanitation.

HOOKWORM

1. Infection with hookworm may vary from symptomless infection to a chronic debilitating disease with a variety of symptoms. This depends on the extent of the infection, the nutritional state of the patient, and the degree of anaemia caused.

Synonyms: Ankylostomiasis, hookworm disease.

2. Occurrence and importance

Hookworms need a hot humid climate for their development. A minimum temperature of 18°C is required, and a soil temperature of 20–32°C is optimal. Hookworm infection is most common in the hot humid areas of Africa. Many people harbour hookworms without any ill effect (hookworm carriers). The nutrition, the daily iron intake, and the total worm load determine whether a carrier develops hookworm anaemia or not. Hookworm anaemia is one of the important causes of anaemia in many communities. Anaemia has a profound effect on the working capacity and the well-being of the individual. The economic loss caused by anaemia is enormous but difficult to calculate. The normal Hb level in a healthy adult male ranges from 13 to 18 g/100 ml. An Hb of 10g/100 ml or less is a sign of disease and needs investigation and treatment (see Table 6.2).

Table 6.2 **Haemoglobin levels**

WHO standards for lower limits of normal haemoglobin (Standards at sea level, in g/100 ml. Increase for altitude: 0.4 g/300 m)		
Adult males	13	Females and children do have slightly lower Hb than adult males. They also have lower iron reserves and therefore suffer more easily from anaemia
Non-pregnant females	12	
Pregnant female	11	
Children — at birth	14–20	
— below 6 months	10	
— 6 months to 4 years	11	
— 5–14 years	12	
Any Hb below these levels indicates disease		

3. Epidemiology

There are two types of hookworm, both nematodes, *Necator americanus* and *Ankylostoma duodenale*. The eggs are already embryonated when passed out with the faeces. When faeces stand for a long time before examination the free larvae can be found.

The larvae leave the faeces and bury themselves in moist damp soil. These larvae are called rhabditiform and are not infective before they have changed into the sheathed filariform stage (about 5 days). The filariform larvae may attach themselves to grass or hide in the soil. As soon as they are touched by something they attach themselves to it. When this happens to be a human leg or foot, they penetrate actively through the skin, and reach the lungs via the venous system and the right side of the heart.

In the lungs they penetrate into the alveoli and are then carried up passively through bronchioli, bronchi, and trachea to the larynx-pharynx. Next they are swallowed and reach the duodenum 3–5 days after penetrating the skin.

The worms are attached to the mucosa with hook-like teeth in their buccal cavity. The whole cycle is complete in about 40 days.

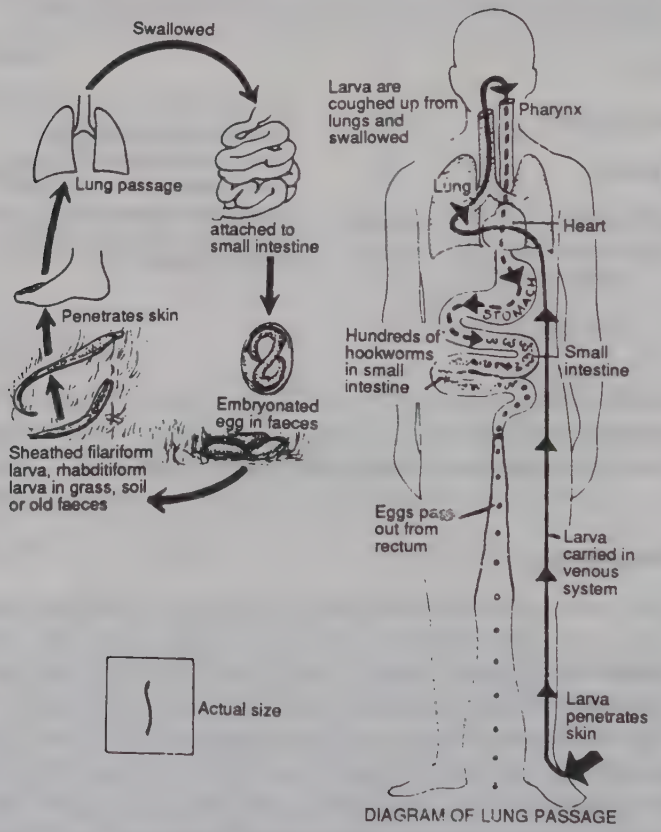


Figure 6.6 **The life cycle and transmission of hookworm**

4. Clinical picture

Hookworm infestation is asymptomatic in the vast majority of cases. Anaemia of the iron-deficiency type, however, develops when a heavy hookworm load overtaxes the iron reserves. The hookworms suck blood from the intestine and this leads to loss of red blood cells, iron, and protein, especially albumin, from the host. When the host's iron stores are depleted, iron-deficiency anaemia sets in, and symptoms related to the anaemia appear. The evolution of this anaemia is slow,

and because of the physiological adjustments it evokes the patient can continue to be up and about with a surprisingly low Hb, say, of less than 5g% — the so-called walking anaemia of hookworm.

Hookworm causes loss of iron

One *A. duodenale* causes a daily blood loss of 0.2 ml. One *N. americanus* causes a daily blood loss of 0.03 ml. A worm load of more than 100 *A. duodenale* or more than 500 *N. americanus* always causes anaemia.

In hookworm disease, firstly the body reserve of iron is used up, then gradually the Hb drops, sometimes to very low levels, with all the signs and symptoms of severe anaemia (fatigue, pallor, oedema, tachycardia, heart dilatation with murmurs, and eventually CCF).

Other signs

Ground itch: At the site where the larvae penetrate the skin itchy, erythematous papules appear. This is most common between the toes and on the back of the feet.

Lung passage: some coughing, eosinophilia.

Gastrointestinal tract: dyspe-psia, abdominal pain, distension, sometimes diarrhoea. In heavy infections, diarrhoea is mixed with blood. The symptoms may be mistaken for those of duodenal or gastric ulcer.

Diagnosis

Hookworm eggs in the faeces: Differentiation of *Necator* and *Ankylostoma* is not possible from the eggs, but the adult worms can be distinguished. More than 100 eggs in an ordinary faecal smear indicates a heavy infection.



Figure 6.7 **Embryonated egg of hookworm in faecal smear**

If the faeces have been standing for a long time, the eggs hatch and the larvae may easily be mistaken for larvae of strongyloides.

5. Management of the individual patient

Not all hookworm infections need treatment. Re-infection is very likely if the community as a whole does not improve its methods of faecal disposal. Where anaemia is present, treatment should aim at eliminating the worms and correcting the anaemia.

Iron deficiency

Anaemia is treated with oral iron (ferrous sulphate 200 mg three time a day for adults, once or twice a day for children for at least 2 months). A high protein diet is necessary to replace protein loss. Blood transfusion may be necessary when the anaemia is very severe. Refer such patients to hospital. Folate deficiency may occur as a result of increased bone-marrow activity when the iron deficiency is being corrected. Folic acid tablets 5 mg daily can prevent this.

De-worming

Light infections may not need treatment, especially where the risk of re-infection is high. Heavy infections have to be treated, and the most commonly used drugs are shown below.

(a) Mebendazole: 1–13 years, one tablet (100 mg) twice a day for 3 days.
14 years and over — two tablets twice a day for 3 days.

(b) Pyrantel pamoate in doses of 10mg/kg body weight daily for 3 days.

(c) Levamisole (Ketrax), dose: 3 tablets single dose. Advantage: can be used in mixed infections, not toxic. Disadvantage: more expensive. Not very effective against *Necator*, the more common hookworm.

(d) Bephenium (Alcopar), dose: one sachet, single dose. More expensive than levamisole and less effective with *Necator americanus* which is more common.

(e) Tetrachlorethylene (TCE). This is a very toxic drug which is no longer used, having been replaced by safer drugs.

6. Prevention and control

Shoes

Wearing of shoes will prevent infection, but usually shoes are not worn during work in the fields, or around the house. It is in the fields, on the way to school, in the compound, or near the rivers that the infection is acquired. The floor of a latrine that does not have a cement slab, may be heavily infected.

Wearing of shoes will only protect the individual who is able to buy and use the shoes properly. The transmission of hookworm in most African countries is likely to continue because not many people can afford shoes.

Faeces disposal

Proper disposal of faeces is the only way to eradicate hookworm infections (see ascariasis).

De-worming campaigns or mass treatment are only effective after the campaign for proper faeces disposal has been effectively implemented.

7. Action

- Faeces disposal: as for ascariasis.
- Health education on personal protection by wearing shoes.
- Health education on a balanced diet to prevent anaemia when iron intake is borderline.
- Mass treatment campaign only when everyone has and uses latrines.
- Regular examination, by mothers and health workers, of mucosae of children to detect anaemia.

8. Summary

Hookworm infection is caused by nematodes. Hookworm disease occurs when continued loss of iron results in anaemia. Anaemia occurs more rapidly when dietary iron intake is borderline. Treatment should aim at correcting the anaemia and eliminating the worms. Prevention in the individual is by wearing shoes. Control in the community is possible only through improvement of sanitation.

STRONGYLOIDIASIS

1. Strongyloidiasis is an infection caused by *Strongyloides stercoralis*, a nematode worm. The female adult worms live in the mucosa of the duodenum and jejunum. Most infections are without symptoms and signs.

2. Occurrence and importance

Geographical distribution is the same as in hookworm infection. Infections are not usually very severe, but extremely heavy infections (e.g. in AIDS or after immunosuppressive drugs) may result in death because of malabsorption syndrome and generalized strongyloidiasis.

3. Epidemiology

Strongyloides stercoralis resembles the hookworm in the appearance of adults, eggs, and larvae. In strongyloides infections, larvae are usually found in the stools. These larvae may either develop into free-living adults which continue to reproduce outside the body, or they may develop directly into infective filariform larvae which penetrate the skin. The rest of the cycle is similar to hookworm infection. Because of direct development into the infective stage, auto-infection is common. Even within the bowel, the larvae may become infective and penetrate the bowel wall. This is called endogenous re-infection.

4. Clinical picture

Usually the infection is symptomless. In very heavy infections the number of worms in the mucosa may interfere with the normal function of the bowel resulting in malabsorption and diarrhoea. The continuous re-infection may cause urticaria and other hypersensitivity reactions. There may be marked eosinophilia due to repeated re-infections by strongyloides.

Diagnosis

Larvae in fresh stool specimen.

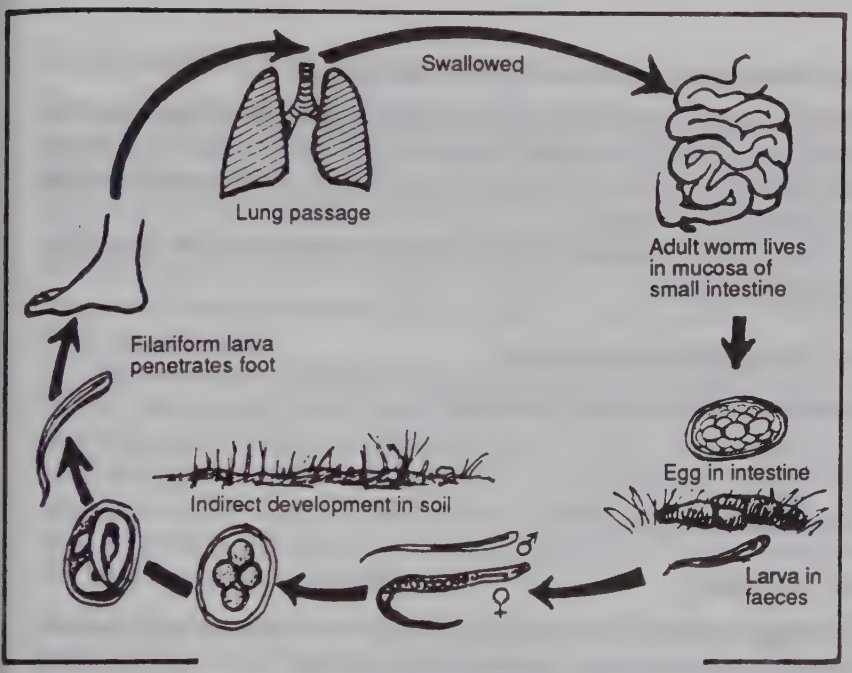


Figure 6.8 **Life cycle and transmission of *Strongyloides stercoralis***

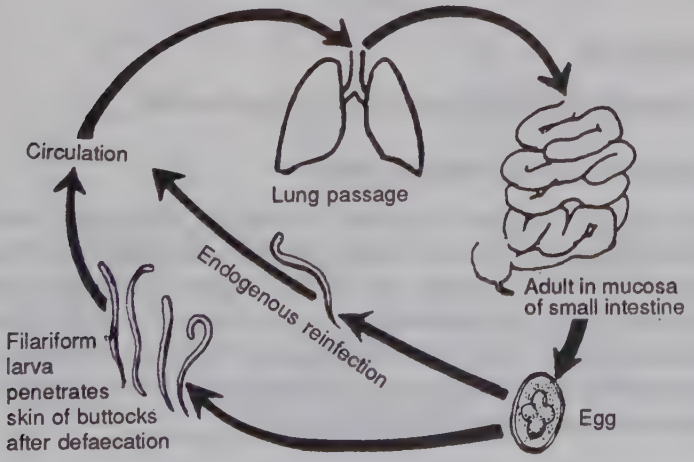


Figure 6.9 **Auto-reinfection cycle**

5. Management of the individual patient

The drug of choice is mebendazole 100 mg three times a day orally for 4 days. This drug is usually available in health centres in essential drug kits. An alternative drug is thiabendazole (Mintezol), 25 mg/kg twice daily for 2 days.

Levamisole (Ketrax) is only effective in about 50% of cases: dose as for hookworm.

6. Prevention and control

Same as in hookworm infections.

7. Action

See ascariasis page 219.

8. Summary

Strongyloidiasis is a nematode infection of the duodenum and jejunum. Usually symptomless, occasionally resulting in a malabsorption syndrome and potentially serious in AIDS. Treatment of choice is mebendazole. Control as for other intestinal helminths.

TAENIASIS

1. Taeniasis is an infection of man by tapeworms.

2. Occurrence and importance

Most cases of taeniasis reported in eastern Africa are caused by *Taenia saginata* (beef tapeworm). The disease is especially common in areas where beef is eaten raw or only lightly cooked. In southern and central Africa taeniasis due to *Taenia solium* (the pork tapeworm) has been reported. The cysticercus stage of *Taenia solium* in man can cause serious problems such as epilepsy, and death. The effects of tapeworms in the bowel are usually mild.

3. Epidemiology

The adult tapeworms live in the small intestine of man. Broken segments of the worm containing gravid uterus and/or the eggs are passed in the stools. The eggs are ingested by cows or pigs. In the gastro-intestinal tract of the animals, the embryos hatch and penetrate the bowel wall and are carried via the bloodstream to striated muscles. Here the larvae invaginate, grow, and form the infective cysts, called cysticerci. Pork or beef containing these is called "measly" pork or beef respectively.

When man ingests meat containing cysticerci, the cysts get dissolved by the gastric acid in the stomach to release embryos. The *Taenia saginata* embryo attaches itself to the wall of the small bowel by its head and grows into an adult worm. The *Taenia solium* embryo may also behave differently. It instead penetrates the bowel wall of man, as it does in the pig, and it is carried to organs like striated muscle or the brain by the blood stream.

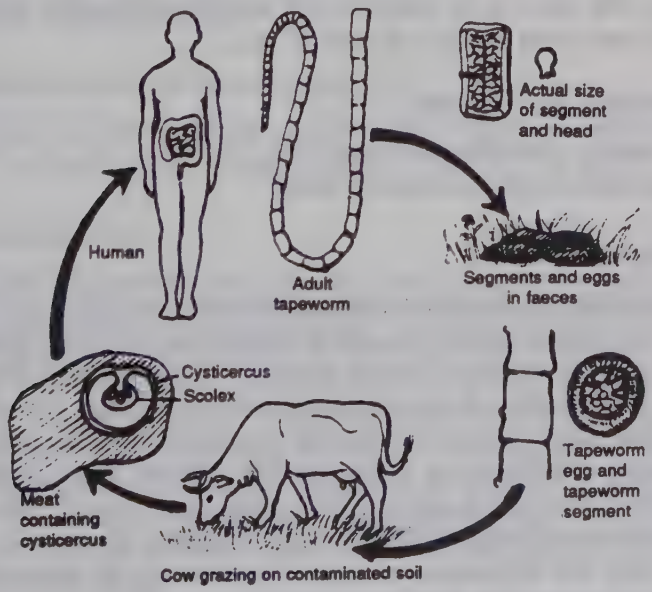


Figure 6.10 Life cycle and transmission of *Taenia saginata*

4. Clinical picture

Most infections with *Taenia saginata* cause no signs or symptoms. In some people they cause loss of weight, abdominal discomfort, and pruritus ani (itching around the anus). There is no eosinophilia. *Taenia solium* infections are dangerous, especially when the cysticerci invade the brain. Patients may present with epilepsy or other neurological signs, and muscular pains.

Diagnosis

The diagnosis can be made by the presence in the stool of the segments or eggs. The eggs are not laid singly and appear only accidentally in the stools. The segments can migrate out of the anus and be found on the buttocks, or they are passed out with the faeces.

5. Management of the individual patient

Niclosamide is effective against both *T. saginata* and *T. solium* infections. The dose is 1g chewed and washed down with water, followed 1 hour later by 1g — a total of 2g.

6. Prevention and control

Faeces disposal: see ascariasis, page 219.
Meat inspection: condemn infected meat.

Health education

Give health education about the dangers of eating meat (beef or pork) which is not cooked thoroughly. Deep freezing of meat will kill all cysticerci in 24 hours, but of course a whole carcass has to be frozen for about 21 days before all parts reach the correct temperature as meat is a good insulator. (Deep freezing will not give protection against other diseases which may be transmitted by infected meat, such as salmonellosis, brucellosis, and anthrax.) Control is also achieved by treating people and educating them on the importance of not polluting pastures with the segments and ova. Cysticercosis in animals is serious for the owners as the meat may be condemned.

7. Action

- See ascariasis for faeces disposal.
- Treat all cases, as they might infect animals.
- Give health education on the danger of eating raw or partially cooked meat.

8. Summary

Taeniasis is an infection with adult tapeworms. Infection is usually symptomless. Infection is exclusively acquired by eating partly raw meat. Treatment is with niclosamide. Control depends on meat inspection, and proper faeces disposal.

HYDATIDOSIS

1. Hydatidosis is a disease caused by the cysts of the dog tapeworm, *Echinococcus granulosus*.

Synonym: Echinococcosis.

2. Occurrence and importance

Hydatidosis is rare in much of Africa but a major problem in some areas, e.g., in Turkana, Kenya.

3. Epidemiology

Dogs, and other carnivores such as lions and jackals, are the final host of the dog tapeworm. Eggs are passed in the dog's faeces and ingested by sheep, goats, cattle, camels, donkeys and wild antelopes.

The eggs hatch in the animal's intestine and the larvae penetrate the intestinal wall and pass into the portal circulation to be carried to the liver and lungs where they form cysts with many daughter cysts. When a dog eats the diseased animal, it is infected by these cysts, which then proceed to develop into mature worms.

Man becomes infected when he accidentally swallows the eggs from a dog's faeces.

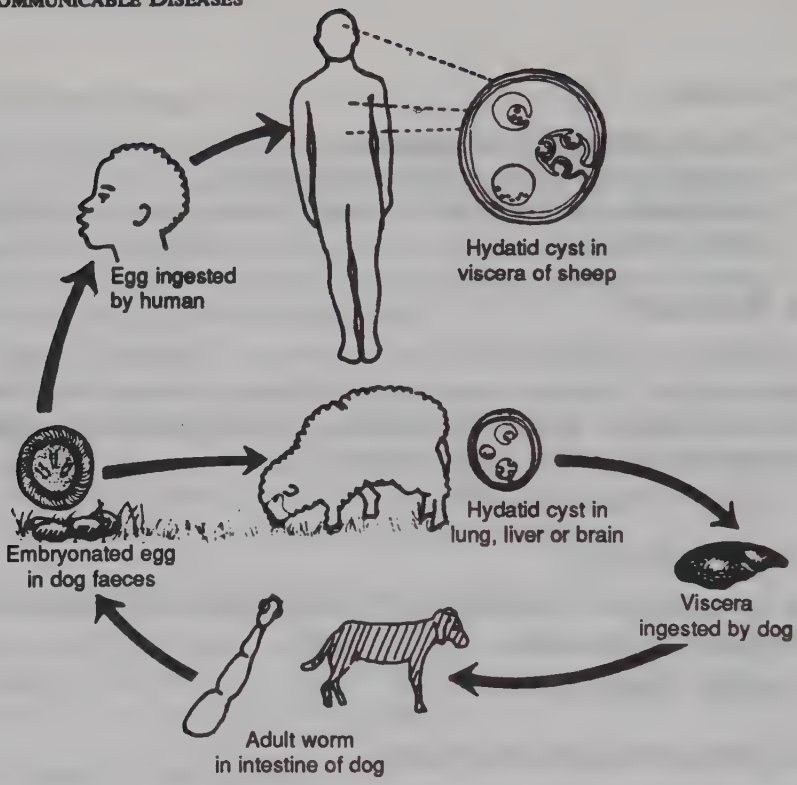


Figure 6.11 **Life cycle of *Echinococcus granulosus***

4. Clinical picture

A slow-growing cyst develops, usually in the liver, but almost any tissue in the body can be affected, the lung being the next commonest site. The liver becomes enlarged — this could be confused with amoebic liver abscess but the general condition of the patient remains good and there is a high eosinophilia.

A chest X-ray or abdominal ultrasound investigation may be done in hospital, and serological tests can be done to assist in making the diagnosis. Refer all suspected cases to hospital.

5. Management of the individual patient

The cyst should not be punctured. This may provoke a severe anaphylactic reaction. Treatment is with albendazole 20 mg/kg for 30 days. This gives a 70% cure rate in single cysts. Multiple cysts may require surgery as well but there may be problems of spillage and failure to enucleate the cysts.

6. Prevention and control

- Meat inspection; condemn infected meat. Do not feed it to dogs.
- De-worm dogs regularly using praziquantel, repeated after 6 weeks. This is quite expensive, however.
- Health education, especially of children, in endemic areas on the dangers of close contact with dogs (licking).
- Dog control — eradication of stray dogs.

7. Action

Health education

- On the dangers of close contact with dogs.
- On the necessity of de-worming dogs regularly.
- On the dangers of feeding dogs with uncooked viscera of sheep, especially if condemned.

8. Summary

Hydatidosis in humans is caused by the development of cysts — generally in liver — of the dog tapeworm. Treatment is with albendazole or surgery. Control is by avoiding intimate contact with dogs, by eradication of stray dogs and by de-worming of dogs.

Chapter seven

Diseases Through Contact with Animals or their Products

INTRODUCTION

Infectious diseases transmitted under natural conditions between vertebrate animals and man are called zoonoses. Diseases which occur all the time in the animals are called enzootic (like endemic in human populations), and when they occur in greater number than expected this is called an epizootic (like epidemic).

In some of these diseases man is usually a dead end for transmission. An example is hydatidosis, where man is an exceptional intermediate host and a dead end to transmission unless his body is eaten by a dog or predator which is the final host. In the case of rabies, the infection usually spreads from animal to animal and man only gets infected accidentally when he is bitten by a rabid animal. The transmission should usually end here, but it is possible for a patient with rabies to bite another person and transmit the disease at the same time. With brucellosis and anthrax, man is also usually a dead end to transmission, but as these are very infectious agents (and one forms spores), it is again possible that, without good nursing and laboratory precautions, the agent could be transmitted accidentally from one person to another.

The organism which causes tetanus (*Clostridium tetani*) is a commensal of cattle and other animals. Tetanus is caused only when there is accidental contamination of a deep lacerated wound or of the umbilical stump with the *Clostridium* spores from faeces of these animals. The spores of the *Clostridium* can survive in the dust where these animals have defecated, and this dust can be carried to distant places by the wind and contaminate an operating theatre or other surfaces, resulting in accidental infection. In this case the animal connection is remote and not so obvious.

Yellow fever is always transmitted from animal to man and from man to man by mosquitoes of the genus *Aedes*. The virus can also be transmitted from monkey to mosquito, from mosquito to monkey and occasionally from mosquito to man (see page 125).

mosquito ——> monkey ——> mosquito ——> monkey

mosquito ——> man ——> mosquito ——> man

In trypanosomiasis, the tsetse fly is the vector transmitting the infection between herbivores, from herbivore to man, and from man to man.

Plague, which is an enzootic in wild rodents in some areas but may become an epizootic, is transmitted by fleas between rodents and man, and then from man to man. However, if man develops the pneumonic form of the disease, the cycle can then go directly from man to man by droplet infection.

Salmonella and *Campylobacter* infections are caused by organisms which are usually present in the systems of various animals. The infections occur in man when he consumes or works with the carcasses of such animals (e.g., a hen), or when he consumes foods contaminated by the faeces of these animals (e.g., rats). Transmission can then occur from man to man via the faeco-oral route.

Man acts as the final host in taeniasis, and the animals (cattle and pigs) are intermediate hosts. If man had better ways of disposing of his faeces the disease would cease to exist in the animals.

Table 7.1 Transmission of the zoonoses

	Disease	Reservoir animals involved	Transmission to man	See Chapter
ZOONOSIS	Plague	Rodents	Vector (flea)	4
	Sleeping sickness	Cattle and game animals	Vector (tsetse fly)	4
	Yellow fever	Monkeys	Vector (*Aedes* mosquito)	4
	Anthrax	Cattle (and game)	Ingestion Inhalation Contact	7
	Salmonellosis	Rodents, poultry, cattle	Ingestion	5
	Campylobacter	Poultry	Ingestion	5
	Brucellosis	Cattle	Ingestion Contact	7
	Rabies	Wild animals Dogs	Bite wound	7
Final host	Taeniasis	Cattle, pigs	Ingestion	6
Intermediate host	Hydatidosis	Dogs Carnivores	Close contact with dogs Ingestion	6
Commensals	Tetanus	Cattle Horses	Contaminated wounds	7

RABIES

1. Rabies is a disease of animals (a zoonosis) incidentally transmitted to human beings by the bite of a rabid animal.

2. Occurrence and importance

Rabies is spreading in many parts of Africa. For example, before 1971 the disease was confined to Iringa, Kigoma, Dodoma and West Lake regions of Tanzania. By 1977, however, the disease had spread to the coast, central plateau, northern and southern highlands and the lake regions.

Rabies is an important public health problem because:

- When no immediate action is taken after the bite by an infectious animal, the disease is invariably fatal once the clinical signs have appeared (case fatality rate is 100%);
- Africa has a large reservoir of diseased wild animals;
- The transmission between wild animals and domestic animals is difficult to control under present conditions.

3. Epidemiology

Rabies is caused by a virus. The virus has preference for the salivary glands and nervous tissue, finally causing an encephalitis, and resulting in death.

The main animal reservoir of the disease is wild animals such as jackals, mongooses, and hyaenas. These animals usually do not live in close contact with man, but they may infect domestic animals, such as dogs and cats. Cattle, horses and donkeys may also become infected (in many parts of the world bats can be infected).

Man becomes infected when he is bitten by a rabid animal, usually a dog. The saliva left behind in the wound contains the virus which finds its way to the brain via the nerves.

4. Clinical picture

In man

The incubation period ranges from 2 weeks to 1 year, with an average of 2–3 months, depending on:

- The site of the bite — whether it is far or near the brain;

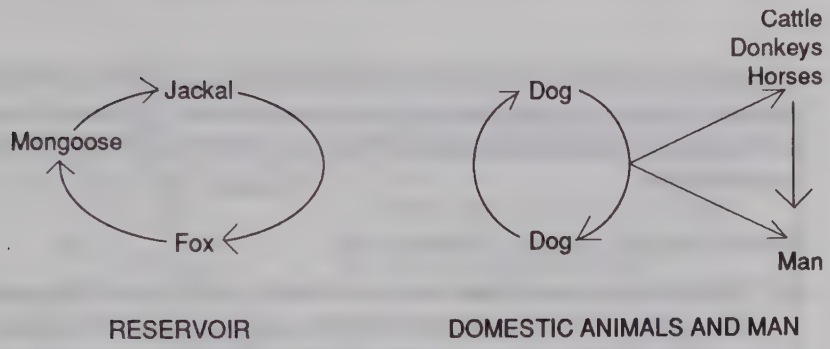


Figure 7.1 **Transmission cycle of rabies**

- The type of wound (abrasion, small wound, wound with extensive tissue damage);
- The dose of virus deposited in the wound.

The earliest symptoms usually consist of increasingly severe pain in the wound. Later, two different clinical pictures emerge:

Furious rabies which is associated with convulsions, spasms, and intense anxiety. This may then lead to:

Paralytic rabies which is associated with depression, paralysis of the limbs, and spasm of the pharyngeal muscles which results in intense hydrophobia (fear of water) because of the severe pain experienced when swallowing water.

Survival for more than a week is unlikely and death follows.

In animals

Abnormal behaviour is one of the earliest signs. The animal may bite at the slightest provocation or without any provocation at all. Animals may also be depressed and curl up in a corner.

Death usually ensues within 10 days of the onset of the disease.

Any wild animal that wanders into a home should be suspected of rabies and should not be picked up

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5. Management of the individual patient

No cure is available once the disease has actually developed. Prevention

is, however, possible before it has reached that stage. Once a person has been bitten by a rabid animal, the disease can be prevented from developing by first-aid treatment of the bite, followed by anti-rabies immunization.

How to handle a case of animal bite

(a) If the animal is available and looks healthy, write down the name and address of the patient and ask him to come back for a check-up after 10 days. Note the date of the appointment in your diary.

The animal must be observed for 10 days. If it remains healthy during this period, there is no danger of rabies. If, on the other hand, the animal shows signs of rabies, trace the patient immediately and refer him with a proper referral letter to the district hospital for immunization.

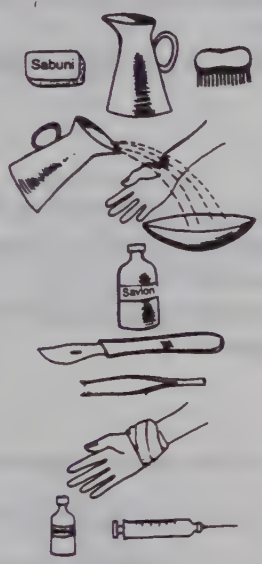
(b) If the animal has disappeared: refer the person for immunization.

(c) If the animal looks rabid and has been caught: lock it up carefully; inform the DMO of your suspicion of rabies, and refer the patient for immunization.

(d) If the dead animal is brought to your dispensary: bury it carefully (unless you make arrangements for investigation of the dog with the local Veterinary Department and they give you detailed instructions on how and where to send the infectious material). Do not send heads of rabid dogs in plastic bags or wrapped in papers by bus or post to a Veterinary Department which does not have the equipment for laboratory confirmation of the diagnosis. Refer the patient to hospital for immunization.

6. Prevention and control

Rabies is a notifiable disease. Its control is the full responsibility of the Veterinary Department. You must, however, play your part by giving health education about preventive measures, such as regular immunization of dogs and cats (at least every 3 years), killing of stray dogs, etc. Routine pre-exposure immunization of people with the rabies vaccine presently available is expensive. This is, for the moment, reserved only for those at high risk of catching the disease, e.g., veterinary officers.



1. Wash out the wound with PLENTY of water, soap and a brush
2. Rinse again with plenty of water
3. Then apply disinfectant
4. Do surgical toilet
5. Do NOT stitch the wound, but apply a swab and a slight compression bandage to stop the bleeding
6. Give tetanus toxoid vaccination

Note
Take a very good history. Refer to hospital for rabies immunization if:

- known animal becomes ill and dies;
- animal unknown and disappears;
- animal looked rabid;
- dead animal brought.

Figure 7.2 Rabies prevention: proper first aid for all animal bites, and immunization

7. Action

In all cases of animal bites:

- Give first aid (see Figure 7.2, above);
- Give tetanus prophylaxis;
- Decide on rabies immunization;
- Observe biting animal for 10 days if available;
- Refer all patients for whom rabies immunization is indicated with a proper referral letter indicating why you refer the patient.
- If you are working in an area where rabies is a problem make arrangements with the Veterinary Department for investigation of suspected dogs and specimens (NB: staff working with such material should of course be immunized);

- If there are increasing numbers of people coming to your clinic with bites from suspected rabid animals, inform your DMO and discuss ways of controlling the situation;
- Encourage people to have their dogs and cats immunized regularly where possible.

8. Summary

Rabies, once manifested in human beings, is a 100% fatal disease. Prevention is possible, but only when immediate action is taken for those who have been bitten by a suspect animal.

The prevention of rabies in domestic animals by immunization is the responsibility of the Veterinary Department.

Notes

Types of anti-rabies vaccine

(a) Human diploid cell substrate vaccine (HDCV). This is the safest and most effective. It is given as follows:

For prevention of cases, following bites or other exposure, it is given 1ml intramuscularly on days 0, 3, 7, 14, 30 and 90;

For pre-exposure immunization of high risk workers (e.g., veterinary officers) two intradermal injections of 0.1 ml 1 month apart with booster intradermally each year. This vaccine is, however, expensive and in some parts of Africa may not be readily available.

(b) There are several other types of vaccine available. Most of these are made from brains of infected animals and contain traces of neural tissue. Because of this, these vaccines often cause side effects and sometimes serious nervous system reactions. Immunization courses involve multiple injections, e.g., daily for 21 days and boosters, or for 7 days with boosters on days 11, 15, 30 and 90. Always read carefully the instructions included with the type of vaccine you have to use.

Indications for vaccine and serum

Immunization against rabies can be done with anti-rabies serum and anti-rabies vaccine.

The anti-rabies serum gives passive immunity. It has an immediate effect but the immunity is short-lived. Anti-rabies serum is expensive and not available at the moment in many parts of Africa.

Anti-rabies vaccine gives active immunity. It takes about 2 weeks before antibodies appear. After active immunization the level of antibodies is high and immunity is long-lasting.

Indications for use of anti-rabies serum and anti-rabies vaccine are given in Table 7.2.

There are two types of anti-rabies serum: anti-rabies of animal origin, and human rabies immune globulin. Both are injected around the bite wound and then given intramuscularly, after a test dose. Anaphylaxis is a real danger with the animal anti-rabies serum, but the human anti-serum is relatively safe if free of the AIDS virus.

Table 7.2 **Anti-rabies immunization**

	Type of exposure	Circumstances	Recommended treatment
1.	Indirect contact; no bite, no licks	Rabid	None
2.	Licks on the skin; scratches or abrasions minor bites (covered areas of arms, trunk and legs (Minor exposure)	(a) Suspected as rabid, unprovoked attack by cat or dog	Start vaccine, stop treatment if animal remains healthy for more than 10 days (see a, c below)
		(b) Rabid: wild animal or animal unavailable for observation	Serum + vaccine
3.	Licks of mucosa: major bites (multiple or on face, head, finger, or neck) (Major exposure)	Suspect or rabid, domestic or wild animal or animal unavailable for observation	Serum + vaccine. Stop treatment if animal remains healthy for 10 days (a, c)

a. Observation period in this chart applies only to dogs and cats.
b. All unprovoked bites in endemic areas should be considered suspect unless proved negative by laboratory examination.
c. Or if brain is found negative by laboratory examination.
d. In general, exposure to rodents and rabbits seldom, if ever, requires specific anti-rabies treatment.

TETANUS

1. Tetanus is an acute disease caused by a toxin produced by tetanus bacilli and is characterized by painful contractions of voluntary muscles.

Synonym: Lockjaw.

2. Occurrence and importance

Tetanus occurs sporadically all over Africa. It is more common in rural areas than in towns. Tetanus is an important disease because of its high case fatality rate of 20–40% in adults. Groups especially at risk are newborns, farmers, soldiers, children and, in general, anyone with a dirty wound. Many of the newborns with clinical tetanus will die (60–89%) even when the nursing care is excellent.

Surveys for neonatal tetanus deaths have been done in several countries. Reported rates per 1,000 live births range from 18 in Côte d'Ivoire in 1982, 15 in Uganda in 1984, to 4 in Zambia in 1986 and 4 in Kenya in 1986.

3. Epidemiology

Tetanus is caused by a toxin produced by *Clostridium tetani*, a Gram-positive rod which forms terminal spores, giving it a characteristic drumstick appearance under the microscope (Figure 7.3).

The organism is an obligate anaerobe, that is, the tetanus bacillus can only live and multiply in the absence of oxygen.

Normally, tetanus bacilli live in the bowels of animals (and man). When the bacilli are passed out in faeces, they form spores in order to survive the harsh conditions outside. Therefore tetanus spores may be present in any soil contaminated by faeces, particularly faeces of cows. Tetanus bacilli continue their life cycle when the spores are swallowed by animals (and man).

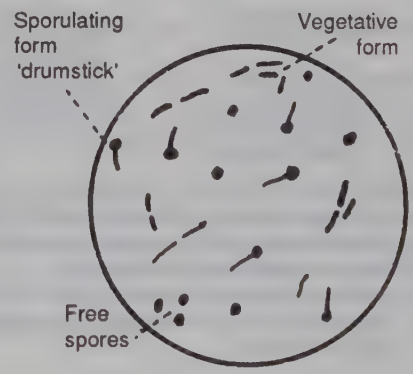


Figure 7.3 **Microscopic appearance of *Clostridium tetani***

The spores are highly resistant to drying and high temperatures; they even withstand boiling for short periods but are destroyed by autoclaving at 115°C for 15 minutes. Tetanus, as a disease in man,

occurs only when the spores enter the body through a contaminated wound. The source of the infection is soil, street dust, or faeces containing spores of tetanus bacilli. Tetanus is more likely to develop therefore in patients with deep penetrating necrotic wounds.

Tissue necrosis is the result of impaired blood supply, as it is the blood that brings nutrients and oxygen to the tissues. In a mixed infection the aerobic bacteria can consume so much oxygen that conditions may become favourable for *Clostridium tetani* to grow.

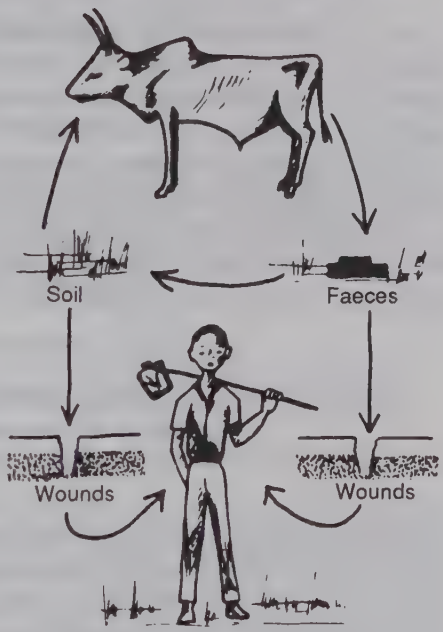


Figure 7.4 **Transmission of *Clostridium tetani***

Wounds which favour tetanus are:

- Umbilical stump in newborn (necrosis);
- Crush wounds (necrosis, poor blood supply);
- Stab wounds (deep);
- Wound with foreign bodies (always infected), human (!) and animal bites;
- Burns (necrosis, blood supply);
- Endometritis (after abortion or childbirth, from the use of poorly sterilized instruments);
- Surgical wounds (from dressings or instruments);
- Chronic ulcers (like jiggers or in leprosy or Guinea worm) and chronic discharging ears;

- Endogenous infection may occur when intestinal tetanus bacilli are introduced into the tissues (during bowel surgery).

In 10–15% of cases of tetanus the site of entry remains undetected. Tetanus bacilli are harmful only when they are lodged in the tissue because from there the toxin can be transported to the central nervous system. When tetanus spores or bacilli are swallowed, the bacilli can live and multiply in the bowel but no harm will be done as the toxin is not readily absorbed from intact bowel.

A special and very important form of tetanus is seen in the newborn. It is called tetanus neonatorum, and it is nearly always fatal, even with the best kind of treatment.

Tetanus neonatorum is preventable by immunization of mothers during pregnancy

After birth, blood stops flowing through the umbilical cord. The cord becomes necrotic, dries up and falls off in a few days. The necrotic tissue in the umbilical stump is an ideal place for tetanus bacilli to enter the body, especially if cow-dung containing tetanus bacilli is used for dressing the cord, as has been the custom in some communities in Africa. The infection can also be introduced if unsterile instruments are used for cutting the umbilical cord.

4. Clinical picture

The usual incubation period is 5–21 days (but it can range from 3 days to 3 months). The time between the first signs and the first generalized spasms is called the onset time. The shorter the incubation period and the onset time the more fulminating is the disease.

The disease starts with increased tone in the jaw muscles, causing trismus and "risus sardonicus" (devil's grin). Later the spasms become more generalized, involving all muscles. Severe spasms are associated with pain and disturbances of swallowing and respiration. The spasms of the neck muscles may resemble the neck stiffness of meningitis, but other neurological signs are negative. There is no drowsiness or change in consciousness, as in meningitis. There may be a high fever and hypertension. Death usually occurs due to asphyxia and exhaustion.

Asphyxia may be due to:

- Spasm of glottis, thoracic muscles, diaphragm;
- Chronic hypoventilation because of muscle stiffness;
- Periods of apnoea;
- Aspiration of feeds and respiratory secretions and subsequent suffocation.

In the newborn the first sign of tetanus is inability to suck in a baby who was first doing well; later spasms appear accompanied by severe apnoea and cyanosis. A typical feature is the extended spine with clenched fists and mouth and flexed toes.

5. Management of the individual patient

Patients must be referred to hospital as quickly as possible.

Spasms

Even at a dispensary or health centre an attempt must be made to control the spasms before referring the patient. The ideal drug to use is diazepam (Valium) in high doses. Start with 10–40 mg intravenously and give the same dose at the same time in the form of crushed tablets through a gastric tube. Maintain the sedation by giving the drug every 3 hours through the tube.

Further doses of diazepam should be given according to the condition of the patient. When spasms continue, more diazepam should be given. Diazepam is usually very well tolerated and the maximum dose is very high (500 mg daily). As the success of tetanus treatment depends entirely on the control of spasms, do not hesitate to increase the dose of this drug until the patient is very well sedated.

However watch for signs of over-sedation. Reduce the dose when patients do not react at all when touched. A properly sedated patient gets spasms lasting only 1 second when awakened by touching or shaking.

Chloral hydrate, phenobarbitone, or chlorpromazine will sustain the effect of diazepam.

Children: Chloral hydrate.

Adults: phenobarbitone 100 mg 4-hourly.

Secondary infection

To combat secondary infection and the tetanus bacilli themselves, crystalline penicillin 1 mU and PPF 1.2 mU are given on admission. PPF 1.2 mU is continued daily for 5 days.

Antibiotics do not alter the severity or the duration of the spasms. Spasms are not caused by bacilli but by the toxin already fixed to the neurones.

ATS (anti-tetanus serum)

The effect of anti-serum in the treatment of tetanus is still doubtful. Dose: adults and children 10,000 units intramuscularly or intravenously once; give test dose first, keeping adrenaline at hand because allergic reactions are common and dangerous.

Surgical treatment

Look for any wound, and clean with spirit or Savlon. Operative procedures in a patient with established tetanus are not recommended because severe, uncontrolled spasms commonly accompany surgery performed in the early stages of the illness. Tracheostomy with artificial respiration can only be performed in specialized centres.

Intensive care

Careful nursing is the most important part of treatment. Formerly tetanus patients were frequently nursed in dark and isolated rooms. This resulted in the nurses forgetting to observe the patients. The patient should be nursed in a well-lighted room where proper observations can be carried out by the nursing staff. If he develops breathing arrest due to spasms, immediate mouth-to-mouth respiration should be performed (tetanus is not infectious). When he is spastic, immediate sedation should be given. Observation from minute to minute will save the life of a tetanus patient.

Do not isolate tetanus patients: they may be forgotten and die

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The patient should be nursed in a semiprone position (never on his back). His position must be changed every 2 hours. Raise the foot of the bed to stimulate lung drainage. This will help to prevent pneumonia.

Every patient must have an observation chart on which medication, fluid input, spasms, and position are recorded.

Toxoid (TT)

Patients with tetanus do not develop immunity against tetanus. When they have recovered, they still have to be immunized with tetanus toxoid to avoid recurrence.

6. Prevention and control

Proper surgical treatment of wounds, such as removal of foreign bodies and excision of necrotic tissue, will diminish the risk of tetanus. Active immunization with tetanus toxoid gives adequate protection. The first three immunizations should be given in childhood, together with diphtheria toxoid and pertussis vaccine (DPT).

In case of injury later in life, if primary immunization was complete, a single booster injection will result in a rapid and high rise in antibody levels which are protective for 7–10 years.

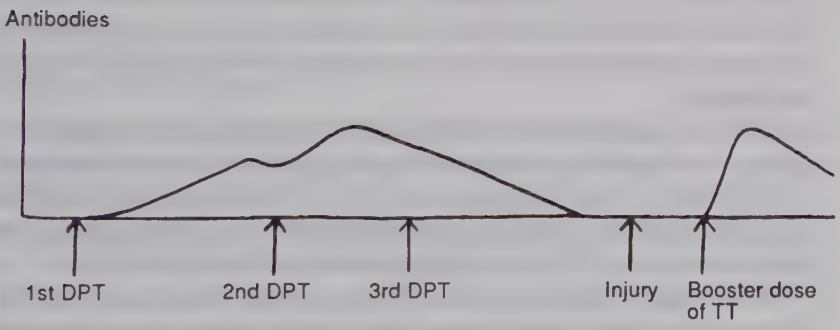

Figure 7.5 **Antibody response to TT immunization**

When no previous active immunization has been performed, the patient presenting with an injury needs prompt protection as the incubation period of tetanus is shorter than the time the body needs to produce active antibodies. This protection can be given in the form of anti-tetanus serum (ATS).

Passive protection lasts only for a very short time (10 days) and therefore ATS should never be given without active immunization

with tetanus toxoid. The routine of prescribing ATS and PPF to all injured patients is an example of bad practice, and should be stopped. ATS is horse serum and therefore a potentially dangerous drug, especially on second administration because of the possibility of anaphylactic reactions. This is expressed diagrammatically in Figure 7.6.

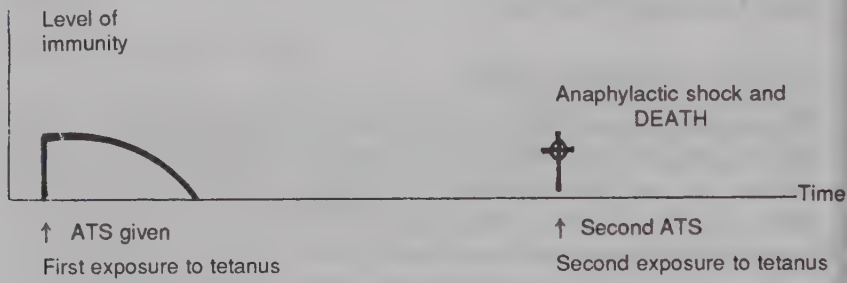

Figure 7.6 **The effect of injudicious use of ATS**

Tetanus of the newborn can be prevented by active immunization of the mother during pregnancy. Usually two injections of tetanus toxoid are given 4 weeks apart. The first one must be given at the first visit to the antenatal clinic. There is no need to wait until 28 weeks of pregnancy.

After the administration of tetanus toxoid, the mother generates antibodies which circulate in her blood. From here they are transferred to the foetus across the placenta. The newborn therefore obtains antibodies passively from the mother and these will protect him from tetanus when the umbilical stump is healing after delivery.

DPT should never be given to adults as the pertussis part of the vaccine may cause severe reactions. DPT cannot replace tetanus toxoid, and so do not give DPT to pregnant mothers when tetanus toxoid is out of stock.

There are no contra-indications for tetanus toxoid, and its side effects are minimal (pain at the injection site for 12–24 hours).

The EPI programme schedule for tetanus immunization for infants in most countries is as follows:

DPT I at 6 weeks;
DPT II at 10 weeks;
DPT III at 14 weeks.

Table 7.3 **The immunization schedule for women**

Dose	When to give	% protection	Duration of protection
TT1	At first contact or as early as possible in pregnancy	Nil	None
TT2	At least 4 weeks after TT1	80	3 years
TT3	At least 6 months after TT2 or in next pregnancy	95	5 years
TT4	At least 1 year after TT3 or in subsequent pregnancy	99	10 years
TT5	At least 1 year after TT4 or in subsequent pregnancy	99	For life

7. Action

Patients with tetanus

- Control spasms with either diazepam intramuscularly 10–40 mg or phenobarbitone 100 mg plus chlorpromazine 50 mg.
- Pass gastric tube.
- Repeat same dose of diazepam immediately by mouth.
- Give PPF 1.2 mU daily for 5 days.
- Write letter of referral and put patient on transport.
- Give tetanus toxoid to your patient after recovery.

Community

- Give TT to all pregnant mothers and women in reproductive age groups.
- Give DPT three times to all under-fives.
- Train and equip village midwives and MCH workers, and teach traditional midwives to refer antenatal cases for immunization, to cut the umbilicus with a sterile instrument and to use a clean sterile dressing (this will also prevent other infections of the umbilicus).

- Give health education on proper clean treatment of wounds and the umbilical stump.
- Immunize schoolchildren with TT.

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3 DPT for all under-fives
TT for all pregnant mothers
TT for all contaminated wounds
TT for patients recovering from tetanus
ATS only when indicated, and always with TT

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8. Summary

Tetanus is a severe complication of wound infections. It can be prevented by immunization.

If routine DPT immunizations were given during childhood only a booster with TT is required. This will result in a rapid and high rise of immunity.

Tetanus can be prevented by immunization

ANTHRAX

1. Anthrax is an acute bacterial disease, primarily of grass-eating animals. Occasionally it infects man.

Synonyms: Hide-porter's disease, wool-sorter's disease, malignant pustule.

2. Occurrence and importance

Anthrax is a disease of cattle, goats and sheep. It can occur suddenly in large numbers (epizootic), especially in times when cattle are moved from one place to another, e.g., after drought or flooding.

Several types of infection occur in man, depending on the portal of entry:

- Anthrax of the skin occurs mostly in persons handling cattle: farmers, butchers, workers in the hide-processing industry;
- Anthrax of the lungs occurs in persons working with infected wool;
- Anthrax of the bowel occurs in families who eat the meat of sick or dead animals.

3. Epidemiology

Anthrax is caused by a spore-forming bacillus (*Bacillus anthracis*). The spores are formed when the bacillus is exposed to air. Spores are extremely resistant to climatic conditions and can survive for years in the soil. Vultures may spread the spores. The spores enter the animals orally.

Anthrax in animals is characterized by a short fever followed by septicaemia and death. Animals which have died of anthrax show:

- Jelly-like oedema of the spleen;
- Dark unclotting blood;
- Enlarged lymph glands;
- Bloody oedema of subcutaneous tissue;
- Haemorrhagic enteritis.

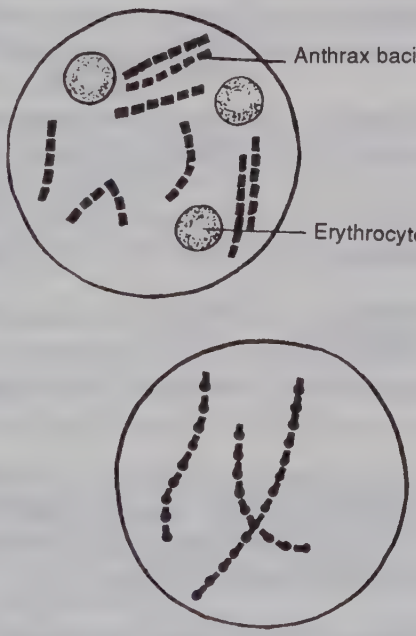


Figure 7.7 **Microscopic appearance of *Bacillus anthracis* and spores**

The body of the sick or dead animal is teeming with bacilli which are easily spread via faeces, urine, saliva, and blood. Animals with anthrax should, of course, not be cut open or have a post-mortem as this spreads the spores. The Veterinary Officer usually just takes blood from an ear for laboratory examination.

4. Clinical picture

Clinical picuture in man depends on portal of entry, as shown in Table 7.4.

Table 7.4 **Clinical picture in man**

Portal of entry	Scratches on skin	Respiratory tract	Digestive tract
Early signs	Papule Blister Malignant pustule	Mild symptoms of URTI	Vomiting Watery diarrhoea
Full picture	Black central necrosis Extensive oedema Painless swelling	Serious respiratory distress (pneumonia) Shock	Fever Sepsis
Case fatality	Low	High	Moderate
Laboratory diagnosis from	Fluid of vesicles	Sputum	Faeces
Treatment	Penicillin	Oxygen/penicillin	Rehydration

The diagnosis is confirmed by finding Gram-positive rods in a specimen (fluid from vesicle, sputum, or faeces).

Anthrax may be complicated by anthrax haemorrhagic meningitis in a small proportion of cases.

5. Management of the individual patient

The drug of choice is penicillin, although any antibiotic can be used. Isolation of the patient is not necessary since he is unlikely to infect others.

6. Prevention and control

The institution of control measures is the responsibiity of the Veterinary Department. These measures include:

- Proper disposal of infected animals. The carcasses must be burnt or buried at least 2 m deep in the ground in quicklime;

- Inspection of all meat offered for sale;
- Immunization of all susceptible animals every year;
- Strict rules for disinfecting skins and hides in the leather-processing industry;
- People at risk may be protected with a vaccine for humans.

7. Action

- In time of famine people will eat diseased or dead animals. Whole families may come to the dispensary, all with gastro-intestinal complaints. In this case rule out anthrax.
- During your regular health talks about nutrition, explain the dangers of eating sick or dead animals, unless the Meat Inspector has approved certain parts of the meat.
- Discuss with the Veterinary Officer the incidence of animal diseases which affect man in your area. You will then know which diseases to expect.
- In case of epidemics among animals, use your influence with the farmers to convince them to have their cattle immunized. Explain why skins of diseased animals can be dangerous if untreated.

8. Summary

Anthrax is a disease of grass-eating animals, occasionally transmitted to man. Infections in man may involve the skin, respiratory tract or digestive tract, depending on the portal of entry. All antibiotics are effective, but penicillin is the drug of choice.

Control of the disease in animals is possible by immunization and proper disposal of infected animals.

Control in man involves proper meat inspection, disinfection procedures in handling skins, and health education.

BRUCELLOSIS

1. Brucellosis is a disease of animals. It may be transmitted to human beings and is characterized by fever, headache, weakness, sweating, joint pains, and generalized aching.

Synonyms: Undulant fever, Malta fever.

2. Occurrence and importance

Brucellosis is an occupational health hazard of farmers, veterinarians, abattoir workers, and butchers. It is more common in males than females. The incidence in Africa is unknown because many cases may be missed due to lack of laboratory facilities. It has been reported mostly from the savannah areas where there are large herds of cattle and goats. The infection in animals causes repeated abortion, presenting an economic problem, particularly for cattle owners. Case fatality rate is less than 2%. Death may be due to complications of the disease.

3. Epidemiology

Close contact with infected cattle provides opportunity for human infection. The disease is transmitted by contact with tissues, blood, urine, vaginal discharges, aborted foetuses or, especially, placentas of infected animals. The disease can also be transmitted by ingestion of milk or milk products from infected animals, but this seems to be uncommon.

Causative organism: *Brucella abortus* (cattle)
Brucella melitensis (goats, sheep)
Brucella suis (pigs).

4. Clinical picture

The patient usually presents with fever of unknown origin (PUO). The onset is usually insidious. A common complaint is pain in the lumbosacral region or hip joint. The pain may be severe.

Diagnosis on clinical grounds is often difficult. The occupation of the patient may raise suspicion. Splenomegaly is common but of little help in a malarious area. Serological diagnosis may be possible in hospital.

Intradermal tests are available but unreliable and therefore not recommended.

5. Management of the individual patient

Tetracycline for 2–6 weeks is the drug of choice. Addition of streptomycin will reduce the relapse rate, but watch out for side-

effects (ototoxicity) in long-term use. Cotrimoxazole has also been used.

6. Prevention and control

Boil milk before use, and use only boiled milk for preparing cheese and butter. Slaughter infected animals and immunize calves in endemic areas (this may be possible only with dairy herds but not in nomadic pastoral areas).

7. Action

If brucellosis is a problem among farmers in your area, contact the Veterinary Officer and discuss the problem. If he confirms a high prevalence of the disease in animals, arrange with your DMO to provide facilities for serological testing of patients with fever who do not respond to antimalarials. Give health education to the farmers.

8. Summary

Brucellosis is an occupational health problem of cattle and goat farmers. Patients present with fever. Diagnosis is difficult. Treat with tetracycline.

Chapter eight

Airborne Diseases

INTRODUCTION

Airborne diseases are of great public health significance in eastern Africa. They include pneumonia and measles, which are the two most common causes of death, especially in children. These two diseases may account for about a quarter of all deaths occurring in hospitals in eastern Africa. Pneumonia deaths account for about one-third of all deaths in children under 5 years in developing countries.

Respiratory infections in Africa are a very important cause of illness in children. For example, in the Gambia in the first two years of life a child may suffer from upper respiratory tract infection for 24% of the time and from lower respiratory tract infection 3% of the time. With each illness there is loss of weight. This may explain the failure to thrive observed in many children in the second year of life. Pneumonia is also worse in a malnourished child.

The organisms causing the diseases in the airborne group enter the body via the respiratory tract. When a patient or carrier of pathogens talks, coughs, laughs, or sneezes, he discharges droplets of fluid into the air. The smallest of these droplets remain in the air for some time and may be inhaled by a new host. The bigger droplets fall to the ground and mix with the dust. Some organisms survive the drying conditions and will cause an infection when inhaled with the dust.

Obviously, airborne disease will spread more easily when there is overcrowding, as in overcrowded houses, classrooms, public transport, canteens, dance halls, and cinemas.

Good ventilation can do much to counteract the effects of overcrowding. Airborne diseases are mostly acquired through the respiratory tract, but not all of them are primary respiratory tract infections (Table 8.1).

Table 8.1 **Acute respiratory tract infections**

Acute respiratory tract infections	Common name	Main sign or symptom	Causative organisms and incubation time	Remarks
Coryza	Common cold	Running nose	Virus 3 days	Self-limiting
Pharyngeal syndrome	Sore throat	Sore throat	Virus Streptococci 2–5 days	Differentiate and treat accordingly
Influenza	Flu	Systemic disturbances	Virus 1–3 days	Often epidemic
Herpangina		Vesicles in mouth	Virus 3–5 days	Self-limiting
Acute laryngo-tracheo-bronchitis	Croup	Inspiratory stridor	Virus Secondary infection 3–5 days	Refer
Bronchitis	Bronchitis	Cough No dyspnoea	Virus Bacteria	Aggravated by dust and smoke
Acute bronchiolitis of infancy		Expiratory wheezing Dyspnoea	Virus 2–4 days	Refer
Pneumonia		Cough Rapid breathing Dyspnoea Fever Consolidation	Pneumococci Haemophilus Virus 1–3 days	Admit

The respiratory infections are caused by a wide variety of organisms and are classified according to the clinical picture as follows:

RESPIRATORY TRACT INFECTION

1. Acute respiratory infections (ARI)

Upper respiratory tract infections (URTI) such as coryza, pharyngitis, tonsillitis, or laryngo-tracheitis.

Lower respiratory tract infections (LRTI) such as bronchitis, bronchiolitis, lobar and/or broncho-pneumonia.

2. Chronic respiratory infections

Whooping cough. Tuberculosis (see Chapter 9).

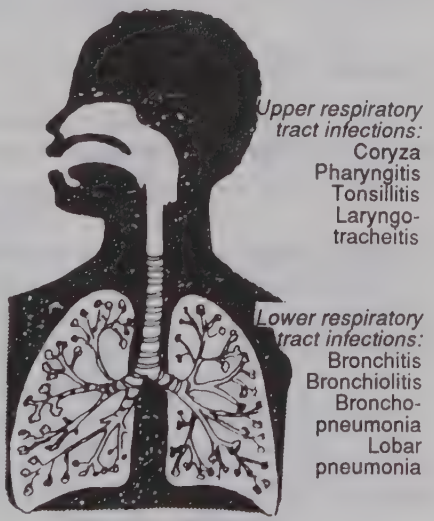


Figure 8.1 **Respiratory tract infections**

The danger of respiratory tract infections is their effect on the gaseous exchange in the lungs.

The main sign of disturbed gas exchange is dyspnoea. Dyspnoea is always a sign of severe disease. All patients with dyspnoea, whatever the cause, need admission and intensive treatment because life is at stake.

From Table 8.1 it can be seen that the diseases above the line do not have dyspnoea as their main symptom. But they can all be complicated by one of the diseases below the line. Note that the last two diseases disrupt the gaseous exchange because of damage lower down in the respiratory tract.

Respiratory tract infections of viral origin are important because of their frequent occurrence and their tendency to be complicated by

secondary bacterial infections.

Other diseases which enter through the respiratory tract are shown in Table 8.2.

Table 8.2 **Other airborne diseases**

Disease	Causative organism	Main way of transmission	Complicated by LRTI	Occurrence in Africa	Approximate fatality rate
Measles	Virus	Airborne droplet	++	++++	± 5 %
Chickenpox	Virus	Airborne droplet	+	++++	v. low
Mumps	Virus	Contaminated articles (eating utensils)	-	++++	v. low
Rubella	Virus	Airborne droplet	-	+++	0
Meningitis	Viruses and bacteria	Airborne droplet or endogenous (depending on organism)	-	+++	± 20%
Leprosy	Myco-bacterium	Droplets and nasal discharge	-	++	0

CONTROL OF AIRBORNE DISEASES

Since the infective particles are spread by droplets from the respiratory tract of patients or carriers, an important part of the control of these diseases is based on preventing droplets from being inhaled by others; in other words, people must not inhale "second-hand" air.

Ventilation

Ventilation removes used air and replaces it with clean air. As soon as droplets are taken outside, the sun sterilizes them. Increased ventilation can be achieved by building houses with windows, thus allowing

natural ventilation. Cinemas and meeting halls should make use of artificial ventilation by means of fans.

Overcrowding

Having too many people in the same room should be avoided. This is especially important in prisons, garrison barracks, dormitories in boarding schools, on ships, and in urban housing where many people may be forced to live in a single room. Building houses with adequate space (even using local materials) is of value.

Isolation

This was a method formerly used to prevent the spread of disease. All people known to be suffering from airborne diseases were isolated.

Examples: isolating TB patients in sanatoria; isolating measles children in infectious disease wards.

It is now known that most diseases are infective before they are recognized. Children with measles are most infective before the measles rash is apparent. TB patients present themselves for treatment only when serious complications like haemoptysis occur. In the meantime, they risk infecting all the people they live with.

Nobody likes to be isolated. Isolation can be frightening. Because of this people who may be suffering from "isolatable" diseases tend to delay reporting to medical facilities. At present isolation is not regarded as important.

Health education about personal hygiene

- To cover the mouth when coughing and sneezing.
- To use a handkerchief or paper for disposal of nasal secretions and sputum.
- Not to spit on the ground in or outside the house.
- Not to share cigarettes, drinking bowls, or eating utensils.
- To seek treatment for any chronic cough.

Wearing of masks in hospitals

A number of pathogenic organisms are present without symptoms or signs in the throats and noses of many people, including hospital staff. To avoid spreading infection to susceptible patients such as premature

babies and patients for operation, masks covering nose and mouth should be worn when dealing with these high risk groups. The masks should be washed frequently and dried in the sun.

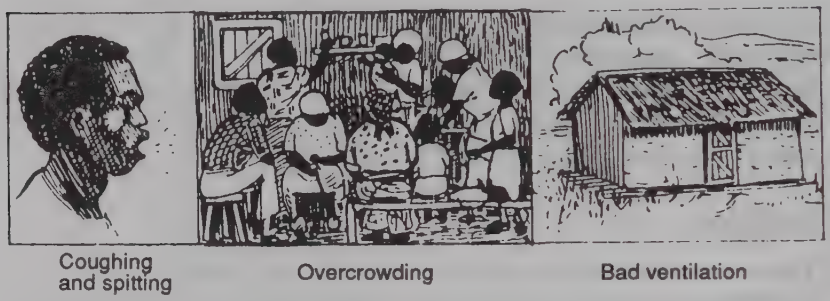


Figure 8.2 **Spread of airborne diseases**

Immunizations

A number of vaccines are available for the prevention of airborne diseases. Diseases for which vaccines exist are:

- Tuberculosis;
- Smallpox (this was eradicated by WHO immunization campaigns in 1977 and accepted as eradicated in 1980);
- Measles;
- Whooping cough;
- Diphtheria;
- Influenza;
- Meningococcal meningitis;
- Rubella;
- Mumps.

The vaccines for the last four diseases are not generally available in most of Africa, but meningococcal meningitis vaccine is used in epidemics.

Action

- Stress in the local development committee the importance of building spacious and well-ventilated houses.

- Immunize all children in the community served by your health unit.
- Inspect the conditions in the local prison and discuss the possibilities of avoiding overcrowding by building more prison wards.
- Give health education about immunization and personal hygiene and explain the reasons.

The general approach to acute respiratory infections (ARI) at the health centre is shown in Table 8.3.

Table 8.3 Classification of ARI in children at health centre or dispensary

1. Very severe [for admission] Signs and symptoms: — Cough or wheeze — Cyanosis — Not able to drink — Respiratory rate over 70/minute **Management:** — Admit — Give oxygen — Give chloramphenicol or ampicillin or amoxycillin
2. Severe [for admission] Signs and symptoms: — Cough and chest indrawing but no wheeze — Wheeze and very fast breathing (over 70/minute) or if has any of following: — Stridor at rest (croup, epiglottitis, diphtheria, foreign body) — Adherent grey membrane in throat (diphtheria) — Convulsions or fits — Sometimes stops breathing (apnoeic attacks) — Difficult to wake up — Severe dehydration **Management:** — Admit — Give oxygen — Give benzyl penicillin

Continued on page 270

Table 8.3 *Classification of ARI in children at health centre or dispensary* (continued from p. 269)

3. **Moderate** [antimicrobial treatment at home plus supportive treatment]
Signs and symptoms:
— Cough and fast breathing (over 50/minute)
— With no chest indrawing
— Wheeze and fast breathing (50–70/min)
— Red eardrum or discharge for less than 2 weeks
— Purulent pharyngitis with large and tender lymph nodes in neck
Management:
— Cotrimoxazole or amoxycillin or ampicillin
— Supportive treatment at home (see below)

4. **Mild** [only supportive treatment at home]
Signs and symptoms:
— Cough, hoarseness
— Wheeze or fever. No fast breathing (less than 50/minute)
— Stridor which goes away when not crying and at rest
— Red throat
— Blocked or running nose
— Ear discharge for more than 2 weeks
Supportive management:
— Fluids. Breast-feeding. Frequent small feeds
— Reduce temperature. Paracetamol. Cleaning nose. Cleaning ear discharge

COMMON COLD

1. The common cold is the most frequent cause of medical consultation throughout the world.

2. Occurrence and importance

An individual can expect to have one or more colds every year. Factors influencing the occurrence and severity of common colds in Africa are:

- Overcrowding;
- Inadequate clothing;
- Young age (below 5 years);
- Cold climates, especially in high-altitude areas.

Common colds are important in the community because they are a frequent cause of loss of man-hours at work and absenteeism from school.

People who suffer from colds may have serious secondary bacterial complications such as sinusitis, otitis media, laryngitis, tracheitis and bronchitis.

Prevention of colds: When sneezing, coughing and having a running nose, do not go visiting in crowded places and avoid giving the cold to small babies.

3. Epidemiology

The common cold is caused by a number of viruses all giving the same clinical picture. The virus is spread by droplets and by indirect transmission such as by freshly infected articles (handkerchiefs).

4. Clinical picture

- Nasal discharge: first watery, then mucoid and purulent.
- Sneezing.
- Some fever may be present.
- Malaise.
- Headache. The headache is caused by obstruction of the canals leading to the air-containing structures in the skull, sinuses, and middle ear. Blockage of these contributes to secondary bacterial infections like sinusitis and otitis media.
- Dry or painful throat.
- Conjunctivitis.

Differential diagnosis

- Early measles.
- Early whooping cough.
- Hay fever (allergy).

5. Management of the individual patient

Since the common cold is caused by a virus, no effective treatment is available. The headache may be relieved by aspirin, or paracetamol. Ephedrine nose drops may give temporary relief, but delay recovery and cure. After their effect has worn off, the congestion usually gets worse because of the rebound phenomenon. Only breast-feeding babies and children with otitis media benefit from 0.25% ephedrine nasal drops.

Antibiotics are, of course, not indicated. When they are given to prevent secondary bacterial infections, they do not reduce the number of complications but select the bacilli causing the complications.

6. Prevention and control

See Introduction, URTI, page 265.

7. Action

Use every opportunity to immunize all children brought to you for treatment of common cold. The common cold is not a contra-indication to any immunizations in the EPI programme and MCH clinic.

8. Summary

The common cold is an acute infection of the upper respiratory tract. It is characterized by coryza and malaise. Fever is not common. It is important because of the high attack rate and secondary bacterial infections, especially in children. Treatment is symptomatic.

SORE THROAT

1. This is an acute inflammation of the throat.

Synonyms: tonsillitis, pharyngitis.

2. Occurrence and importance

Equally common as the common cold and may accompany it. Streptococcal tonsillitis may be followed by allergic reaction in some cases, which may result in:

- Acute glomerulonephritis
- Acute rheumatic fever
- Rheumatic heart disease.

3. Epidemiology

Most sore throats are caused by viruses. In some cases the causative organism may be streptococci.

4. Clinical picture

- Painful swallowing.
- Pharynx, tonsils, and adenoids may be red and swollen.
- Differentiation between streptococcal and viral throat infection can be made with the aid of Table 8.4.
- When the sore throat is caused by herpangina, small painful blisters are present on the mucosa of the throat and the cheeks.

Table 8.4. **Differential diagnosis of viral and streptococcal sore throat**

Disease	Signs and symptoms		
	High fever	Pus on tonsils	Marked swelling of cervical glands
Streptococcal tonsillitis	+	+	+
Viral tonsillitis	–	–	–

5. Management of the individual patient

Streptococcal: Procaine penicillin fortified (PPF) 0.8 mega units for 10 days to prevent rheumatic fever.

A single dose of benzathine penicillin G 1.2 mega units is also effective. Patients with rheumatic fever or established rheumatic heart disease should be protected against further infection with either benzathine penicillin 1.2 mega units every 3 weeks till age 21 or oral penicillin V 125-250 mg daily.

Viral: Gargle with salt water, and take paracetamol for relief of fever and pain. Antiseptic lozenges are not of much use.

Herpangina: No definitive treatment—usually self-limiting within a few days. Give symptomatic treatment as for viral sore throat, above.

6. Prevention and control

See Introduction, and page 266.

7. Action

- Check the use of penicillin in your health centre.
- Discuss with your staff the (strict) indications for antibiotics.
- Examine routinely all patients complaining of sore throat.

8. Summary

Sore throat is a common condition caused by viruses or streptococci. Treat according to cause. Remember rheumatic fever may be a complication of streptococcal infection and that patients will require continuous antibiotic prophylaxis.

INFLUENZA

1. Influenza is an acute respiratory tract infection of specific viral origin characterized by sudden onset of headache, myalgia, fever, prostration and cough.

Synonym: "flu".

2. Occurrence and importance

Influenza is important because of its high attack rate, and the seriousness of its complications. Epidemics are associated with a rise in general mortality (excess mortality).

Deaths occur especially in the elderly and in those with chronic diseases.

Influenza can occur in pandemics (1889, 1918, 1957, 1968, 1974) and in major epidemics with intervals of 2–3 years for influenza A and 4–6 years for type B. Type C occurs sporadically and in minor epidemics.

3. Epidemiology

The incubation period is 1–3 days.

4. Clinical picture

Sudden onset of:

- Headache;

- Malaise;
- Muscle pains;
- Fever;
- Prostration;
- Congested nose;
- Cough.

Complications

- Bronchopneumonia caused by *Haemophilus influenzae* or *Streptococcus pneumoniae* or *Staphylococcus aureus*.
- Sinusitis.

Differential diagnosis

- Malaria.
- Relapsing fever.
- Other viral URTI infections.

5. Management of the individual patient

- Bed rest.
- Paracetamol may relieve pain and diminish fever.
- Antibiotics if signs of pneumonia.

6. Prevention and control

Immunization is possible, but due to its expense and the limited protection it gives it is not considered cost effective in Africa.

7. Action

See Introduction, page 266–269.

8. Summary

Influenza is an acute infection of the upper respiratory tract characterized by general symptoms. It is important because of its rapid spread, high attack rate in all age groups, and secondary bacterial infections leading to excess mortality. Control by immunization is not universal at the moment because of the cost of the vaccines.

ACUTE LARYNGO-TRACHEO-BRONCHITIS (LTB)

1. An acute bacterial or viral infection of the larynx, trachea, and bronchi, resulting in narrowing of the airways, which may easily lead to obstruction, especially in children.

Synonym: Croup.

2. Occurrence and importance

The disease is usually due to a variety of viruses or a complication of measles. In children, the larynx, trachea and bronchi are generally narrow. Inflammatory swelling of these structures due to infection, and the sticky mucus thus produced, may easily obstruct the airways. This can cause death. In adults, the larynx, trachea and bronchi are wider and they permit some inflammataory swelling without causing severe distress or death.

3. Epidemiology

LTB is usually of viral origin, but it may be complicated by secondary bacterial infections like *Haemophilus influenzae*.

4. Clinical picture

The first sign is a hoarse voice. The child may develop a barking cough and dyspnoea rather suddenly. This dyspnoea is inspiratory in nature. There is noisy inspiration (stridor). When the obstruction is severe, retraction of intercostal and supraclavicular spaces can be seen during inspiration. The child becomes restless and fights for air.

5. Management of the individual patient

- Put the child in a well-ventilated airy place. Avoid the traditional method of treatment with steam to increase humidity because this may aggravate the condition as the heat generated causes dehydration.
- Rehydrate the patient with oral or intravenous fluids. Maintain adequate hydration.
- *Antibiotics:* use broad-spectrum antibiotics (e.g., ampicillin) to prevent secondary bacterial infection.

- *Corticosteroids:* the effect of these drugs in this acute condition is doubtful, but hydrocortisone can be given.
- *Monitor vital signs:* a rising heart rate indicates increasing hypoxaemia.
- *Intubation* may be life-saving but requires a skilled paediatric anaesthetist and so should be done in hospital. Remove tube after 2–5 days.
- *Tracheostomy:* facilities for immediate tracheostomy should be available when intubating; again this is only possible in hospital. This procedure needs continuous experienced nursing care: if this is not available, tracheostomy will surely cause the death of the child.
- *First-aid measure:* insertion of a wide-bore intravenous cannula through the cricothyroid membrane into the trachea may be life-saving in emergencies.

6. Prevention and control

See Introduction, page 266. Measles as an underlying cause of LTB is preventable by immunization, usually given at 9 months of age.

7. Action

Check your MCH services: ensure that all eligible children receive measles vaccine.

8. Summary

Acute laryngo-tracheo-bronchitis is characterized by inspiratory stridor. It usually occurs as a complication of other respiratory tract infections, especially measles. Treatment is by removing the obstruction, adequate fluids and antibiotics. It can be controlled by preventing measles and better nutrition.

PNEUMONIA (IN CHILDREN AND ADULTS)

1. Pneumonia is an acute respiratory infection with fever, cough, and dyspnoea. Pneumonia should be strongly suspected in an acutely ill patient with severe dyspnoea and tachypnoea.

(Refer to Table 8.3 for classification of ARI in children.)

2. Occurrence and importance

Pneumonia is a common disease of infancy and old age. Pneumonia is by far the commonest cause of death in patients admitted to most hospitals in Africa.

Before the time of antibiotics the case fatality rate was between 20 and 40%. At present the fatality rate is about 5%. Low-birth-weight infants are very susceptible and have high mortality.

Factors predisposing to pneumonia are upper respiratory tract infections, especially influenza in the elderly and measles and whooping cough in children.

These infections cause damage to the epithelium of the lungs and so clear the way for bacterial superinfection: acute and chronic bronchitis, and sinusitis can all be complicated by pneumonia. Other conditions associated with pneumonia are aspiration of a foreign body, and lung oedema.

Patients who are chronically ill and bedridden do not ventilate the lower parts of the lungs very well. Therefore they easily develop pneumonia of the lower lobes (hypostatic pneumonia). Pneumonia is serious in infants who are anaemic from malaria or hookworm infection.

3. Epidemiology

Pneumonia can be caused by several organisms. In Africa, the following bacteria are important and require antibiotic treatment:

- Pneumococci;
- Streptococci;
- Staphylococci;
- Klebsiellae;
- *Haemophilus influenzae*.

Viruses which cause upper respiratory tract infections can also cause pneumonitis.(See sections on upper respiratory tract infections, measles and chickenpox.)

The specific lung infections that may occur in anthrax, plague, and tuberculosis are considered elsewhere in this book.

Pneumonitis is a feature of the passage of larvae of ascaris, strongyloides, and hookworm through the lungs. A chemical pneumonia is the main danger resulting from kerosene poisoning, especially if ill-advised attempts are made to wash out the stomach.

Because of its close association with measles and influenza, the incidence of pneumonia follows the pattern of these diseases.

Small outbreaks of pneumococcal pneumonia can occur in institutions, barracks and on board ships where people are crowded in their living and sleeping quarters.

Many healthy people are carriers of organisms which cause pneumonia. Transmission is by droplet spread, direct oral contact, or indirectly through freshly infected articles. This results in the pneumococci colonizing the nasopharynx but not necessarily in disease.

4. Clinical picture

The key symptoms of pneumonia are cough, fever, and tachypnoea (respiratory rate of over 50). Key signs are in-drawing of the intercostal muscles, and flaring of the alae nasi in children.

Bronchopneumonia

This is the most common form of pneumonia. It is very often a complication of an existing disease: whooping cough, measles, typhoid, influenza, acute or chronic bronchitis, sinusitis.

Bronchopneumonia also occurs in chronically ill patients (hypostatic pneumonia), after aspiration, in lung oedema, in bronchostenosis (bronchial carcinoma) and after lung infarction.

The onset of bronchopneumonia is usually not abrupt. There may be remittent, intermittent, or continuous fever; herpes labialis is a rare feature, unike in lobar pneumonia. The sputum is purulent but there is no brick-coloured (rusty) sputum.

Findings: Increased vocal fremitus, bilateral areas of impaired resonance, variable fine crepitations, especially over the lower lobes and bronchial breathing with bronchophony. WBC count is high, with many polymorphs.

Bilateral signs

- Palpation — increased vocal fremitus.
- Percussion — impaired resonance.
- Auscultation — bronchial breathing, variable fine crepitations.

Lobar pneumonia

The onset is often very sudden in a previously healthy person, with chills, stabbing pains in the chest and dyspnoea. Fever is high and continuous (39–40°C); general symptoms are prostration, anorexia, headache, insomnia and vomiting.

The cough is dry on the first day but later becomes productive. On the third day typical sputum is produced which is glassy, sticky and with a rusty-red colour (brick-coloured sputum). About half of the patients have herpes labialis.

Chest examination will show signs of consolidation over one lobe: increased vocal fremitus, dullness on percussion, bronchial breathing, and fine or coarse crepitations.

In the very old there may be little fever. Diagnosis depends on finding signs of consolidation, and a high WBC count with many polymorphs.

Consolidation in one lobe

- Palpation — increased vocal fremitus.
- Percussion — dullness.
- Auscultation — bronchial breathing, fine crepitations.

Differential diagnosis

- Malaria can mimic pneumonia — exclude by doing a blood slide.
- Typhoid also presents with fever and cough. If suspected, refer for blood culture.
- Bronchitis. Acute bronchitis presents with fever and cough, but gaseous exchange is not disturbed and there is no dyspnoea and/or cyanosis. Crepitations or rales originate from the bronchi and are

coarse. Coarse crepitations should be differentiated from fine crepitations originating in the alveoli. Bronchitis is often wrongly diagnosed as pneumonia, and treated with penicillin which is often not effective. If antibiotics are indicated in the treatment of bronchitis, cotrimoxazole or broad-spectrum antibiotics should be used. Think of HIV/AIDS. A variety of lung infections become more serious and common as immunity wanes with HIV infection.

Complications of pneumonia

- Delayed resolution (watch out for TB, or foreign body in the lungs).
- Pleural effusion.
- Empyema, lung abscess.
- Heart failure and atrial fibrillation.
- Pneumococcal meningitis, pericarditis, or arthritis.

5. Management of the individual patient

Frequently a patient with mild pneumonia can be treated as an outpatient, particularly if he is living near the health unit. Pneumococcal pneumonia responds very well to penicillin.

Dyspnoeic patients with severe pneumonia should be admitted for initial treatment with crystalline penicillin 0.5–1.0 mega units 6-hourly. Oxygen is useful when there is cyanosis, restlessness or delirium (indicating cerebral hypoxia), especially in children.

If there is no improvement within 48 hours, the causative organism is most likely not sensitive to penicillin (e.g., *H. influenzae, Staph. aureus, Klebsiella pneumoniae*): change to ampicillin or Erythromycin. In neonates it is always wise to start with broad-spectrum antibiotic cover.

If the patient responds well to the treatment and/or can be treated as an outpatient, give PPF 0.4–1.2 mega units daily, depending on the age of the patient.

Dehydration should be prevented by ample fluid intake or be corrected if it has already occurred.

Adequate humidity is important, especially in dry climates. Humidity can be raised by hanging wet bed clothes (wrung out in water) with a fan blowing on them, if electricity is available.

Expectorants are not useful. Cough suppressants are often added to cough mixtures. These drugs will prevent mucus being coughed up and so will prolong the duration of the disease. Cough suppressants may only be of help in a very tired patient who needs rest at night. They act by sedation. As a rule, avoid cough mixtures because they are of no medical value in the treatment of pneumonia. Purchases only benefit the sellers and the manufacturers.

When a patient with pneumonia does not improve on treatment, examine the sputum for TB

Benzathine penicillin and oral penicillin V are not effective in treating pneumonia because, when administered, they do not achieve the required high serum levels.

Cotrimoxazole is cheap and effective against *Strep. pneumoniae*, *H. influenzae*, and sometimes *Staph. aureus* and can be given twice a day. Amoxycillin or ampicillin are other second choices.

Benzyl penicillin given 6 hourly is effective in severe pneumonia. Chloramphenicol is the drug of choice for very severe haemophilus infections — given intramuscularly if the child cannot drink. It is not used in children under 2 months of age because it may depress the baby's bone marrow, causing the grey baby syndrome.

6. Prevention and control

The control of pneumonia is based on the general principles governing control of the airborne diseases.

- Immunization to prevent diseases frequently complicated by pneumonia, such as measles and whooping cough, is of utmost importance — watch cases for signs of pneumonia.
- Mortality can be greatly reduced by early diagnosis and treatment, especially in children and the elderly; diagnosis should be made at community, dispensary and health centre level; prompt adequate therapy should be started in these units.

- A health education programme should be started and parents and community health workers should be taught:
  - How to distinguish mild and serious ARI;
  - What supportive measures can be given at home;
  - The importance of breast-feeding and immunization;
  - Where to go if there are signs of severe ARI;
  - The need to avoid restriction of food and fluids;
  - The need to give extra meals after an illness in order to achieve "catch-up" growth;
  - The value of green vegetables as sources of vitamin A.

Early treatment reduces mortality

7. Action

In all patients with acute respiratory infection, grade severity and give appropriate treatment.

8. Summary

Pneumonia is characterized by cough, fever, and rapid respiratory rate (over 50/minute) in children. It often occurs as a complication of upper respiratory tract infections. The drug of choice is penicillin by injection. Patients are treated as inpatients or outpatients, depending on the severity of their symptoms.

Control of pneumonia also depends on control of predisposing conditions such as measles.

The high mortality rate of pneumonia in children can be greatly reduced by early diagnosis and treatment. Parents and community health workers have an important role to play in ensuring early recognition of the disease and adequate treatment or referral.

HAEMOLYTIC STREPTOCOCCAL DISEASE

1. Streptococci are part of the bacterial flora present on the skin, and in the nose and throat of healthy people. Some types, e.g., the haemolytic streptococci, may cause disease.

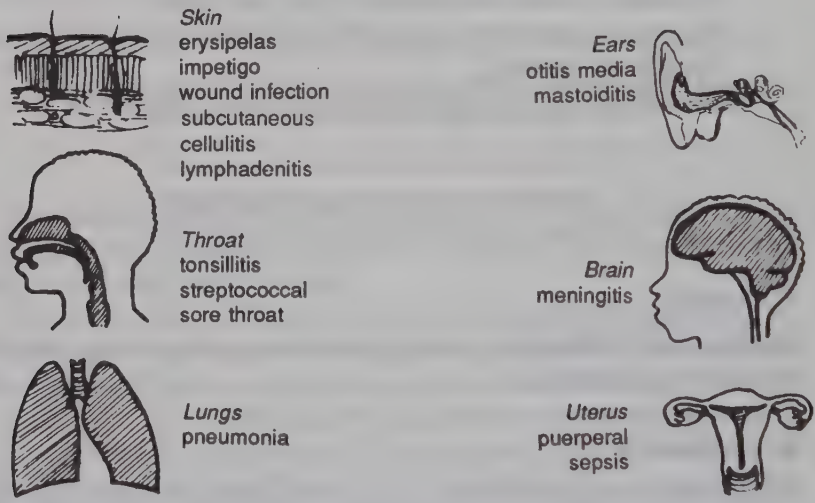


Figure 8.3 **Different types of streptococcal infection**

2. Occurrence and importance

Diseases caused by haemolytic streptococci are common. Scabies lesions are often secondarily infected with streptococci.

In a small proportion of children between the ages of 5 and 15 years, streptococcal infections in the upper respiratory tract and skin may be followed by secondary effects on the heart and kidneys, respectively. These are thought to be allergic (auto-immune) reactions to streptococcal infections. These reactions may result in acute rheumatic fever, involving the heart and joints, or acute glomerulonephritis, involving the kidneys. In rheumatic fever the joints are swollen and the heart inflamed. The resulting carditis may

be followed by heart-valve damage, e.g., mitral stenosis, which may manifest as heart failure later in life. Acute glomerulonephritis is often self-limiting but may proceed to chronic glomerulonephritis and kidney failure in a small proportion of children.

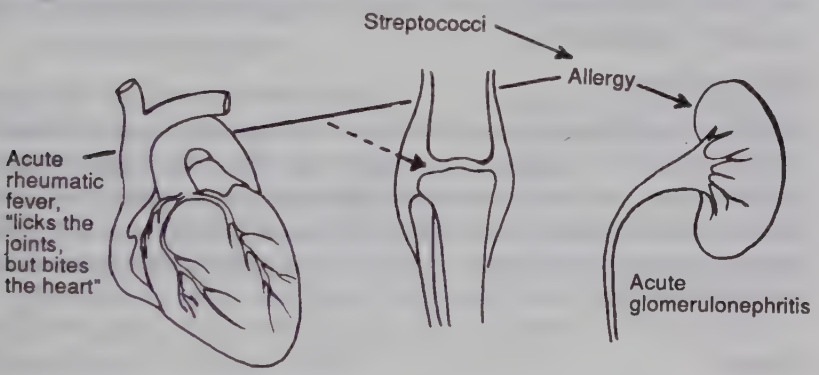


Figure 8.4 **Allergic complications after infection with streptococci**

3. Epidemiology

Streptococci are spread by skin-to-skin contact or by droplet infection. Healthy carriers may harbour streptococci in the nasopharynx.

Explosive outbreaks of streptococcal sore throat may follow ingestion of contaminated milk or other food.

4. Clinical picture

This depends on the site of infection.

Throat

Tonsillitis and sore throat, see Acute respiratory tract infections (page 272).

Skin

Erysipelas is a rapidly spreading infection of the skin with systemic disturbance. The onset is acute with high fever, headache and vomiting.

The skin lesion is a painful, spreading swelling with a well-defined raised edge. Impetigo consists of superficial vesiculopustular lesions in the epidermis. The lesions rapidly burst and the resulting skin defects are covered with crusts. Lesions heal without scarring. Impetigo is very often secondary to other skin diseases, e.g., impetigo of head and neck in pediculosis capitis (head louse). Impetigo may also be caused by staphylococci.

Subcutaneous

Cellulitis — inflammation of skin and subcutaneous tissue. The area of skin affected is oedematous, shiny and hot. The edges of the inflamed area are sharply marked. Regional lymph glands may be inflamed and the infection may be complicated by septicaemia. When cellulitis occurs in the hands or feet, tendon sheaths and bones may become involved.

Uterus

Puerperal sepsis — invasion of the uterus by streptococci happens mostly during childbirth and abortion. This results in endometritis, presenting as foul-smelling lochia, with fever. When the fallopian tubes and peritoneum are invaded there is high fever, and severe low abdominal pain.

Ears

Otitis media occurs as a complication of upper respiratory tract infections. Blockage of the eustachian tubes prevents drainage of the middle ear. As a result, pus accumulates until the drum perforates. Fever and pain are the main symptoms. Mastoiditis is a complication of otitis media. There is tenderness and swelling over the mastoid, often associated with fever and vomiting. Meningitis may follow mastoiditis.

Lungs

Pneumonia (page 278).

Brain

Meningitis (page 291).

Diagnosis

Gram's stain of pus, sputum, or CSF, will show Gram-positive cocci in chains.

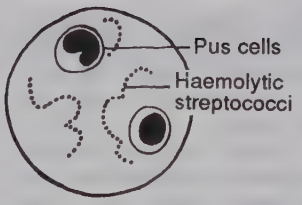


Figure 8.5 **Microscopic appearance of streptococci**

5. Management of the individual patient

Superficial skin infections and mild, small wound infections do not need treatment with antibiotics. Impetigo, if severe, can be treated with an antibiotic by injection or orally. Gentian violet paint 1% is now seldom recommended.

All other streptococcal conditions and infections of the newborn are potentially dangerous and should be treated with antibiotics. Penicillin is the drug of choice.

6. Prevention and control

General hygiene (washing with soap and water) will reduce predisposing infections like scabies and louse infestation.

Aseptic techniques in hospitals will reduce wound infections and puerperal sepsis. For control of tonsillitis and pneumonia, see acute respiratory tract infections above. For control of meningitis, see below.

7. Action

- Isolate septic patients in a septic ward. If you do not have a separate ward, make a septic corner in the ward.
- Place a basin with water and soap in each ward to make it easy for everyone to wash their hands regularly.
- Allocate enough forceps and scissors for each individual patient.

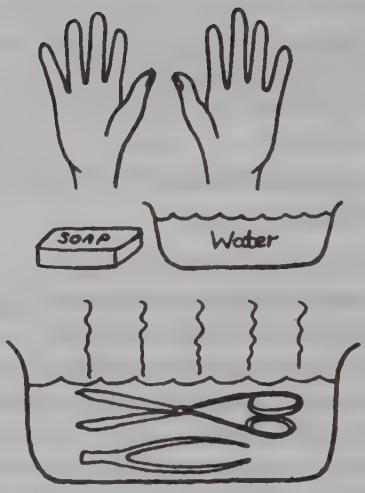
Note

Wiping forceps with an antiseptic solution does *not* kill the bacteria immediately. The quickest way to kill bacteria is to boil the instruments.

Do not dispose of dirty dressings in a forgotten corner near the health centre within flying distance of flies.

Dispose of dirty dressings by wrapping them in used paper (used stationery or newspapers) and burning them daily in your incinerator.

It is useless to give health education on environmental sanitation unless your health centre is a good example itself.



Boil instruments to kill bacteria

- Make the wards, dressing rooms and theatre, fly-proof. It is better for the patients, and in the long run for your drug vote, to invest some money in screening material rather than "covering" all patients with antibiotics.

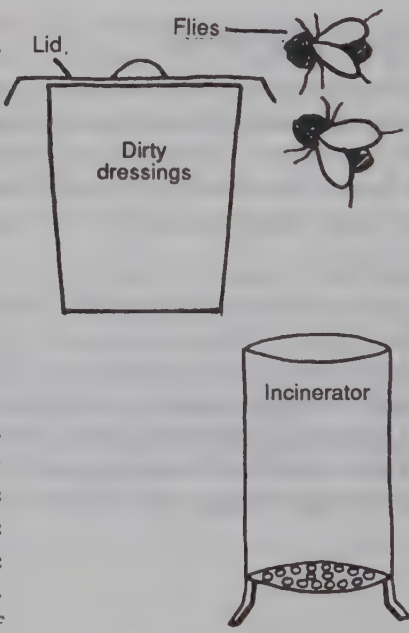


Figure 8.6 **Prevention of streptococcal infection**

8. Summary

Haemolytic streptococcal disease may occur in skin and inner organs. Dangerous complications to the heart and kidney may arise due to an allergic response to streptococci in some individuals. The drug of choice for streptococcal infections is penicillin.

STAPHYLOCOCCAL DISEASES

1. Staphylococci are bacteria which produce different clinical pictures depending on where they are. They often produce pus.

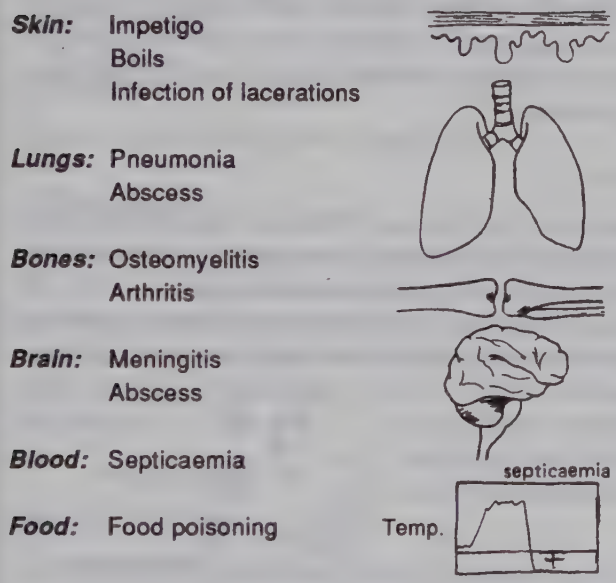


Figure 8.7 **Staphylococcal infections**

2. Occurrence and importance

Diseases caused by staphylococci are common.

Infections of the skin are of minor importance, but they may be the portals of entry for the bacteria into the inner organs.

Staphylococcal infections of the internal organs are dangerous: they are especially likely in people with a weakened defence system; after viral infection; in the chronically ill; and in patients undergoing major surgery.

3. Epidemiology

Staphylococci are part of the normal bacterial flora living on the skin, in the nose or in the throat. Between 30 and 40% of healthy persons are carriers of harmful staphylococci. They may produce staphylococci in their nasal secretions and infect others (this is the reason why face

masks must be worn during operations). Due to the overuse of antibiotics in hospitals, many strains of staphylococci have now developed resistance to most antibiotics.

These resistant strains of staphylococci may cause very serious infections in hospitalized patients. Patients with purulent wounds may infect all operation patients. The staphylococci may be spread by flies, by the fingers of the nursing and medical staff, and by invisible droplets containing bacteria.

4. Clinical picture

This depends on the site of the infection. When the bacteria are only causing superficial infection of the skin, toxins are not absorbed into the bloodstream and no signs of general infection appear.

If an unripe abscess is incised, or if boils are squeezed, bacteria may enter the bloodstream and give rise to a septicaemia with fever, malaise, and headache.

When the bacteria are localized in the internal organs, the clinical picture is related to the disturbance of these organs.

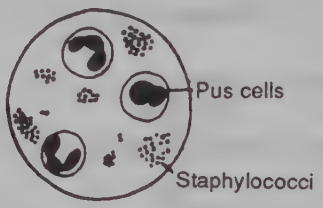


Figure 8.8 **Microscopic appearance of staphylococci**

Diagnosis

Gram's stain of pus, sputum, or CSF will show Gram-positive cocci in clusters.

5. Management of the individual patient

- Superficial skin infections: no systemic antibiotic treatment is needed. Local application of antiseptics, e.g., flavine, eusol, gentian violet 1%, hibitane, and frequent changes of dressings will do.
- In case of involvement of internal organs: quick treatment with high doses of broad-spectrum antibiotics, such as erythromycin, tetracycline and chloramphenicol, is indicated. Some of the penicillinase-resistant or broad-spectrum penicillins such as cloxacillin may have to be used.
- In a newborn with generalized skin infection, systemic cloxacillin or ampiclox treatment is indicated.

6. Prevention and control

- Known patients with purulent lesions must be isolated.
- Dressings from purulent wounds must be handled with care.
- Aseptic techniques should be taught to, and practised by, all nursing staff.

7. Action

See streptococcal diseases (page 287).

8. Summary

Staphylococcal infections are common. They are dangerous when they occur in internal organs. Sources of infection are patients with purulent wounds, or healthy carriers. Control consists of strict application of antiseptic measures. Resistance to commonly-used antibiotics is common and sensitivity tests must be done for systemic infections.

MENINGITIS

1. Meningitis is an inflammation of the meninges (pia and arachnoid membranes) which cover the brain. It may be either acute or chronic, purulent or aseptic (sterile).

2. Occurrence and importance

All forms of meningitis occur in Africa. Meningococcal meningitis can occur sporadically or in the form of epidemic outbreaks, especially in crowded institutions such as barracks, camps, and prisons. Very large epidemics often sweep across Africa in the drier western and central sub-Saharan zone and also move into Sudan, northern Uganda, Ethiopia and northern Kenya and northern Tanzania.

In the cold dry season, the temperatures drop to very low levels at night. This makes people crowd together in badly ventilated rooms. Their mucous membranes are also irritated by dust and by the smoke of firewood. Under such conditions the meningococci spread easily.

Other forms of meningitis always occur sporadically as they are usually complications of other diseases. All forms can obstruct normal CSF flow and so cause hydrocephalus, and can damage cranial

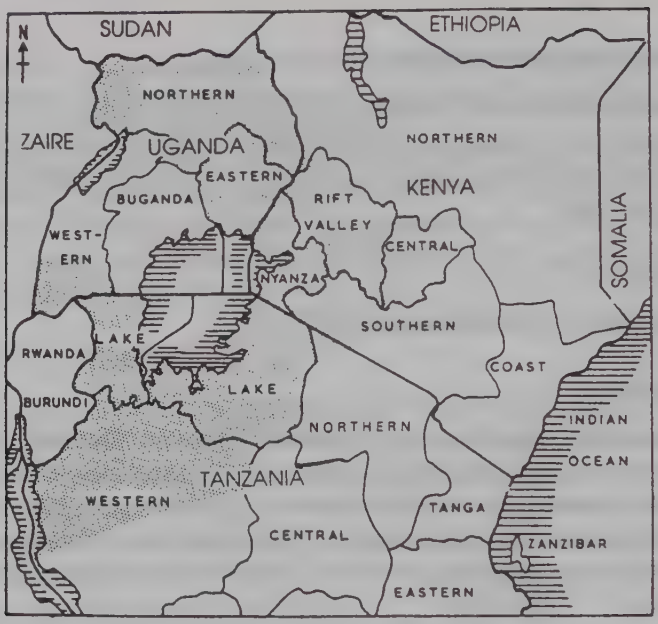


Figure 8.9 The meningitis crescent in east Africa

nerves proximally by adhesions which can result in paralysis or loss of senses (e.g., deafness, blindness). If meningitis is properly treated, mortality should not exceed 5%, but if treatment is delayed, mortality may be over 30% and commonly those who do survive have permanent damage.

Table 8.5. **Causative and predisposing factors in meningitis**

Causative organism	Age group most at risk	Predisposing condition
Pneumococci Streptococci	Adults	Mastoiditis, otitis media, sinusitis, pneumonia, head injury, puerperium, pregnancy
H. influenzae	Children	Respiratory tract infection, otitis media, mastoiditis
Meningococcus	Children, young adults	Overcrowding
M. tuberculosis	Children	Malnutrition
Salmonellae	<2 years	Diarrhoea or septicaemia
Virus	Children	Epidemics of mumps, measles, polio, chickenpox, and other viruses

3. Epidemiology

A wide variety of organisms may cause meningitis (see Table 8.5). Meningitis should be differentiated from meningism or meningeal irritation as is seen, for example, in malaria, by doing a lumbar puncture. Chronic meningitis may be caused by TB.

The acute forms of meningitis may be caused by several viruses and bacteria. Aseptic or viral meningitis differs from bacterial meningitis because it is usually a self-limiting disease or associated with other clinical entities such as mumps, poliomyelitis and measles.

Acute bacterial meningitis may be caused by (in order of frequency):

- Pneumococci (*Streptococcus pneumoniae*);
- Haemophilus influenza (*Haemophilus influenzae*);
- Meningococci (*Neisseria meningitidis*);
- Coliforms (*Escherichia coli*);
- Salmonella (*Salmonella typhi*) ;
- Staphylococci (*Staphylococcus aureus*);
- Streptococci (*Streptococcus pyogenes*).

Twenty-five per cent of healthy people may carry meningococci and the other organisms mentioned above; therefore healthy carriers are common.

Transmission occurs by direct contact and droplet spread of discharges from nose and throat of infected persons (mostly carriers).

Epidemic meningococcal meningitis is caused by *N. meningitidis*. It is divided into serotypes, of which A, B and C are the most important. The large outbreaks in Africa have been mostly due to group A, but group C has also been found in Nigeria, Niger and Burkina Faso. Group B has been found in the Sudan. A combined vaccine is available against the organisms of groups A and C.

The incidence of disease in epidemics is highest in the 5–15 year age group (the school age). Major epidemics occur in 5–10 year cycles.

4. Clinical picture

The onset is acute with headache, fever and often rigors. The headache becomes severe and spreads down the neck. There may be pain in the back and limbs. In children, convulsions are common at the onset. The patient is irritable, becomes confused and drowsy, and later comatose.

Signs of meningeal irritation are clear and present at an early stage: neck rigidity, Kernig's sign and Brudzinski's signs are positive (in babies the anterior fontanelle may bulge).



Figure 8.10 **Clinical signs of meningitis**

When signs of meningeal irritation are present a lumbar puncture (LP) must be done without delay to confirm the diagnosis and start appropriate therapy immediately.

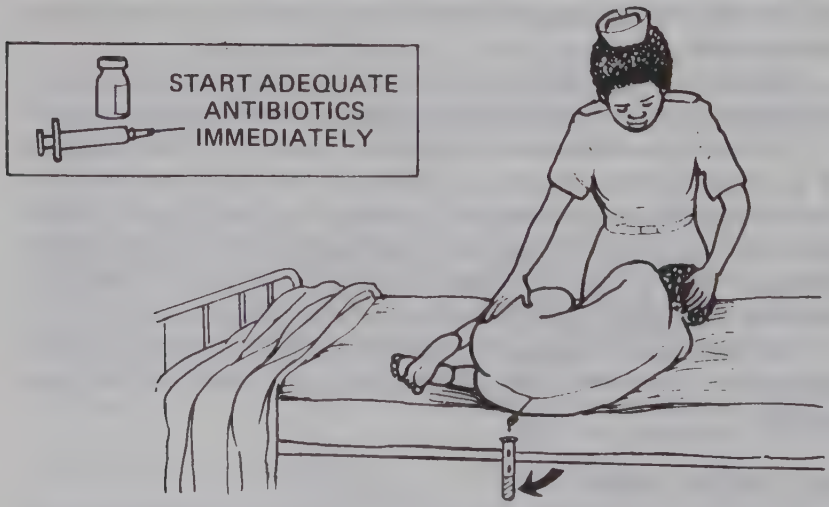


Figure 8.11 **The correct position for doing a lumbar puncture**

In meningitis the CSF is under pressure. When it is an aseptic (viral) meningitis the fluid is most often clear, though it may be turbid due to the presence of many lymphocytes (protein is then slightly increased and sugar is normal). Differentiation between such an aseptic meningitis and tuberculous meningitis may be extremely difficult (history). In bacterial meningitis the CSF is always turbid and contains hundreds of polymorph leucocytes (the protein content is greatly raised and glucose content markedly lowered).

Table 8.6 **Differential diagnosis of meningitis**

Disease	CSF	Cells	Protein	Glucose
Acute bacterial	Always turbid	Increased polymorphs	Raised	Low
TB	Opalescent or clear	Lymphocytes increased	Raised	Low
Aseptic or viral	Opalescent or clear	Lymphocytes increased	Raised	Normal
Meningism	Clear	Normal	Normal	Normal

In an epidemic of meningococcal meningitis, cases of acute meningococcaemia may occur with petechial haemorrhages and circulatory collapse. Give intravenous fluids and massive doses of crystalline penicillin.

5. Management of the individual patient

The following situations may occur:

(a) LP not possible, patient can reach a hospital within 3 hours: make a blood slide, give chloroquine, and refer the patient for LP.
(b) LP not possible, hospital cannot be reached within 3 hours: examine blood slide, give chloroquine, put the patient on chloramphenicol and crystalline penicillin, and refer to hospital.
(c) LP possible: do LP without delay, examine blood slide. Put patient on chloramphenicol and crystalline penicillin after doing LP and while waiting for results. Then continue treatment according to results (see Table 8.7), and refer the patient.

Table 8.7 **The different types of bacterial (pyogenic) meningitis**

Organism	Stained form	Arrangement	Drug of choice
Pneumococci	Gram +ve cocci	Diplococci	Crystalline benzyl penicillin 6 mega units at once 3 mega units 6-hourly
H. influenzae	Gram -ve rods	Different shapes	Chloramphenicol 1 g 6-hourly
Meningococci	Gram -ve cocci	Diplococci	Crystalline benzyl penicillin 6 mega units at once 3 mega units 6-hourly
E. coli	Gram -ve rods		Chloramphenicol
Salmonellae	Gram -ve rods		Chloramphenicol 1 g 6-hourly
Staphylococci	Gram +ve cocci	Clusters	Chloramphenicol
Streptococci	Gram +ve cocci	Chains	Crystalline benzyl penicillin 6 mega units at once 3 mega units 6-hourly

Refer all cases of purulent meningitis to hospital

Additional drugs to be given are: aspirin to reduce fever; phenobarbitone to prevent convulsions. Rehydration is also very important; feed by gastric tube if necessary.

To prevent complications like hydrocephalus and death, it is essential that antibiotic treatment is started early and is given in proper doses. Thus the most important task for the health centre is to start adequate treatment before referral to hospital. This will have a profound effect on the prognosis of all cases of meningitis.

Treat purulent meningitis for at least 2 weeks

6. Prevention and control

When an outbreak of meningococcal meningitis occurs, emphasis must be placed on careful surveillance, aiming at early diagnosis and immediate treatment of suspected cases. Health education on the dangers of sleeping in crowded, badly ventilated houses will help to reduce transmission.

It has been shown that when overcrowding is reduced and people sleep at least 3–5 ft apart, both the infection rate and the number of carriers fall.

Since there are many sulphonamide-resistant strains of meningococci, the use of sulphonamides as a chemoprophylaxis is no longer recommended. Alternative drugs such as penicillin (single dose PPF 0.6 mega units) may be used. Note that chemoprophylaxis is only indicated for exposed household members and not for the community.

Immunization with combined group A and C vaccine is very useful when there is an epidemic.

7. Action

- Order lumbar puncture needles for your health centre.
- Do LP in all cases of neck stiffness.
- Suspect meningitis in children who are drowsy or who have a fit.
- Start immediate treatment with crystalline penicillin and chloramphenicol when the CSF is cloudy.
- Refer all cases of purulent meningitis to hospital.
- Report meningococcal meningitis immediately to the DMO and discuss what other investigations or measures, such as immunization and surveillance, should be undertaken.
- Give penicillin prophylaxis to household contacts in cases of meningococcal meningitis.
- Stress the dangers of overcrowding.
- Stress the importance of early reporting and treatment.

8. Summary

Meningitis occurs all over Africa in both sporadic and epidemic forms. The disease is spread by droplet infection, the reservoir is the asymptomatic carrier. Treatment should start early with very high

doses of penicillin and chloramphenicol. Report to DMO. Correct overcrowding.

MEASLES

1. Measles is an acute general infection caused by a virus. The clinical features are the results of the reactions of the defence mechanisms of the body against the virus.

The disease is very infectious. In general it is a disease affecting children.

Synonyms: Morbilli, rubeola (note: German measles is rubella, see page 311).

2. Occurrence and importance

Measles is found all over the world and Africa is no exception. The high fatality rate of measles in Africa has been attributed to malnutrition and under-nutrition. In malnourished children the defence mechanisms are slow and weak, so the virus has more opportunity to do damage before it is arrested. Children are most likely to suffer from under-nutrition in the weaning period. This is also the time when unimmunized African children get measles most.

Another reason suggested for the high incidence of severe cases and high case fatality in Africa is that severity is related to dose of virus. In secondary cases in the same overcrowded house or compound the child has probably been in very close contact with the index case for the whole infective period and has thus obtained a very high dose of virus. It is in these secondary cases that the highest case fatality has been observed in West Africa.

In Africa most children develop measles before the age of 3 years. In Europe, most children are immunized, and the few who are not get measles after the age of five.

The overall fatality rate of measles in eastern Africa is between 3 and 5%. This is about 400 times higher than the fatality rate of measles in Europe. Thus, there is an urgent need to increase immunization coverage of under-1-year-olds to 80%.

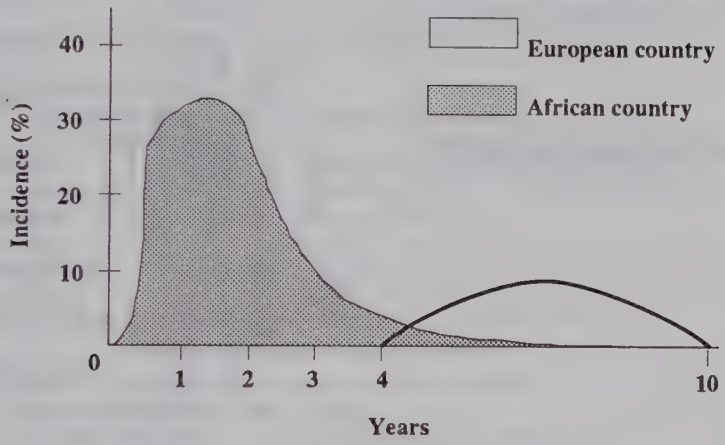


Figure 8.12 **Comparison of age distribution of measles in a European and an African country**

3. Epidemiology

Measles is spread by small invisible droplets containing virus particles. The virus particles come from the secretions of the respiratory tract of patients suffering from measles. The disease spreads very easily. Before it becomes clear that a child is suffering from measles, the disease has already spread to his contacts.

The disease gives a life-long immunity. Nearly everybody gets the disease once in his life, if he is not immunized.

One out of every 20 children may die from measles if the disease is not prevented by immunization

No routine antibiotics

Measles causes malnutrition

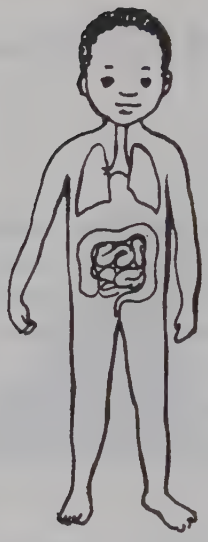


Figure 8.13 **Complications of measles**

4. Clinical picture and **5. Management of the individual patient** are covered in Table 8.8.

6. Prevention and control

The only successful way of preventing the serious complications of measles is by immunizing all children. Measles vaccine is available and the damage done by the disease justifies the expense of wastage of vaccine even if a multidose vial is opened for one child.

Measles is infectious before it is evident that the child is suffering from the disease. During the incubation period a child will have a high fever. This is a frequent reason for seeking medical attention. Many children are admitted during the prodromal stage. The result is that under-fives clinics and children's wards are centres of measles infections. Most children attending clinics or admitted to hospitals are going to be infected with the measles virus if they are not immunized.

The measles vaccine takes 8–10 days after its administration before conferring protection to the child. This is about 2 days shorter than the incubation period of measles, which is 10–12 days. It is therefore possible to protect children in under-fives clinics and wards

Table 8.8 Clinical features and management of measles

4. Clinical picture	5. Management of the individual patient
Uncomplicated measles	
Well nourished child or slightly underweight	Treat as an outpatient
Fever	Ensure proper fluid intake and good food
Conjunctivitis	Tepid sponging
Rhinitis	Paracetamol
Cough	Daily follow-up
Koplik's spots (or stomatitis)	Single dose vitamin A 200,000 units orally
Skin rash	Advise extra meals — 5 a day
Weigh all children with measles	
Complicated measles	Admit:
Child very underweight or with other signs of malnutrition	Give balanced diet with protein and energy-rich foods
Dyspnoea, nasal flaring, rapid respiration	Pneumonia (see p. 278)
Hoarseness, barking cough, inspiratory stridor	Laryngo-tracheo-bronchitis (see p. 276)
Sore mouth, inability to suck	Stomatitis (clean mouth 4–6 hourly)
Diarrhoea, vomiting	Gastro-enteritis (see page 168 or *Child Health* manual) Oral fluids
Vomiting, convulsions	Exclude malaria, meningitis Give phenobarbitone
Dryness of eyes, photophobia, hazy cornea	Xerophthalmia, give vitamin A 200,000 units/day orally, and chloramphenicol eye ointment, atropine eye ointment
Red eardrum or discharge from ear	Otitis media, treat with PPF
Persistent pneumonia	Suspect TB (see Chapter 9)

who come in contact with other children with measles by promptly immunizing them.

Give measles immunization to all children, both the healthy and the sick who have not been previously immunized

The vaccine is made from live attenuated virus. It is difficult to preserve. Exposure to heat or light, or contact with antiseptics may destroy it. The cold chain is very important and refrigerator temperatures must always be charted.

Immunization can be given to children when the passive immunity inherited from the mother has worn off — that is from the age of 9 months onwards. (If the vaccination is given between the ages of 6 and 9 months, it must then be repeated at the age of 1 year in order to ensure effective protection.)

Measles immunization (3-teeth age) is an essential part of the MCH clinic



Small child — age 9 months

Figure 8.14 **Prevent measles by immunization**

Another task of the MCH clinic is to prevent and treat under-nutrition. This is done through nutrition education to mothers and by weighing the children and plotting the weights on the growth card. When this is efficiently done, mortality following measles will go down because of better nutrition.

Reduce infant mortality by better nutrition coupled with measles immunization at 9 months of age

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7. Action

- Arrange with your DMO to get a regular supply of measles vaccine.
- Be sure your health centre refrigerator is functioning properly.
- If your health centre refrigerator is not working make arrangements with an individual to repair it and arrange for another store for your vaccine. (Even if the health centre has a good refrigerator, it might be advisable to make prior arrangements in case your refrigerator breaks down.)
- Check your cold box or vaccine carriers and ice packs periodically.
- Immunize all children at 9 months or over who are admitted to the children's ward.
- Immunize all infants who come to the health centre with other complaints. Explain to the mothers the importance of immunization and the possible minor side effects.
- Routinely immunize all eligible children, sick or healthy, coming to the MCH clinic. Discuss indications and lack of contra-indications with the staff.
- Ensure that your MCH clinic weighs all children under 3 years and that the weights are plotted on each child's growth chart.
- Teach your staff to take action when a child is underweight.
- Give vitamin A to all measles cases.

8. Summary

Measles is an important cause of under-five mortality in Africa. It can be prevented by immunization. For details see the *Child Health* manual.

WHOOPING COUGH (PERTUSSIS)

1. Whooping cough is caused by a Gram-negative bacillus *Bordetella pertussis*. The bacteria damage the cells of the respiratory tract. The damage produces a clear, very sticky mucus which blocks the lumen of the bronchioli. Children suffering from whooping cough try to get rid of the mucus by coughing. The cough is present in typical attacks which end in the characteristic whoop. The cough may last for many weeks or even months.

Synonym: Pertussis.

2. Occurrence and importance

Whooping cough is a very common disease of children in Africa.

The case fatality rate is highest in children under 1 year. Newborns get little or no protective antibodies from their mothers. Newborns develop an atypical illness which is seldom recognized as whooping cough.

Most African children will have been exposed to whooping cough early in life because of social interactions. The child is usually carried on his mother's back when she visits the market, or other families, thus exposing him to other children who might have whooping cough. Due to the "extended family" system,

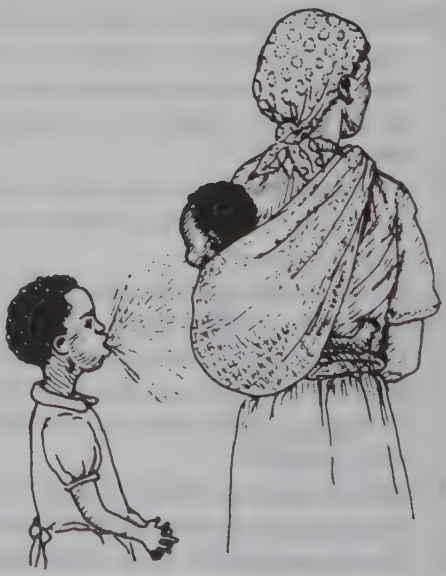


Figure 8.15 **Whooping cough: spread through exposure in early life**

an infant will always be in contact with other children.

This early exposure is responsible for the high case fatality rate of whooping cough in Africa.

The persistent cough and vomiting and the accompanying sticky mucus may prevent the child from sucking well. The supply of breast milk may then be reduced due to incomplete emptying of the breast. These factors may lead to malnutrition and death. Thus, whooping cough has serious complications which make it an important disease.

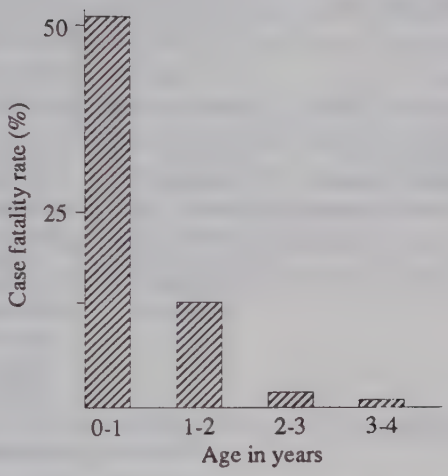


Figure 8.16 **Distribution of deaths by age group**

3. Epidemiology

Whooping cough is spread by droplets. The droplets come from secretions of the upper respiratory tract. The disease is already infectious before the typical attacks appear.

4. Clinical picture

The incubation period has a mean of 7 days and a range of 6–10 days. The clinical picture of whooping cough in children under 3 months old is quite different from that in older children.

The cough subsides slowly and, as already stated, it may last a very long time. The disease may recur after initial improvement (recrudescence).

Complications are encephalitis, pneumonia, and otitis media.

Table 8.9 **Clinical picture of whooping cough**

Clinical picture	Children under 3 months old	Children over 3 months old	Remarks
Catarrhal period, duration 14 days	Little cough Nasal discharge	Cough Nasal discharge	Like the common cold
Paroxysmal period, duration 6 weeks–3 months	Attacks of breathing arrest Cyanosis May present as a brocho-pneumonia	Typical attacks of cough with whooping followed by vomiting Sticky secretion Subconjunctival haemorrhages due to the violence of the cough	In older children diagnosis is made from the typical whoop. In young children diagnosis is frequently missed
	Young babies do not whoop		

Diagnosis

The diagnosis can be made from the history of the characteristic whoop in older children. Clinical examination may be unremarkable, but by observing the child cough one may arrive at a diagnosis. A WBC count of over 30,000 with marked lymphocytosis may also suggest the diagnosis.

Absolute lymphocytosis after infancy suggests whooping cough

5. Management of the individual patient

Supportive treatment includes:

- Maintenance of nutrition and hydration;
- Avoidance of factors which provoke attacks of coughing;
- Oxygen;
- Gentle suction;
- Avoid sedatives and cough suppressants because they may make the illness worse.

Antibiotic therapy does not shorten the paroxysmal stage of the disease. Erythromycin, ampicillin or chloramphenicol may eliminate the pertussis organism within 3–4 days, thereby shortening the period of communicability. Erythromycin may abort or eliminate pertussis when given in the first week of the disease.

It is most important that the child gets enough fluid. Without adequate fluid he will become dehydrated; dehydration also follows feeding difficulties and vomiting. The mother should be encouraged to breast-feed the child immediately after an attack as there will then be a quiet period during which the child will not cough or vomit.

Isolate known cases of whooping cough from other small children who have not been immunized.

6. Prevention and control

The only way to control whooping cough is by good immunization coverage with triple vaccine (DPT).

Start immunization against whooping cough early — at the age of 6 weeks. Give DPT 2 at 10 weeks and DPT 3 at 14 weeks. This is suggested for the following reasons:

(a) Very little or no passive immunity is inherited from the mother;
(b) It is in the first 3 months of life that whooping cough has a very high mortality.

Whooping cough vaccine is part of triple DPT vaccine. Since it is made of dead bacilli, the immune response is not as effective as after immunization with live attenuated organisms.

Therefore DPT must be given three times.

Infants or young children who are contacts of known pertussis cases may be given erythromycin as chemoprophylaxis together with immunization.

Prevent whooping cough by DPT immunization

7. Action

- Make sure you get a regular supply of DPT.
- Start DPT immunizations at the age of 6 weeks.

- Compare the number of immunizations monthly with the number of children you calculate to be in your catchment area. Make it a routine to check the immunization status of every child you see. Advise mothers to bring their children for immunization.
- Give young contacts erythromycin or ampicillin together with DPT immunization.
- Check the storage conditions of your vaccines. Make a habit of checking every day that the refrigerator is functioning properly. Defrost the refrigerator weekly. Make sure DPT vaccine has not been frozen.

8. Summary

Whooping cough is an important airborne disease. Mortality is highest in very young children. The clinical picture in young children is quite different from that in older ones. Treatment is difficult. The most important control measure is DPT immunization starting at 6 weeks.

CHICKENPOX

1. Chickenpox is a mild viral disease, characterized by fever followed by a typical skin rash.

Synonyms: Varicella.

2. Occurrence and importance

Chickenpox is a very common children's disease. It can occur in very young babies. It occurs all over Africa. It is usually a very mild disease. Case fatality rate is very low.

3. Epidemiology

Chickenpox is caused by varicella-zoster virus. The virus is spread by droplet infections containing virus particles from the respiratory tract. The scabs from the skin are not infectious. The disease leaves immunity against chickenpox, but the virus stays within the body and may reappear as herpes zoster when the immunity of the person is weakened, e.g., in AIDS, diabetes, old age, and leukaemia.

4. Clinical picture

Incubation period 13–21 days.
Prodrome: minor fever, sore throat in older children and adults.

In small children there are no prodromal symptoms and the rash is the first sign of chickenpox.

Skin

A maculopapular rash, which becomes vesicular within a few hours, appears on the trunk and then spreads to the face, scalp, and sometimes the extremities. Vesicles are oval and superficial. Pustules may form, but usually the vesicles collapse and dry after 3–4 days, leaving no scars.

Distribution

Mostly on the trunk, including the armpits; seldom on palms and soles.

Course

Groups of new pocks will appear over many days, therefore pocks at different stages of development can be found.

Complications

Unusual; secondary infection.

5. Management of the individual patient

Symptomatic — calamine lotion may relieve itching.

6. Prevention and control

As chickenpox is already infectious in the incubation period isolation will not help in controlling the disease. Since it is a self-limiting non-fatal condition, no major attempts have been made to try to prevent it.

7. Action

Prevent complications such as secondary bacterial infections.

8. Summary

Chickenpox is a common infection of children. It is a mild disease.

Herpes zoster is a late manifestation of infection with varicella virus and is now often one of the first signs of diminishing immunity in human immunodeficiency virus infection.

Chickenpox was more important before the eradication of smallpox, because of the difficulty of differential diagnosis. The worldwide eradication of smallpox, by 1977, was a very important success for an international vaccination programme.

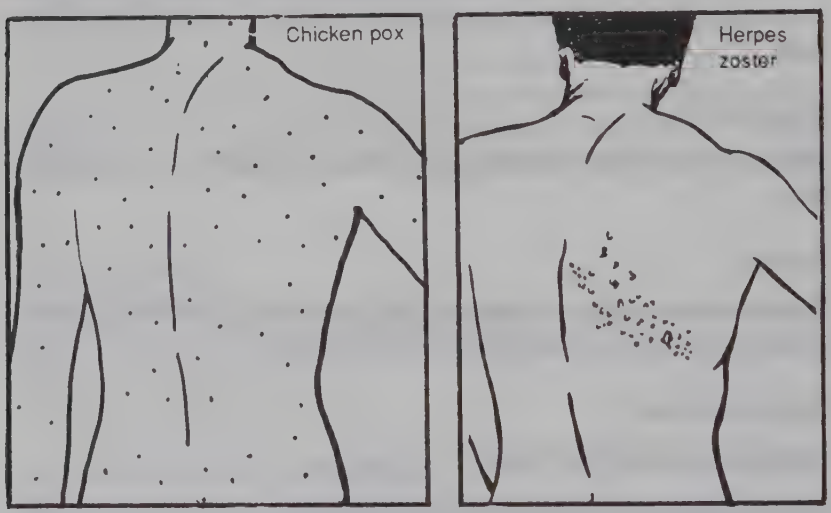


Figure 8.17 **The distribution of rash in chickenpox and herpes zoster**

HERPES ZOSTER

A clinical variant of chickenpox.

Synonym: Shingles.

Herpes zoster is a vesicular eruption of the skin confined to the area served by one nerve. The vesicles dry up into a crust in a week or so.

The condition may be very painful. Herpes zoster is caused by the same virus as chickenpox, and may break out many years after the original infection for reasons which are not always known.

Previously most cases were seen in older people or in cases of lymphomas, but now the most common reason is the waning of immunity with human immunodeficiency virus infection. So herpes zoster is seen more commonly now in people in the 20–40 year age group. It has a serious prognosis if the person's blood test is HIV positive and AIDS often follows within 2–3 years.

RUBELLA

1. Rubella is a mild febrile disease with a 3-day rash resembling the rash of measles and difficult to see on a dark skin. Often there is enlargement of the lymph nodes, typically the post-auricular, sub-occipital, or posterior cervical group of lymph nodes.

Synonym: German measles.

2. Occurrence and importance

Rubella is primarily a childhood disease. For children it is a harmless disease. It is important only when the infection occurs in females during the first few months of pregnancy when it can result in extensive foetal infection with congenital defects in the infant: congenital rubella syndrome.

In eastern Africa this congenital syndrome is not frequent, probably because most people contract the disease during childhood and develop life-long immunity. When people live further apart and living standards go up, infection during childhood will be reduced and congenital rubella syndrome could become a problem.

3. Epidemiology

Transmission is by droplet infection.

4. Clinical picture

Rubella is a very mild disease and is often not recognized. It is easily confused with measles and so people might give a history of a second

measles infection. Congenital rubella may result in one or more of the following defects:

Eyes: Cataract or microphthalmia (small eye).
Ears: Partial or complete deafness.
Heart: Patent ductus and other abnormalities.
CNS: Microcephaly (small head); mental retardation.
Hepatosplenomegaly with jaundice.
Thrombocytopenia with purpura.

Rubella is mild and harmless except in pregnancy

Table 8.10 **Differences between measles and rubella**

Differential diagnosis	Rubella	Measles
Koplik's spots	No	Yes
Suboccipital lymph nodes	Yes	No
Cough and conjunctivitis	Rare	Usual
Fever	Minimal	High

5. Management of the individual patient

No treatment is available or necessary for patients suffering from rubella. Pregnant women should be kept away from known cases of rubella.

Keep pregnant women away from rubella

6. Prevention and control

Prevention is not necessary except for pregnant women or those in whom pregnancy is a possibility. Avoidance of infection in childhood is hazardous because it may result in infection at an older age and during pregnancy; the reverse — artificial exposure of girls during childhood — is advisable and has been practised. Immunization is

possible with a live attenuated vaccine but is of less value in Africa where most children acquire natural immunity before puberty.

It is better to encourage people to expose themselves to the natural disease in childhood. This will result in natural active immunity and will prevent rubella syndrome later. Since the disease is mild in itself, natural immunity is preferable to artificial immunization.

When a pregnant mother has been exposed to a known case of rubella, passive immunization with human gammaglobulin is possible. However, it is not always successful.

7. Action

Bring young girls into contact with a rubella patient.

8. Summary

Rubella is a viral infection which is harmless except in pregnancy.

MUMPS

1. Mumps is an acute systemic viral disease characterized by fever and painful swelling of the salivary glands, usually the parotid and sometimes also the sublingual or submaxillary.

2. Occurrence and importance

Death from mumps is extremely rare but can occur as the result of a complicating encephalitis. Mumps is a relatively trivial illness in young children, but if contracted after puberty it can have serious complications.

These are: orchitis in 20–35% of the males; oophoritis in about 5% of females. Sterility may follow bilateral orchitis and oophoritis, but this is rare. Other rare complications include pancreatitis, encephalitis, unilateral deafness and thyroiditis.

3. Epidemiology

Transmission is by droplet spread and direct contact with saliva of an infected person, or indirect through freshly infected articles.

4. Clinical picture

Uncomplicated: fever, difficulty in swallowing, swelling and tenderness of salivary gland, at first unilateral, later both sides are affected. There is no rash and the fever and swelling subside in a few days. The complications are:

Orchitis: swelling and severe pain, usually one-sided.
Oophoritis: severe lower abdominal pain, vomiting.
Prostatitis: unexplained fever, dysuria.
Mastitis: pain and swelling (can occur in both sexes).
Pancreatitis: severe upper abdominal pain, vomiting, and fever.
Meningitis: common. Associated with headache, fever, vomiting, neck rigidity; prognosis is excellent, subsides in a few days.
Encephalitis: rare. Severe headache, fever, vomiting, cranial nerve palsies, convulsions, drowsiness and coma; mortality about 2%.

Diagnosis

Mumps must be differentiated from:

- Cervical lymphadenitis, in which the ear lobe is not lifted upwards and outwards (exclude TB);
- Kwashiorkor may cause swelling of parotid glands but no tenderness and fever;
- Burkitt's tumour: swelling is unilateral, firm, fixed to the jaw.

5. Management of the individual patient

No specific treatment is available, only bed rest and symptomatic treatment. Ensure proper fluid intake.

Patients with orchitis can be given analgesics, a scrotal support and bed rest. The sterility due to mumps orchitis is probably caused by prolonged oedema of the testes. The testes are surrounded by the tense fibrous tunica vaginalis which does not give way. Prolonged oedema of the testes will therefore result in diminished blood supply followed by atrophy of the testes. The tension in the testes can be relieved by surgical decompression or reduction of the oedema by use of corticosteroids.

If the testes are tense and very tender on palpation, refer to hospital for surgical decompression or steroid treatment.

6. Prevention and control

Because mumps is such a minor disease during childhood, there are undoubtedly advantages in getting over it before puberty, particularly as immunity is life-long. There is a live attenuated vaccine available and one which is combined with measles and rubella. It is not in use in Africa because there is very little priority for the prevention of mumps.

7. Action

Not relevant.

8. Summary

Mumps is a mild childhood disease. Specific treatment is not available. Mumps immunization is not a priority in Africa.

Chapter nine

Tuberculosis and Leprosy

INTRODUCTION

Tuberculosis (TB) and leprosy are dealt with together in this chapter because both diseases have much in common and both are referred to as chronic endemic communicable diseases. Thus, they differ from the other communicable diseases. The first part of this chapter deals with the common features of both diseases. The rest of the chapter covers the more specific aspects of each of them.

The organisms that cause tuberculosis and leprosy

Tuberculosis and leprosy are caused by bacteria that belong to the family of Mycobacteria. Tuberculosis is caused by *Mycobacterium tuberculosis*, and leprosy by *Mycobacterium leprae*. There are many more mycobacteria, but only a few others cause diseases in man.

M. tuberculosis and *M. leprae* can quite easily be demonstrated in the laboratory by means of a special staining procedure using the Ziehl-Neelsen (ZN) stain. These bacteria have a characteristic property of retaining the red in the ZN stain even after decolorization with acid and alcohol. The result is that the bacteria can be seen as bright red rods on a blue or green background. *M. tuberculosis* and *M. leprae* are, therefore, also called acid-fast bacilli (AFBs).

Mycobacteria are relatively slow-growing organisms, especially the leprosy mycobacterium which divides, on average, only once

every 2 weeks. This very slow growth is responsible for the very long incubation period. It takes a long time to develop the disease, and it also takes a relatively long time to be cured. These diseases are therefore characterized as chronic communicable diseases. In addition, not all the mycobacteria are equally active in the same patient. Some may be present in a so-called dormant or sleeping stage. They do not divide during such a dormant stage and also they do not metabolize. Mycobacteria in the dormant or inactive state are not susceptible to drugs, since they do not take up any substances from the surrounding fluids. However, they do not stay in this dormant state for ever. Sooner or later they become active again, and therefore susceptible to drugs.

In order to kill all the mycobacteria, treatment must, therefore, be continued over a long period of time so as to give time for all the dormant mycobacteria to become active again and to be affected by the drugs. Drugs will effectively kill all active mycobacteria fairly quickly, and the patient will experience a tremendous improvement fairly soon after starting treatment. However, in order to kill those that were dormant initially, and will become active later, the treatment must be continued. Stopping treatment too soon will increase the risk of a relapse caused by these dormant mycobacteria that become active again.

Infection and disease

Both leprosy and tuberculosis are communicable diseases, thus these infections can spread from an infectious patient to a susceptible contact. The risk of becoming infected with these organisms is determined by the number of organisms coughed (TB) or sneezed (leprosy) into the air as a fine spray of droplets (aerosol), and the time that such aerosol remains in a room where other people are. Both leprosy and tuberculosis are transmitted in this way.

Fortunately not all persons who are infected this way will develop clinical disease. A great proportion of those infected will be able to overcome the infection and will thus develop a natural immunity. However, some will not be able to do so. They will develop clinical disease, either as a direct progression from the initial infection or later on in life. Factors that determine whether someone will develop disease or overcome the infection are not all clearly identified. The most important ones are mentioned in Table 9.1.

Table 9.1

Risk factors for becoming infected with tuberculosis or leprosy

1. Staying with an infectious patient in a poorly ventilated room
2. Staying in contact with a patient who is not on treatment or has not completed his full course
3. Overcrowding, since this increases the chances of catching the infection from many other people

Risk factors for developing disease after being infected with *M. tuberculosis* or *M. leprae*

1. Poor general nutritional and health status (poverty)
2. Not having received a BCG vaccination
3. HIV infection at the same time
4. History of recent measles (especially for paediatric TB)

With the current human immunodeficiency virus (HIV) epidemic, many HIV-positive patients who had successfully overcome mycobacterial infection in the past experience a flare-up of this infection leading to clinical disease. This is particularly true for tuberculosis. HIV infection causes a breakdown in the immune defence mechanism, and thus the few mycobacteria that were kept under control by the immune system get a chance to become active again and cause disease.

General comments on treatment

As mentioned before, the treatment of these two diseases is a long-term affair. Stopping treatment too soon will increase the risk of relapse.

Since patients cannot be expected to stay under daily supervision for a very long time, you must make sure that the patient co-operates in continuing his treatment. Without the patient's co-operation the treatment with drugs is bound to be a failure. It is therefore essential that the management of both tuberculosis and leprosy patients be seen as consisting of two components:

1. Treatment by means of effective drugs;
2. Education of the patient about the disease.

Unfortunately, at times, the second component has not been given the attention it deserves. Education does not simply mean informing the patient about the details of the disease. The information must be understood, and must convince the patient that in order to be cured he/she must comply with the instructions for continuation of treatment at home. In many countries health workers often put the blame for non-compliance on the ignorance or stubborn attitude and behaviour of patients. It is important, however, to realize that most patients do want to be cured, and will comply if the health-education message is clear to them.

In many control schemes, therefore, health workers may have to sit down and review their methods of health education, and devise ways of improving the acceptance of this "health education treatment". At times it may require a novel approach to the patient: no stethoscope, no white coats, no western-medicine approach, but a more basic and understandable way of communicating. Consider the condition of the patients from their own points of view, and try to find out how they feel about having tuberculosis or leprosy. This is often a basis for further dialogue. Once the patient realizes that you, as a health worker, are sincerely concerned about him, he is bound to honour such "friendship for better health".

Treatment with drugs, without adequate patient education, is only half the job

Control of tuberculosis and leprosy

Tuberculosis and leprosy can be controlled by identifying the infectious cases (sputum-positive TB and smear-positive leprosy), and putting the patients on proper treatment so as to render them non-infectious. While BCG immunization alone may help to boost the immune status of the individual, by itself it is not an effective way of controlling these diseases.

Chemotherapy is the most effective way of reducing the infectious pool of patients in the community. Once there are fewer infectious

patients around, then others have a smaller chance of becoming infected.

Obviously this strategy is effective only if every infectious patient is identified before he has a chance to infect someone else. Early case-finding is therefore essential.

At the same time, patients who have been rendered non-infectious by means of treatment should be made to remain so for the rest of their lives. This can be achieved by what is referred to as case-holding and patient-compliance. The clinic remains in constant long-term contact with a patient during his treatment and even after.

Vigorous case-finding with poor case-holding will hardly have any effect on the risk of infection in the community since the cases found have a high chance of becoming infectious again if they default from treatment.

In order to achieve effective control, all health workers must know how to diagnose and treat tuberculosis and leprosy.

Summary of introduction

1. Tuberculosis and leprosy are chronic diseases caused by mycobacteria.
2. Tuberculosis and leprosy are curable through effective treatment.
3. Health education of patients and the relatives is an essential component of the treatment.
4. Tuberculosis and leprosy control can only be achieved through effective early case-finding and case-holding.

TUBERCULOSIS

1. Tuberculosis is a systemic mycobacterial disease generally caused by *M. tuberculosis*, but also occasionally by *M. bovis*. In most cases the lungs are involved (pulmonary tuberculosis or PTB), but other organs such as the brain, intestines and bones may also be affected.

2. Occurrence and importance

Tuberculosis is endemic in most developing countries and is very common in many African countries. Most adults will have been exposed to TB infection at some stage in their lives.

The disease is on the increase, especially in those countries with a serious HIV problem. If left untreated or treated inadequately, tuberculosis can kill or at least disable the patient for the rest of his life.

Because of its frequency and severity, tuberculosis is always an important disease — sometimes the most important public health problem.

3. Epidemiology

Tuberculosis is spread from infectious patients to healthy susceptible individuals by droplet infection. This occurs when a patient with open pulmonary TB coughs out heavily-infected sputum into the atmosphere. Many people may thus become exposed to tuberculous infection, yet only a few will progress to develop actual disease. The rest will overcome the infection by acquiring active natural immunity to the disease. However, not all mycobacteria are eliminated from the body in the process of acquiring natural immunity. Quite a few may still live on, but their numbers and activity are under tight control of the immune system so that they do not cause disease. The infected person will notice very little, if any, of this. However, when the immunity is generally depressed (e.g., in old age, during times of famine, or by HIV infection or immune-suppressing drugs such as corticosteroids) tuberculosis may flare up again and cause obvious clinical disease.

While the clinical presentation of tuberculosis may vary greatly according to the organ or system affected, almost all patients will have been initially infected by a sputum-positive patient. These patients, therefore, represent the infectious reservoir for tuberculosis in the community. The transmission and spread of tuberculosis can only be significantly reduced if this reservoir is reduced.

Since not all infected persons will progress to develop clinical disease, TB patients will most likely have been exposed to multiple risk factors (see Table 9.1, page 319).

Development of tuberculosis in a patient

A healthy individual becomes infected with *M. tuberculosis* when the organisms are breathed into the lung and settle in a distant part of lung tissue. There they multiply and cause some tissue destruction. While this happens the immune system sends many lymphocytes to the site

in an attempt to conquer the invading organisms. What happens next depends on the strength of the person's immune response. As mentioned before, most people overcome this stage without any problems. The place in the lung where the mycobacteria first settle is surrounded by lymphocytes and other immune response cells. These cytotoxic cells try to eliminate the mycobacteria by surrounding and killing them. This fight is associated with enlargement of the regional lymph nodes at the bifurcation of the trachea.

The combination of a solitary lesion in the lung with enlargement of regional lymph nodes is known as the primary complex. Most primary complexes heal spontaneously, but some mycobacteria may still remain alive in the lung tissue or in the lymph nodes in a more or less dormant state for a while afterwards. They cause no further problems because the immune system has them under constant surveillance and control.

If the immune response is weak or immunity declines, the mycobacteria may gain the upper hand and spread to other parts of the lungs. There they cause characteristic lesions. These solid lesions, containing mycobacteria and immune response cells, then gradually involve more and more lung tissue.

Before long, because of continuing tissue destruction, the middle parts of the solid lesions begin to liquefy into cheese-like material which is released into the small airways. The patient starts to cough. The cough soon begins to produce purulent sputum. This sputum contains the material from inside the lesions and can be heavily laden with mycobacteria. This is the infectious stage of the disease. The patient has now become infectious to others.

As the mycobacteria destroy lung tissue, the destruction may cause disruption of some small (or sometimes larger) blood vessels. This results in blood appearing in the sputum. This is known as haemoptysis, and it is highly suggestive of tuberculosis. Occasionally a patient may die as a result of a massive haemorrhage.

The disease can spread outside the lungs in several ways. The mycobacteria may migrate into the bloodstream and thus be carried to other parts of the body. They can also spread through the lymph vessels, causing swellings of lymph nodes, especially those of the neck (cervical lymphadenopathy). The patient may swallow some of the sputum he coughs up, and thus introduce the germs into the intestines.

Wherever the mycobacteria spread and settle, they cause similar lesions, whether it is in the meninges (lining of the brain and spinal cord), kidneys, bones and joints, the pericardium or the peritoneum. TB in these areas is referred to as extra-pulmonary tuberculosis to distinguish it from PTB. When the mycobacteria enter the bloodstream and are disseminated widely to both lungs, this is called miliary tuberculosis, a very serious form of the disease.

The infection, as it spreads, causes an inflammatory reaction in the tissues and organs it has spread to. Involvement of the pleura (covering of the lungs), pericardium (covering of the heart), and peritoneum (inside lining of the abdomen) results in TB pleuritis, pericarditis, and peritonitis, respectively. Subsequent accumulation of inflammatory fluid at these sites causes pleural and pericardial effusions, and ascites, respectively. All these are serious complications which must be dealt with in a hospital.

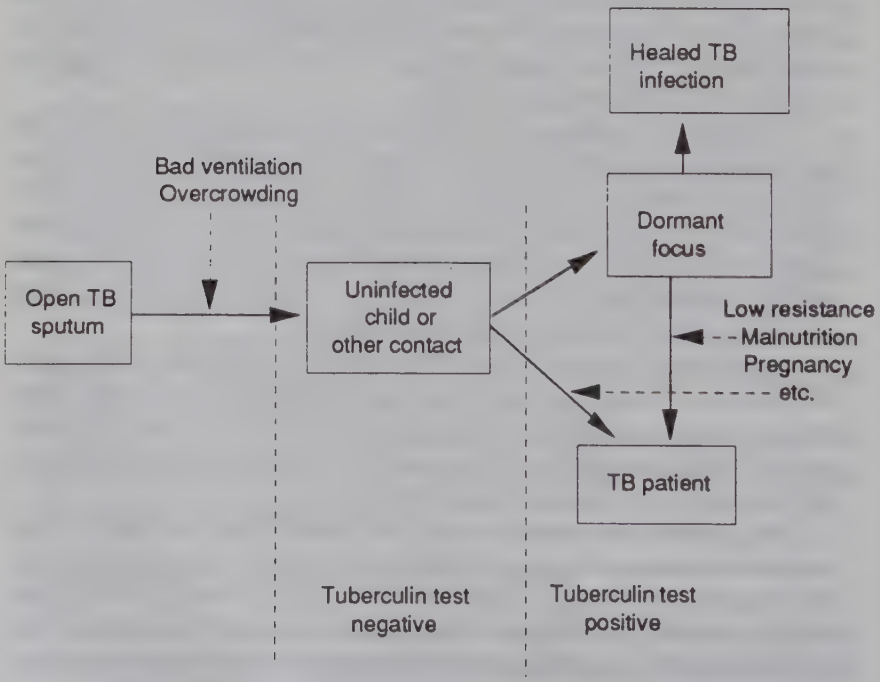


Figure 9.1 **The course of TB infection**

4. Clinical picture

The signs and symptoms of TB can be easily understood from the clinical history of the disease.

Early signs and symptoms

- Productive cough for more than a few weeks.
- Positive tuberculin test.
- Unexplained loss of weight.
- Night sweats.

Late signs and symptoms

- Blood-stained sputum.
- Enlargement of lymph nodes.
- Difficulty in breathing.
- Severe loss of weight.
- Symptoms of other organs involved.

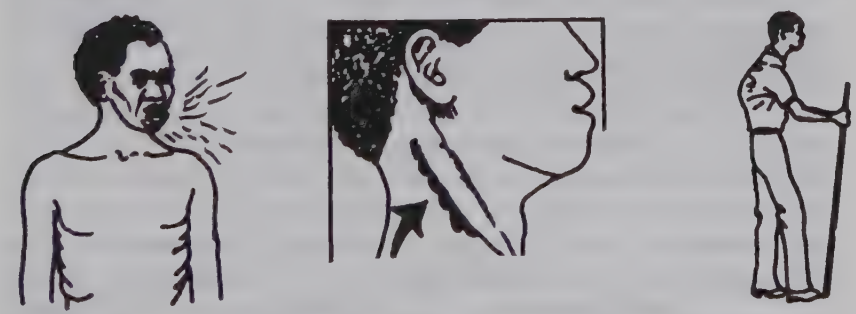


Figure 9.2 **Some clinical features of TB**

Diagnosis

One of the best ways of diagnosing tuberculosis is by means of a direct sputum smear examination in the laboratory using the Ziehl-Neelsen staining technique for acid-fast bacilli. At least three early morning specimens must be examined. It is important that real sputum is produced and not just some saliva.

There are two skin tests, the Heaf and Mantoux tests, which are also used for investigating patients for tuberculosis, but they are not

as reliable as sputum examination for AFBs. Many people, in particular children after a BCG immunization, react positively to these tests while they have no clinical disease.

The tuberculin or Mantoux test determines whether the immune system has been previously exposed to *M. tuberculosis* or not. It is really a test for infection rather than disease, but if the test is strongly positive, it may suggest active tuberculous disease. Therefore, the test must never be reported simply as positive or negative, but rather in millimetres of induration (swelling at the site of injection). An induration of more than 12 mm in diameter is very suggestive of tuberculous disease, while measurements of more than 15 mm may be considered proof.

How to do the Mantoux test

A small amount of tuberculin (10 tuberculin units in 0.1 ml of saline) is injected intradermally on the front of the forearm. A small swelling (or weal), just like an insect bite, results. This site is examined for induration 3 days later (not before!). The diameter of this induration in millimetres is recorded as the result of the test.

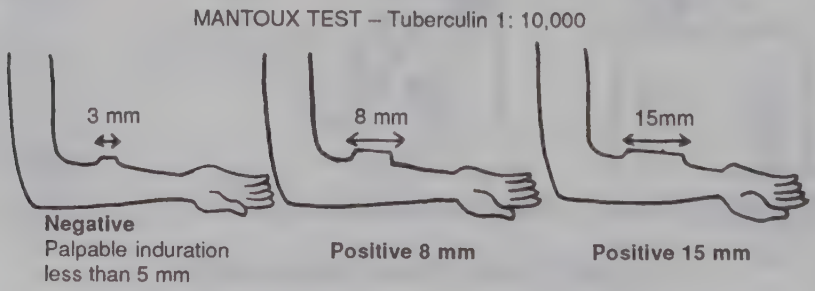


Figure 9.3 **Results of Mantoux test**

Make sure that you use potent tuberculin (look at the expiry date). Tuberculin loses its potency when exposed to sunlight, and should, therefore, be stored in a dark cool place (refrigerator), and only be taken out for a test. Do not leave the bottle standing on your desk the whole day: this will only make it useless after some time.

What to do with the Mantoux test results

An induration of over 10 mm suggests either previous exposure of the immune system to tuberculosis (BCG vaccination, or natural infection), or indicates active tuberculous disease. Active disease is associated with induration of larger diameters.

An induration below 10 mm in diameter generally excludes active tuberculosis, unless the patient's general condition is so poor that the immune system has no power left to respond. This may be seen in children with severe malnutrition, or recent measles, and in adults with very poor health or with HIV infection, AIDS, or disseminated miliary TB.

More sophisticated methods of investigation such as sputum culture for mycobacteria and X-ray examination, may be available in hospitals, but these are not always necessary for diagnosis of TB. Good history-taking, thorough physical examination and good routine laboratory investigations are often sufficient for arriving at the right diagnosis even in a health centre or a hospital without sophisticated investigative facilities.

However, the clinical presentation of tuberculosis in patients with HIV infection or AIDS can be very varied indeed. Such patients tend to have extra-pulmonary tuberculosis rather than pulmonary TB. This can be very difficult to diagnose. Moreover, the pulmonary symptoms of AIDS related complex (ARC) or frank AIDS can be easily confused with tuberculosis. While TB may well be present at the same time as HIV infection, this is not always so. Other bacteria can cause very similar symptoms in AIDS patients. Such patients should, therefore, be referred for specialist attention in a referral hospital.

5. Management of the individual patient

Treatment of tuberculosis serves two main purposes:

- To cure the individual patient;
- To prevent spread of the disease to others.

The aim of the treatment is to kill the mycobacteria as efficiently as possible, and within the shortest possible time.

There are many different treatment regimens, which are basically combinations of several anti-tuberculosis drugs. Combination treatment using two or more drugs is far superior to single-drug

treatment because the risk of the mycobacteria developing resistance to a single drug is much higher than the risk of their developing resistance to several drugs used at the same time.

Treatment regimens may vary from country to country, depending on national policy guidelines for the treatment of tuberculosis in each of them. However, tuberculosis is always treated in two phases:

- The intensive-therapy phase;
- The continuation-therapy phase.

During the intensive-therapy phase a combination of the most powerful drugs is used to kill as many of the tubercle bacilli as quickly as possible. Unfortunately these drugs are also the most expensive ones!

During this phase the patient may be treated as an inpatient, or be seen daily as an outpatient. It is very important to make sure that treatment is supervised daily. This phase usually lasts from 1 to 2 months.

After the intensive-therapy phase, the patient is discharged to continue with the rest of the treatment unsupervised at home. However, he reports to the hospital or health centre every month to collect a fresh supply of continuation-phase drugs, and also for review of his general health and response to treatment. If he defaults for any reason he should be visited at his home to find out the reason.

The most commonly used drugs are:
Streptomycin
Thiacetazone
Isoniazid
Rifampicin
Pyrazinamide
Ethambutol

Streptomycin is given by daily intramuscular injection; the others can be taken by mouth. Rifampicin and pyrazinamide are very expensive drugs, and not all countries can afford them. In many countries ethambutol is used in combination with others as a second line treatment.

Some combination tablets

Thiazina is a well known, cheap and effective combination of thiacetazone and isoniazid.

Rimactane is a combination of rifampicin and isoniazid.

Rifater is a combination of rifampicin, pyrazinamide and isoniazid.

After completing the full regimen, the patient is re-examined and then discharged from the clinic if he is fully cured. However, a small proportion of patients may suffer a relapse afterwards. All patients should be alerted to come back to the clinic as soon as possible if they detect any signs of relapse.

Since the co-operation of the patient is essential, adequate health education throughout treatment is as important as the treatment itself.

Table 9.2 **Different anti-tuberculosis drug regimens (daily administration)**

	Intensive phase		+	Continuation phase
A.	*Two months*:	Streptomycin Rifampicin Pyrazinamide Isoniazid	+	6 months thiazina
B.	*Two months*:	Streptomycin Thiazina	+	10 months thiazina
C.	*One month*:	Streptomycin Thiazina	+	18 months thiazina

Regimen A is the best, but it is also the most expensive and it is potentially toxic. In some countries, this regimen is reserved for sputum-positive cases and patients with TB meningitis or miliary TB.

It is, however, important that you stick to the national policy guidelines of your country. If you are not aware of these, consult your local or regional Tuberculosis Control Officer.

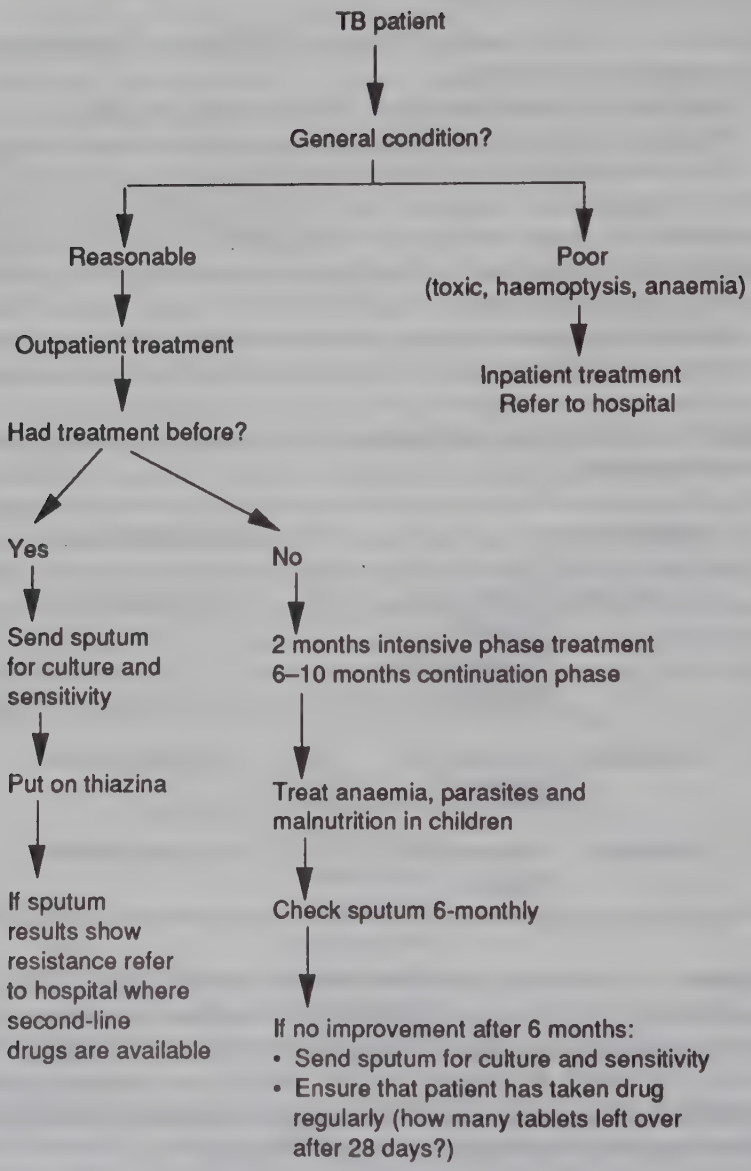


Figure 9.4 **TB treatment schedule**

Management of complications

Tuberculosis has a high case-fatality rate (12–15%), mainly because of its complications, which are often very serious. It is therefore important during the periodic reviews, to be on the look-out so as to recognize and detect these complications early since the prognosis depends on early detection and management. Serious complications are best managed in a hospital by an experienced doctor, and patients with such complications should therefore be referred as soon as possible.

The most common complications of tuberculosis are:

- Severe haemoptysis (coughing up blood);
- Respiratory failure;
- Pleural/pericardial effusion;
- Meningitis;
- Kidney failure;
- Vertebral collapse (Pott's disease).

Drug reactions

Some patients develop reactions to anti-TB drugs. Such drug reactions may range from mild to life-threatening (e.g., Stevens-Johnson syndrome). HIV-positive and AIDS patients are particularly prone to serious anti-TB drug reactions.

One of the commonest mild drug reactions is an itchy skin rash which responds well to anti-histamine drugs. In most cases it is very difficult to determine which of the drugs is responsible for the reaction. However, thiacetazone is notorious for such reactions.

Patients with severe reactions need to be referred to hospital for admission and proper management.

HIV and tuberculosis

Patients with HIV infection are very prone to tuberculosis because of the breakdown of their immune systems. The disease may be due to reactivation of primary infection, often long after the first infection took place, or it may be due to a fresh infection easily acquired from an infectious contact since the patient has lost immunity to the disease. This accounts for the increase in the number of tuberculosis cases in countries with a serious HIV problem.

Table 9.3 **Side effects of the common TB drugs**

Drug	Side effect
Streptomycin*	Allergic reaction (rash, fever) Numbness around the mouth Dizziness (mild) Hearing strange noises Very dizzy Ataxic gait Deafness
Rifampicin	Flu syndrome Liver problems Skin rash
INH	Peripheral neuritis Pellagra-like dermatitis
Thiacetazone	Nausea and vomiting Persistent vomiting HIV positive patients may have violent skin reactions: exfoliative dermatitis, Stevens-Johnson syndrome
Pyrazinamide	Anorexia, nausea, flushing Anaemia (rare) Gout
Ethambutol	Ocular toxicity

*Streptomycin must be avoided in pregnancy

The diagnosis of TB can be very difficult and tricky in HIV-positive patients because they often develop all kinds of other opportunistic lung infections which may be difficult to distinguish from tuberculosis. The clinical presentation of tuberculosis in these patients may be quite different from the classical picture. The symptoms are often very vague, and may be overshadowed by the pathology of other AIDS-related disorders.

Extra-pulmonary and miliary tuberculosis are particularly common in these patients. TB lymphadenitis, especially of the lymph nodes in the neck, is also a common feature. A thorough diagnostic work-up is required in these patients.

In some cases, patients may present with pulmonary symptoms suggestive of TB without anyone being aware that they are also suffering from AIDS. Tuberculosis may be diagnosed, while the underlying disorder is missed. It is therefore important, when examining a patient, to be on the look-out for other disease conditions which may be present in the same patient, especially where HIV infection and TB are commonly known to occur together.

On the other hand, one must be careful not to diagnose TB too readily in AIDS patients with non-specific pulmonary disease. One must make a correct diagnosis before starting anti-TB treatment since AIDS patients are prone to severe drug reactions, such as the Stevens-Johnson syndrome which carries a very high mortality rate.

TB in AIDS patients should, however, be vigorously treated, although the prognosis is not always very good. The main aims of treatment are to relieve symptoms and to render the patient non-infectious so that he does not infect others. Remember that these patients may spread two diseases: HIV and TB.

The actual treatment of TB does not differ from that for HIV-negative patients. There is as yet no justification for prolonged use of anti-tuberculosis drugs in HIV-positive patients after completion of the full course, except when there is a relapse. Likewise, chemoprophylaxis is not indicated in HIV-positive individuals who are free from tuberculosis.

Tuberculosis in children

It is very difficult to diagnose tuberculosis in children, mainly because of the difficulty of obtaining sputum specimens. Gastric lavage can occasionally be carried out to obtain specimens for ZN staining to demonstrate swallowed mycobacteria, but this is a messy procedure which does not always give reliable results. Physical and X-ray examinations may not reveal much. Therefore, it takes a TB specialist or an experienced paediatrician to make a diagnosis of tuberculosis in children. Refer all suspected cases of TB in children to hospital.

6. Prevention and control

Case-finding and treatment

The control of tuberculosis depends on the success of treatment of registered TB patients and contact-tracing. Emphasis must be placed on those who are sputum-smear-positive, to make them smear-negative.

It is important to put all patients with open tuberculosis on treatment as early as possible to prevent them from infecting others. Early case-finding is an important aspect of TB control. All health workers should know the early symptoms of tuberculosis so that they can diagnose it whenever a patient reports to a health centre with suggestive complaints (passive case-finding). In addition, the Community Health Worker must visit the homes of all new TB cases to look for other cases (contact tracing or active case-finding).

A patient who is rendered non-infectious by treatment does not spread TB any longer, and is therefore not a threat to the community. However, this can only be maintained if he completes the course of treatment without defaulting. A patient who defaults runs the risk of relapse, and may again become infectious to others. This is prevented by case-holding.

Whether a patient completes his course of treatment or not largely depends on the efficiency and reliability of the health services. It is good to advertise the routine methods of TB treatment in the health centre. It may be worthwhile selecting a special day in a month when all TB patients are seen. This will prevent long queues for the patients. It will also make work easier for the health worker because the register and the records will be close at hand in the special clinic. During these clinics the patients must also have proper health education.

Intensive health education must be given to the patient throughout the course of his treatment to minimize defaulting and to ensure case-holding. The community also requires health education on TB so that control measures can be instituted, and cases can be identified at an early stage. We emphasize, once again, that health education is more than simply giving a lecture: it should be a good dialogue between patients and health staff.

The role of BCG immunization

BCG has long been considered an important tool in the control of tuberculosis. It provides active immunity against *M. tuberculosis*, and to a lesser extent to *M. leprae*. This immunity is, however, not complete. BCG immunization does not prevent the development of tuberculosis completely, but there is evidence that infants immunized with BCG are less likely to develop the more serious forms of the disease such as TB meningitis and miliary TB than those who are not immunized.

BCG is most effective when given before any "natural" infection with mycobacteria takes place. Newborn babies should, therefore, be immunized at birth or as soon as possible after birth. A booster dose of BCG should be given at school age to ensure longer lasting immunity.

BCG is given as an intradermal injection (until a weal shows) on the left forearm near the elbow, or on the right shoulder. An ulcer develops at the injection site, and it takes about 6 weeks to heal, leaving a scar behind. This scar is future proof of a successful BCG immunization. Absence of such a scar after BCG administration means immunization failure, and thus it should be repeated.

The BCG vaccine is not very stable and it loses its potency rapidly when exposed to sunlight. It should, therefore, be stored in a refrigerator when not in use, and in an ice box when being transported for use in the clinic or field.

BCG should not be given to a baby who has AIDS because it has been reported to develop into a very serious systemic disease that looks very much like TB. There is still some controversy over whether or not BCG should be given to HIV-positive babies, but in general there is as yet no evidence of increased risk of disseminated BCG disease in such babies when they are given BCG at birth.

BCG is not very effective in the baby if the mother has open tuberculosis or is breast-feeding while on anti-tuberculosis treatment. The anti-TB drugs secreted in breast milk are ingested by the baby and they will neutralize any BCG vaccine administered. It is, therefore, useless to immunize such babies with BCG. Instead these babies should receive INH prophylaxis for as long as the mother is on treatment, and be given BCG only when the treatment is over.

Other measures

A TB patient should be advised not to spit anywhere carelessly. He should be provided with a sputum mug in which to spit. Such sputum should be disposed of carefully in a fire or a pit latrine, and the soiled mug dried in bright sunlight after disposal of it contents.

Overcrowding and poor ventilation at home should be avoided whenever possible. These measures will limit spread of the disease.

However early effective treatment will rapidly make the patient sputum-negative so then disposal of sputum is no longer an issue.



1. Cover mouth when coughing. 2. Spit into a container with cover. 3. Wash and sterilize container in the sun. 4. Eating utensils should be dried in the sun.

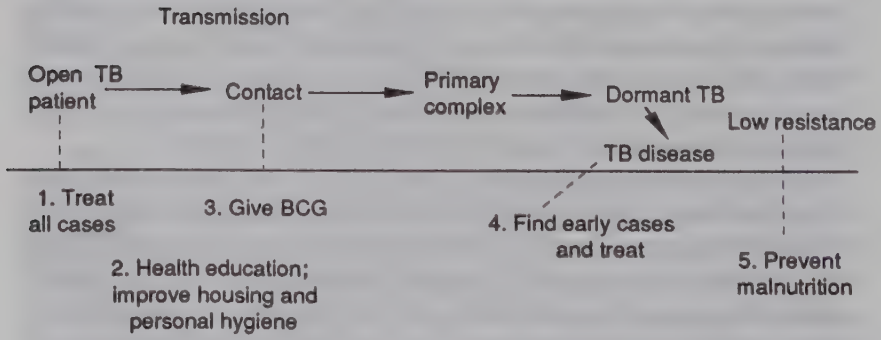


Figure 9.5 **TB control**

7. Summary

- Tuberculosis is a common infectious disease which is curable if diagnosed early and treated adequately with a full course of combination anti-TB drugs.
- Sputum-smear-positive cases represent the infectious reservoir in the community.
- HIV (AIDS) can provoke a previously dormant TB infection into clinical disease.
- Heath education for patients, relatives and the community is an essential part of treatment for successful TB control.
- BCG vaccination must be given early in life, preferably at birth, to protect against the more serious forms of tuberculosis.

LEPROSY

1. Leprosy is a chronic infectious disease of man caused by the leprosy bacillus, *Mycobacterium leprae*, and the tissue reaction against it. It mainly affects the peripheral nerves and skin, but also the mucous membranes of the upper respiratory tract, and some internal organs such as testes, kidney, and liver.

Synonym: Hansen's disease.

2. Occurrence and importance

Leprosy is not a fatal disease: it is a crippling disease. The life expectancy of an individual with leprosy is not much less than the life expectancy of a healthy person, but leprosy is very disabling and deforming and therefore frightening.

Deformities occur in the hands, feet, and face. Eye involvement may causes blindness. Testicular atrophy may result in impotence and sterility. Liver involvement may manifest as gynaecomastia. These deformities are almost inevitable if leprosy is not treated early or left untreated. They will permanently mark the patient as suffering from leprosy and prevent a normal life by making it difficult for the sufferer to work or cultivate his fields. Often the patient suffering from leprosy finds that other members of the community avoid him. This is because of traditional beliefs about its cause and even incorrect modern ideas about its contagiousness. No other disease, except AIDS, causes such

a reaction in the community and so much distress and unhappiness to the patient and his family.

This stigma may follow leprosy patients throughout their lives and bring a permanent shadow over their families and their professional and social activities. Leprosy patients may lose their jobs without proper justification, simply because of the disease. They are often divorced or the community may prevent them from marrying. They frequently have to leave their villages either because they are known to have leprosy or because they fear that their disease will become known when people see them going for treatment. Some patients are even deserted by their families.

According to the WHO definition, leprosy is hyperendemic in an area when the prevalence is over 10 per thousand. About one-third of the leprosy cases in the world occur in Africa.

Leprosy is an important disease from the public health point of view because of its (a) high prevalence, (b) severe disability and deformities and chronic course, and (c) social aspects and stigma.

A lot of research has been done to determine the risk factors for getting leprosy. Below are some of the risk factors involved:

Race: no known racial immunity; all races are susceptible.

Sex: infectious forms are more frequent in males.

Age: no age group is more susceptible than another. Most new cases are diagnosed between 15 and 25 years of age. This is not due to susceptibility but to opportunities for contact and length of the incubation period.

Heredity: the incidence of leprosy among children of lepromatous parents is 10 times higher than that found in the population at large. It is not fully understood whether this is due to severe exposure or to hereditary (genetic) factors. Leprosy should not, however, be regarded as a hereditary disease.

AIDS: there is evidence that patients with HIV infection or AIDS are at a higher risk of catching leprosy.

3. Epidemiology

Leprosy is caused by *Mycobacterium leprae*, which resembles *Mycobacterium tuberculosis* in staining properties. Not all patients suffering from leprosy are equally infectious to others. Those in whom the skin smears are negative are not regarded as 'open'

(infectious) cases and will not spread the disease. Patients with positive skin smears may infect others if they are not under regular treatment. When treatment is started and continued regularly more of the bacilli will stain irregularly and take on abnormal forms. This shows that the bacilli have died and the patient is no longer infectious.

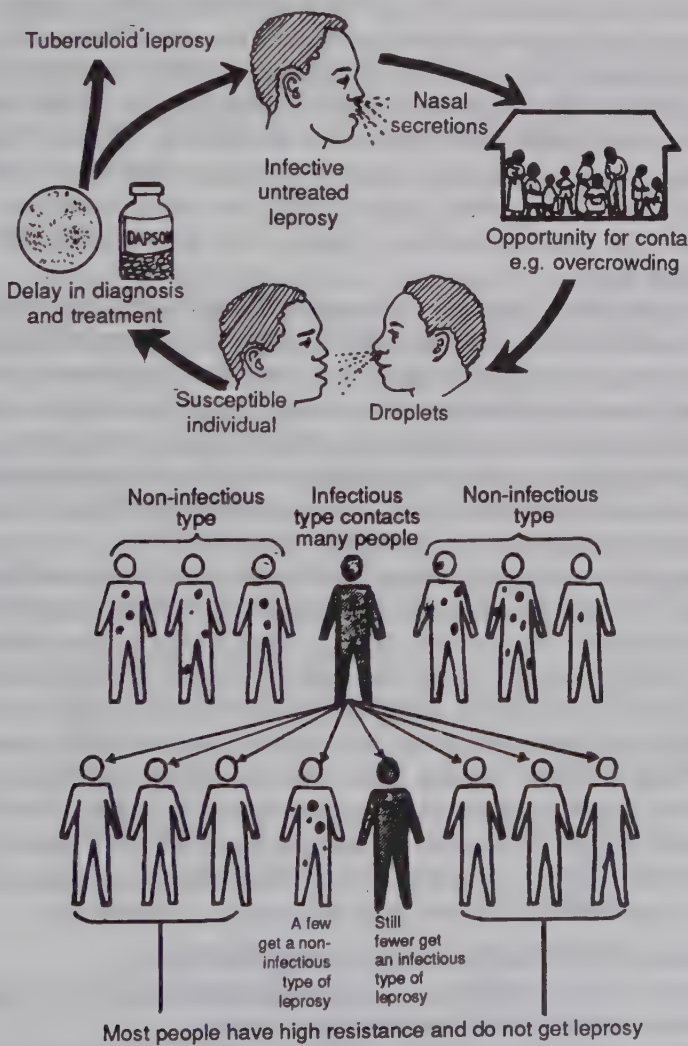


Figure 9.6 Transmission of leprosy

Epidemics occur when leprosy is introduced in a community for the first time. When the epidemic has passed the disease settles down into an endemic state. In most of Africa leprosy occurs in the endemic form.

There are, broadly speaking, two forms of leprosy: the tuberculoid form and the lepromatous form. There are many more mycobacteria in lepromatous leprosy than in tuberculoid leprosy. The most heavily infected sites in patients with lepromatous leprosy are the skin, the nose, and the upper respiratory tract. Mycobacteria are detectable in the nose even before skin lesions are evident. At this site they are found very near to the surface, while in the skin lesions there is usually a clear zone at the surface. Large numbers of viable bacteria can be found in the nasal secretions: a patient may discharge up to 1,000 million a day.

It is very likely that leprosy is transmitted by sneezing, coughing, spitting and unhygienic nose-cleaning habits. This is probably by way of droplets, as in tuberculosis. It is very unlikely that leprosy bacilli are able to penetrate the intact skin. It may be that they are able to enter through small wounds but this is probably not the usual route. It is, therefore, still uncertain whether the portal of entry for leprosy bacilli is the skin or the respiratory tract. In any case, leprosy is most common among household contacts (as is the case with TB).

Only patients who discharge live leprosy bacilli can infect other people. Most people who come into contact with leprosy bacilli do not develop leprosy because they already have a high natural resistance to *M. leprae* and their bodies are able to kill any live mycobacteria which might enter them. Only a small number of people who lack adequate resistance get leprosy from an infectious patient. Fortunately, most such patients develop a non-infectious type of leprosy. This shows that leprosy is not easily caught by other people. This fact is also demonstrated by the incidence of leprosy among leprosy health workers which is not any higher than that in the general population.

The incidence of leprosy may increase in a community:

- When there are many infectious untreated cases;
- When social conditions provide an opportunity for contact: overcrowding, social customs, poor houses;
- When there are many susceptible newcomers in a given area, or when leprosy is introduced into a previously leprosy-free area;

- When patients tend to hide their disease; this will delay early diagnosis and treatment thus giving a chance for the disease to spread in the community.

4. Clinical picture

Incubation period

M. leprae bacilli have a long multiplication time. They divide once every 2 weeks. This accounts for the very long incubation period and the slow course of the disease. The mean incubation period is 6 years (range 1–12 years).

Pathology

The lepra bacillus multiplies in macrophages of the skin and nerve fibres (Schwann cells). The organisms multiply best in the cooler parts of the body so that the skin of the face and limbs and their more superficial nerves are invaded first. There is an inflammatory reaction as a result of the mycobacterial invasion of these structures. Clinically a small vague hypopigmented macule appears. The lesion is called indeterminate* as there is no indication as to how it will develop. Most indeterminate lesions heal spontaneously; many are never noticed. Lesions which do not heal may develop into tuberculoid, borderline, or lepromatous leprosy. This depends on the ability of the host to develop cellular immunity against *M. leprae.*

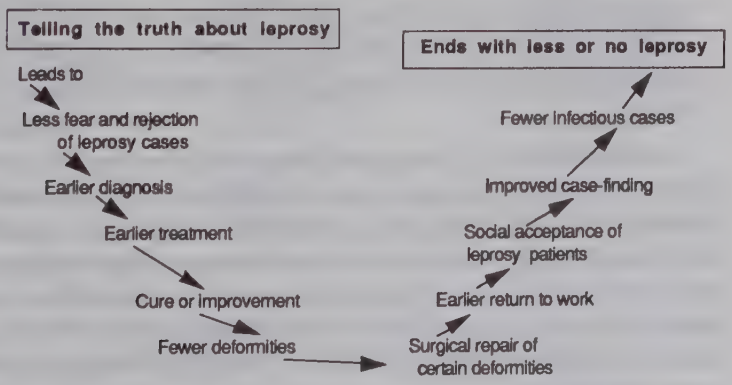


Figure 9.7 **Being truthful about leprosy helps**

* Do not confuse "indeterminate" with "intermediate".
Indeterminate: it cannot be determined or defined.
Intermediate: in between.

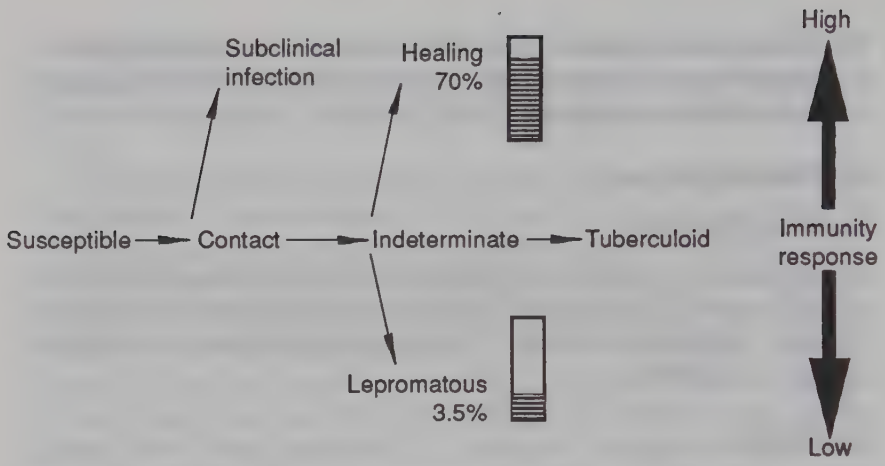


Figure 9.8 **The development of different forms of leprosy is dependent on the host's cellular immunity**

Depending on the degree of cellular response by the host, the following can occur:

- In tuberculoid leprosy there is a strong focal reaction to the bacteria. The intracutaneous nerves are infiltrated by lymphocytes and completely destroyed, resulting in anaesthesia. Acid-fast bacilli are usually few or absent. Invasion of peripheral nerve trunks is early.
- In lepromatous leprosy there is little response; very few lymphocytes are found, nerve twigs are not infiltrated by lymphocytes and masses of bacilli can be seen. Damage to peripheral nerve trunks is late.

The body reaction, however, is not always as clear cut as in these tuberculoid or lepromatous types. Borderline leprosy shows features of both the tuberculoid type and the lepromatous type. Lymphocytes are present, but there is only slight infiltration of the fine nerve twigs; acid-fast bacilli occur in moderate numbers. Borderline leprosy is unstable and can change into one of the definite forms (but this is very unusual). Because of the instability, widespread damage to peripheral nerves may occur.

Leprosy is divided into four clinical forms as shown on the following page:

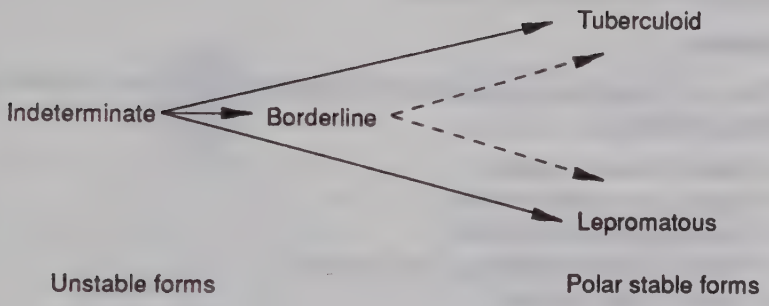


Figure 9.9 **Clinical types of leprosy**

The clinical characteristics of the different types of leprosy are an expression of the cellular immunity or natural resistance of the patient.

Table 9.4 Clinical characteristics of the different types of leprosy

	Signs of low resistance	Signs of high resistance
Number of lesions	Many	Few
Size of lesions	Small	Large
Distribution	Symmetrical	Asymmetrical
Definition of lesions	Ill-defined	Clearly defined
Hypopigmentation	Slight or none	Marked
Central healing	None	Marked
Progression	Progressive	Self-healing
Loss of sensation	None	Clear
Skin smear	Positive	Negative

TUBERCULOID LEPROSY

Lesions

The skin patches in tuberculoid leprosy have the following characteristics:

- Usually few in number;
- Patches are raised or flat;
- The edge is well defined;
- There is loss of sweating (dry surface);
- There is loss of hairs;.
- There is loss of sensation;
- Often there is central healing;
- Hypopigmentation is marked;
- The patches are large and asymmetrically distributed.

Nerves

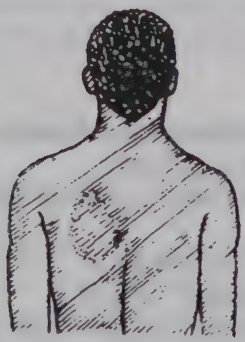
The cutaneous nerves may be enlarged in areas where there are patches. The main nerve trunks become enlarged over a part of their lengths. Nerves are always involved early in the course of tuberculoid leprosy. Paralysis is common.

Smear

Skin smear is usually negative.

Tuberculoid leprosy is sometimes divided into two sub-groups: TT and T. Patients with high resistance only develop a few (less than five) self-healing patches. If resistance is lower there will be more patches, with a tendency to a more symmetrical distribution, especially on the knees and elbows. The patches may show the typical features of tuberculoid leprosy:

- Small patches near larger ones (satellites);
- Spreading of the edge of a patch in an irregular way, like oil spreading on the skin or water running out of a puddle (streaming);
- Two or more patches joining together to form one major patch (confluence).



Skin lesions: Usually few
Raised or flat, large or small
Well-defined edges, regular or irregular
Surface dry
Always some loss of feeling, sometimes quite marked
Often central healing
Some colour change in patch, red or pale
Asymmetrical

Nerve trunks: Sometimes one or two trunks involved early

Smears: Negative

Figure 9.10 **Tuberculoid leprosy**

LEPROMATOUS LEPROSY

Lesions

Early stage: numerous, small, ill-defined, slightly raised or flat patches, macules, or nodules. When the whole skin is involved little abnormality can be detected except that the skin, on palpation, feels thicker than normal. This is most prominent on the face and ears. There is no loss of hair, no loss of sweating, and no loss of sensation.

Nerves

The nerves are involved late. Long segments of nerve trunks are symmetrically enlarged. Anaesthesia of glove-and-stocking type occurs, and leads more commonly to disability than paralysis.

Smear

Positive skin smear.

Other organs involved

Nose: Saddle-nose deformity, because of destruction of nasal cartilage; ulceration of nasal mucosa; blocked nose; epistaxis.
Eye: Iritis and damage to ciliary body; hazy cornea; cataract; glaucoma.
Larynx: Hoarseness of the voice.
Testis: Atrophy resulting in impotence, and sterility.
Bones: Leprous osteitis resulting in "banana" fingers.
Teeth: Loss of upper central incisors due to damage to the maxilla.

Skin lesions: Many lesions, e.g.,
- nodules
- raised patches
- flat red patches
- areas of thick skin



Other organs involved: Eyes
Hands and feet
Testicles



Nerve trunks: Many involved but late in the disease

Smears: Strongly positive

Figure 9.11 **Lepromatous leprosy**

Lepromatous leprosy is sometimes divided into two sub-groups: L and LL.

If resistance is almost completely absent the onset of lepromatous leprosy is insidious with vague slightly erythematous macules which are very difficult to see. Loss of eyebrows (madurosis) may be the only clear sign. When resistance is higher, the lesions are more obvious because they are usually slightly elevated in their centres.

The lesions are shiny and smooth with vague edges. There is no loss of sensation and because of spread through the bloodstream the lesions are distributed symmetrically over the body.

BORDERLINE LEPROSY

Borderline leprosy has features of both tuberculoid and lepromatous leprosy. It is an unstable form and may change from having more tuberculoid to more lepromatous features and vice versa. Borderline leprosy is divided into three sub-groups: BT or borderline tuberculoid; BB or typical borderline; and BL or borderline lepromatous.

In most cases either the tuberculoid or the lepromatous element predominates and BB is seldom diagnosed by expert leprologists. There should, however, be little worry about distinguishing these varieties and all should be considered "borderline".

Lesions

The lesions are not so clearly defined. There may be only a slight loss of sensation. The borders of the lesions are not raised very much and



Skin lesions: Very many
Edges may be well-defined
Outline often irregular
Raised or flat
Some loss of feeling
No healing centre
Symmetrical

Nerve trunks: Often many
involved fairly early

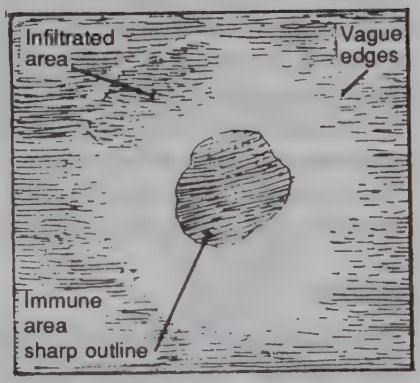
Smears: Range from negative
to positive

Figure 9.12 **Borderline leprosy**

the lesions tend to be symmetrically distributed, but not as much as in lepromatous leprosy. The centre of the lesions is often more raised than the edges (especially in BB and BL).

Nerves

The nerves are very often affected at the beginning as well as at the end of the disease. Nerve involvement is more generalized than in T leprosy.



Smear

Often positive in BB and BL. The number of bacilli found is not as high as in L leprosy.

Other organs

These are not usually involved in uncomplicated cases. Other typical signs of borderline leprosy are:

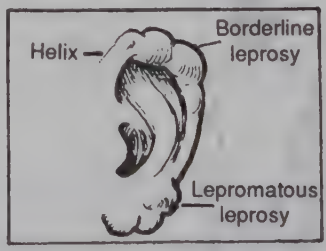


Figure 9.13 **Lesions of borderline and lepromatous leprosy**

- A patient may show clear patches (as in T leprosy) together with nodules (as in L leprosy);
- Lesions may show immune areas. These look like areas of central healing but are in fact normal skin in the centre of an infiltrating patch separated from it by a sharp boundary. They are a characteristic feature of borderline leprosy;
- Lepromatous nodules occur on the ear lobes. The nodules in borderline leprosy occur on the upper part (the helix) of the ear.

INDETERMINATE LEPROSY

The onset of indeterminate leprosy is frequently visible as an ill-defined flat patch or macule, with a slight loss of pigment. There is no loss of sensation and no bacilli are found in smears. Organs and nerves are not involved.



Duration of onset: Short, 3–6 months maximum

Skin lesions: One patch, 2–3 at most
Edges not well defined
Surface flat
Loss of feeling slight

Nerve trunks: Not involved

Smears: Negative

Figure 9.14 **Indeterminate leprosy**

Reactions

Everything in leprosy tends to be slow and chronic, but sudden changes do occur in all types of patients. Such a sudden change is called a reaction. Reactions are the result of a change in the balance between the immunity of a patient and the bacilli. There are two main types of reaction.

Type I reaction or reversal reaction

This is a change in cellular immunity. Usually the immunity increases suddenly resulting in a rapidly increased response of the body to the leprosy bacilli. This "fight" causes sudden inflammation in places where the leprosy bacilli are present. Type I reactions are especially common in the unstable forms of leprosy, that is, in borderline leprosy, and are mostly seen during the first year of treatment. (Often

they are the reason the patient comes to hospital, where he is diagnosed for the first time.)

Type II reaction or erythema nodosum leprosum (ENL)

This is caused by a reaction between dead leprosy bacilli and circulating antibodies. In L leprosy the cellular immune response is low but there are many circulating antibodies. These are ineffective against live mycobacteria, and do not help the patient very much. Type II reactions occur especially in BL and L leprosy and are mostly seen 6 months or more after the start of treatment. They may continue for years.

Reactions are provoked by:

- Other diseases, e.g., malnutrition, malaria, worm diseases, anaemia and, especially, TB;
- Extra stress or strains, physical or emotional, e.g., hard work, worries, cold weather, etc.;
- Hormonal changes, menstruation, pregnancy, childbirth, abortion, puberty;
- Other drugs, iodine (orally), traditional medicines;
- Immunizations, BCG;
- Infected wounds, osteomyelitis.

Formerly it was believed that treatment could provoke a reaction. To avoid reactions, the treatment was started with low doses and increased slowly. Leprosy specialists then found out that reactions do not occur because of the treatment. We now start treatment right away with the full dose.

Reactions are characterized by local inflammation with all its typical signs:

- Redness;
- Hotness;
- Swelling;
- Pain;
- Loss of function.

In Type I reaction the inflammation is seen in the skin lesions and nerves.

Table 9.5 **Summary of reaction types**

Type	Classification	Nerve signs	Skin signs	Management
Type I or Reversal	in BT BB BL	Nerve damage common and obvious	Existing lesions become red and raised	Refer to hospital
Type II or ENL	in L and BL	Nerve damage not common Eyes, joints, testes may be involved	Fresh red lumps appear	Treat mild cases Refer severe and non-responding cases

Lesions

The lesions become warm, tender, reddish, and raised.

Nerves

The nerves become tender and thickened. Acute paralysis is common. Pain is severe and widespread. There may be generalized oedema, especially of the hands, feet, and face. Oedema readily causes nerve damage.

Type I reactions are important because the acute paralysis is a major cause of permanent disability and deformity. The reactions may be extensive, involving many patches and nerves.

In Type II reactions (L and B), the inflammation is seen in the form of painful red nodules of recent onset. These nodules are called erythema nodosum leprosum (ENL). ENL may be mild or severe.

Mild ENL: Few lumps, mainly on face, outer sides of arms, and front of legs; patient does not feel very ill.

Severe ENL: Many lumps which may be spread all over the body; patient feels ill, and has fever and pain everywhere. Complications are common. Look for:

- Lymphadenitis;
- Iritis;
- Arthritis;
- Epistaxis or blocked nose;
- Hoarseness of the voice, sometimes stridor;

- Periosteitis (hands, feet, or long bones);
- Nephritis (proteinuria);
- Orchitis;
- Ulcerating nodules.

Relapse or worsening of leprosy must be differentiated from reactions. The main difference is the speed and severity of the events occurring and usually differentiation is not difficult (see Table 9.6).

Nerve damage and disability

The primary cause of disability is the destruction of nerves.

- Damage to sensory nerves —> anaesthesia.
- Damage to motor nerves —> paralysis.
- Damage to autonomic nerves —> impaired circulation, skin atrophy, loss of sweating.

(a) Anaesthesia

Minor injuries are not noticed as a result of the anaesthesia. Infection of the injuries causes wounds of which the patient may not be aware. These may fill with dirt leading to the spread of the infection into deeper tissues. Only advanced infection will alarm the patient (fever, a painful lymphadenitis or a grossly swollen foot or hand).

Pressure is not noticed. Normally a person will shift his weight from one foot to the other or from toes to heel. When a foot or a hand is anaesthetic, prolonged pressure in one area is not noticed. This results in ischaemia (too little blood flow) of the tissues, and then injury. This particularly happens in those areas where skin directly overlies bone.

A normal person feels the stress when a joint is being strained. His leg gives way and he falls down, or he strains other muscles supporting the joint. An anaesthetic joint will not feel this stress and a patient may put all his weight on a sprained ankle and tear ligaments without realizing there is an injury.

Anaesthesia delays recognition of infection just as it delays recognition of injury. As a result infection is only recognized when already advanced (e.g., osteomyelitis). When the bones are inflamed, osteoporosis occurs and there is increased risk of fractures and further deformity. In chronic osteomyelitis pieces of dead bone are expelled

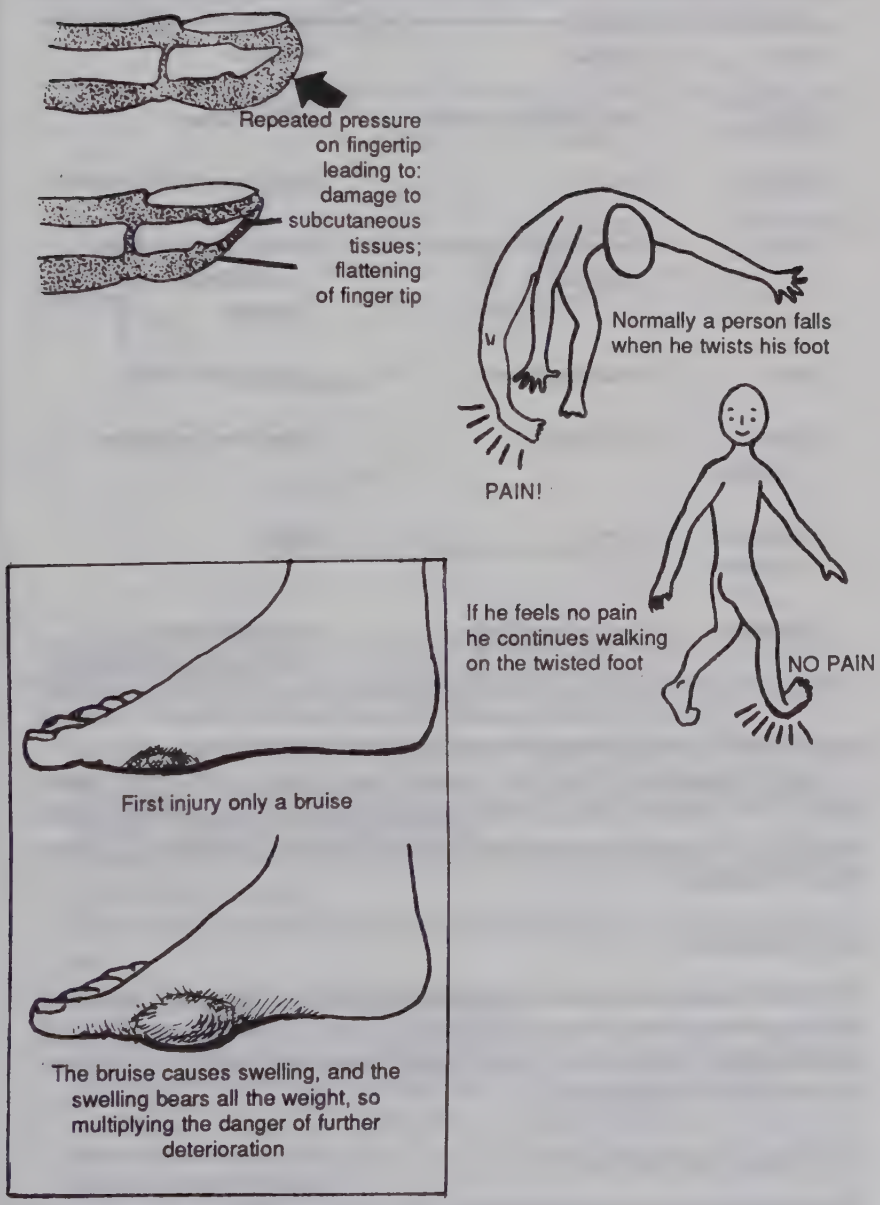


Figure 9.15 **Mechanisms of damage due to anaesthesia**

Table 9.6 Differences between worsening or relapse, and reaction

Signs	Relapse	Reaction
Onset	Slow, weeks or months	Sudden, hours
Damage	Slow	Rapid, especially in nerves
Sites	New patches anywhere on body	Usually only in patches or infiltration already present
Pain	Usually painless	Nerves often painful
General condition	Not affected	Often fever, joint pains
Response to treatment	Slow	Rapid
Treatment	Anti-leprosy drugs	Anti-inflammatory, e.g., rest, aspirin, chloroquine

by the body causing shortening of the fingers and toes.

Scar formation: When the wounds and injuries heal they leave scars. Scar tissue is weaker than normal tissue and has poorer blood supply. It is therefore more liable to new injuries resulting in more scar formation.

(b) Paralysis

Paralysis causes muscular imbalance and leads to abnormal positions of fingers and toes. These deformities result in abnormal pressure points which increase the risk of injury. Paralysis therefore worsens the effects of anaesthesia.

(c) Autonomic nerves

The autonomic nerves control blood flow in small blood vessels and the activity of sweat glands. Damage results in impaired circulation

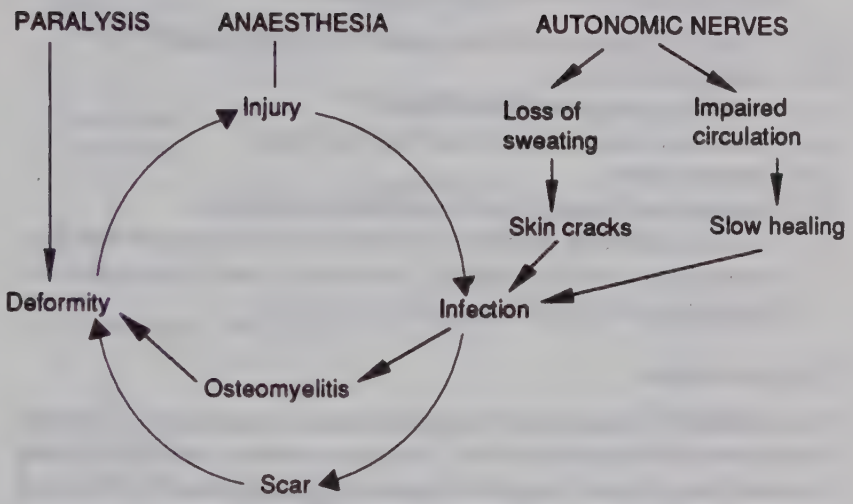


Figure 9.16 The cycle of injury

and absence of sweating. As a result, wounds heal slowly, and the loss of sweating results in a dry skin which cracks easily. The cracks may become infected and increase the damage already done.

Leprosy patients are likely to get three types of ulcers on their feet:

- Those due to injuries and burns, usually unnoticed until secondarily infected (because of anaesthesia);
- Cracks of the heel and/or lateral border of the foot due to absence of normal sweating;
- The specific plantar ulcer which is due to walking on an anaesthetic foot.

There are two types of plantar ulcers:

- *Primary:* occurring at certain elective sites, over bony points exposed to maximum pressure and friction during walking;
- *Secondary:* occurring over bony points exposed to maximum pressure and friction after the feet have become deformed by previous osteomyelitis.

In the primary ulcer the order of events is:

(a) An area of tender swelling, boggy but not fluctuant;
(b) A necrotic blister;
(c) An ulcer going down to the bone;
(d) Secondary infection not involving the bone;
(e) Osteomyelitis;
(f) Sequestrum formation with persistent ulceration;
(g) Further ulceration elsewhere on the plantar surface of the foot, consequent upon deformity following bone absorption or destruction (followed by secondary ulcers).

Eye problems

Eye conditions are important in leprosy because many leprosy patients have insensitive hands and feet. They are unable to use their feeling for examining objects or for walking and they need their eyes to avoid injuries. Impairment of vision means an extra, severe, handicap. There are three main ways in which vision can be impaired:

- Anaesthesia of the cornea means that foreign bodies entering the eye may not be noticed, leading to corneal ulcers;
- Paralysis of the eyelids may result in loss of blink reflex or in lagophthalmos (inability to close the eye), dryness of the cornea, and corneal ulcers;
- Leprosy reactions resulting in inflammation of the iris (iritis), adhesions, glaucoma, and blindness.

Diagnosis of leprosy

The diagnosis of leprosy depends on the demonstration of one or more of the following cardinal signs:

(a) Hypopigmented skin patches with loss of sensation;
(b) Abnormally large peripheral nerves at predilection sites;
(c) Acid-fast bacilli in skin smear.

(a) Loss of sensation

Loss of sensation is tested with cotton wool, rolled at one end to a thin point. Explain to the patient what you are going to do. Ask the patient to undress. Have a trial on normal skin to show the patient the

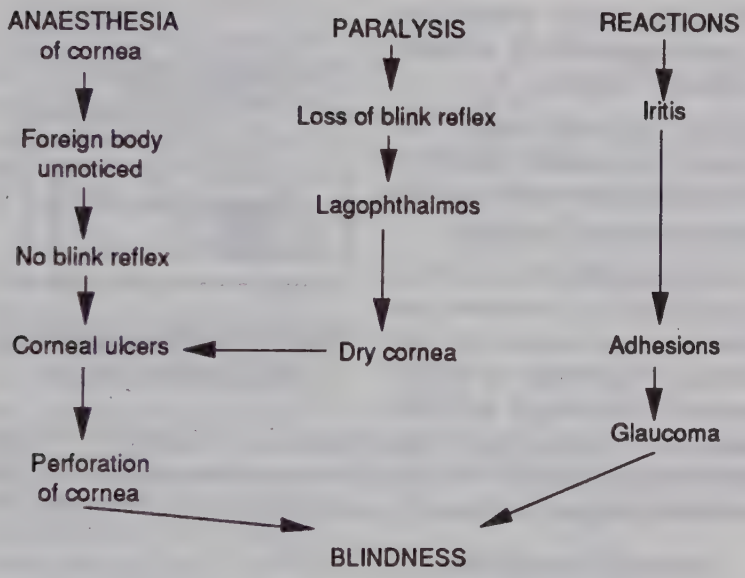


Figure 9.17 **The pathways to blindness**

procedure. Ask the patient to point to the place where you touch him. Start the examination on his back, touching normal skin, and try only to touch the patches when you are satisfied that he understands the procedure. Cover his eyes when you touch parts of the body which he can see, but reserve this till the end of the examination when he has got used to it. If a patient points more than 2 cm away from the spot you touched, we call this mislocation. Mislocation is the earliest sign of loss of sensation.

(b) Enlargement of the peripheral nerves

Involvement of the peripheral nerves is most easily diagnosed at the so-called sites of predilection. There, the nerves are lying superficially and enlarged nerves are most easily palpable. Always compare left and right sides of the body before you decide that a nerve is enlarged.

Facial nerve: Predilection site over jaw joint. If affected: lagophthalmos and/or dropped mouth. If severely damaged: palsy of all muscles of facial expression.

Trigeminal nerve: Predilection site over eyebrows. If affected: anaesthesia of cornea.

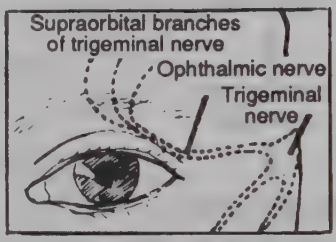


Figure 9.18 **Enlargement of the trigeminal nerve**

Greater auricular nerve: Predilection site posterior border of the sternomastoid muscle halfway between origin and insertion.

Ulnar nerve: Predilection site is the medial side of the elbow just behind the epicondyle of the humerus. If affected, it causes weakness of the fourth and fifth fingers, and it makes it impossible to achieve the lumbrical position of these fingers. There is wasting of the hypothenar muscles and the ulnar side of the hand becomes dry and insensitive (see Figure 9.19).

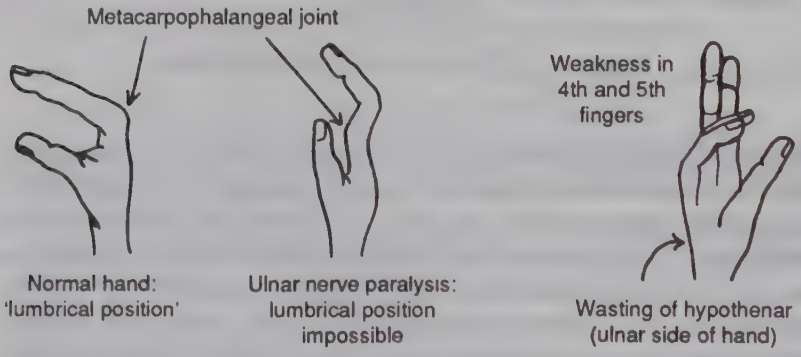


Figure 9.19 **Ulnar nerve damage**

Median nerve: Predilection site is at the middle of the inner side of the wrist. If affected, a claw hand deformity results (always in combination with ulnar palsy). There is dryness, and insensitivity of the inner hand.

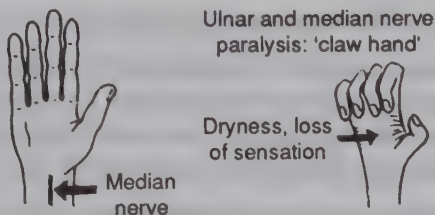


Figure 9.20 **Median nerve damage**

Radial nerve: Predilection sites are the radial side of the wrist and the upper arm underneath the biceps. If affected, wrist drop results, and there is loss of sensation in the radial part of hand.

Peroneal nerve: Predilection site is the outer side of the knee joint just behind the neck of the fibula. If affected, drop foot develops, resulting in what is referred to as cock's gait.

Posterior tibial nerve: Predilection site is behind the medial malleolus of the ankle joint. If affected, it causes clawing of the toes and anaesthesia of the foot.

(c) Skin smear

Bacilli are most likely to be found at the edge of an active lesion (active means the lesion is spreading, raised, or red). In tuberculoid patients the chances of finding bacilli are small. (For details of the technique see M. King, *A Medical Laboratory for Developing Countries*, Chapter 11, Figure 11–4; Oxford University Press, 1975.)



Figure 9.21 **Radial nerve damage**

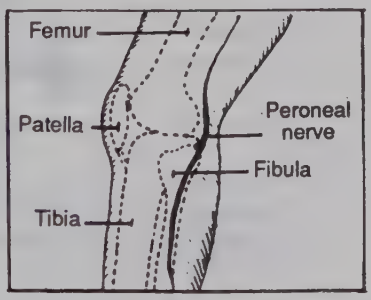


Figure 9.22 **Side view of the knee area showing peroneal nerve**

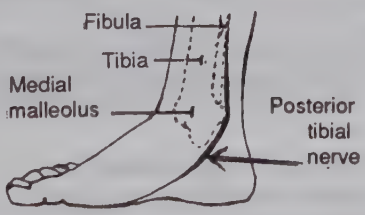


Figure 9.23 **The posterior tibial nerve**

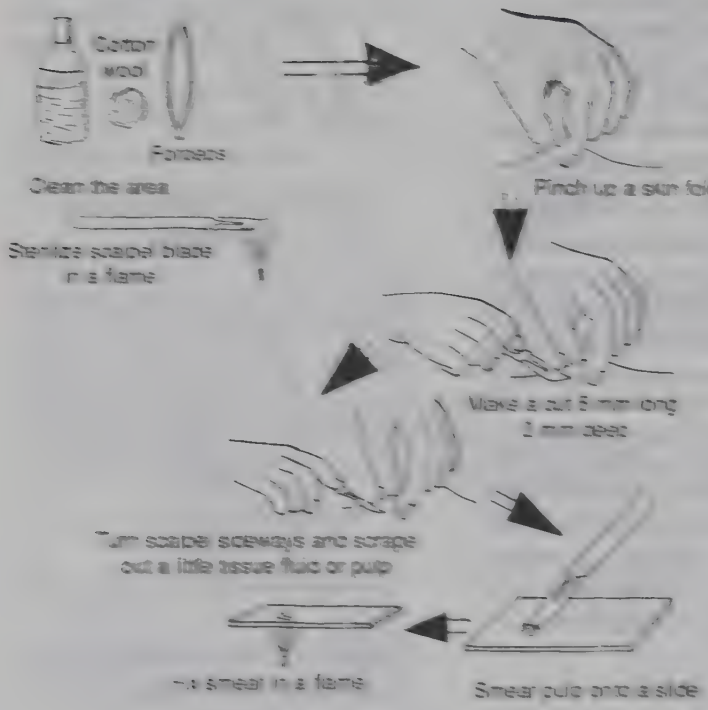


Figure 9.24 Preparation of a skin smear

In advanced cases diagnosis is easily made if you find loss of sensation and enlarged nerves. Differential diagnosis may be difficult in:

- Indeterminate macules;
- Early tuberculoid macules;
- Early diffuse lepromatous leprosy.

Never make the diagnosis by exclusion or by therapeutic trial. If the diagnosis is in doubt, refer the patient for a skin biopsy. In early lesions without nerve enlargement it is safe to wait some months and see the patient again. Never start treating a patient for leprosy before making a definite diagnosis.

Classification

In the past, an accurate but complicated classification system describing the three main forms of leprosy (tuberculoid, borderline and lepromatous) and sub-groups was used. For those experienced in leprosy, this is still a very good way of classifying patients, since each type of leprosy has its peculiar signs and symptoms and complications.

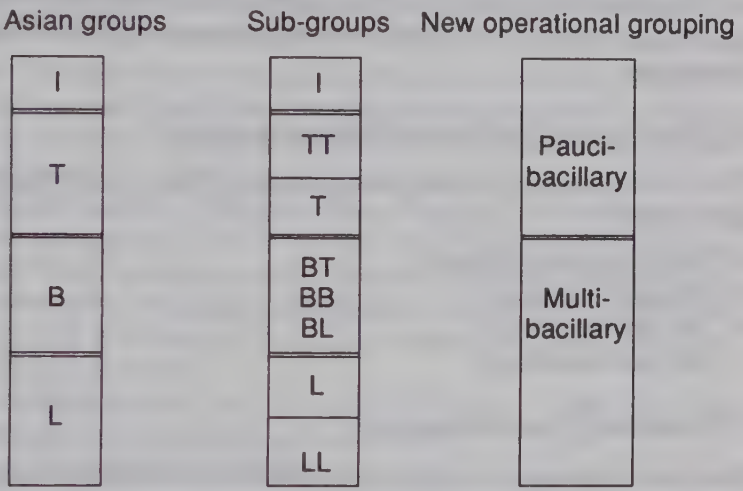


Figure 9.25 **Methods of classifying leprosy**

For operational convenience, however, a simpler classification system was devised. This is for the use of those who are not very familiar with leprosy. It is based on the number of bacilli in the lesions as determined by skin smears, and the two kinds distinguished in this way are:

- Pauci-bacillary (PB) leprosy;
- Multi-bacillary (MB) leprosy.

Pauci-bacillary leprosy is the type of leprosy with scanty bacilli ("pauci" means few). This includes indeterminate and tuberculoid (TT) types of leprosy.

Multi-bacillary leprosy is the type of leprosy with many bacilli ("multi" means many). This includes the borderline (BT, BB, BL) and lepromatous (L, LL) types of leprosy.

Patients with multi-bacillary leprosy are those clinically classified as having BT, BB, BL, L, and LL leprosy. In order to make a correct classification, a skin smear must be taken before starting multi-drug therapy (MDT). All smear-positive cases are therefore classified as multi-bacillary. Even borderline (BT, BB or rarely BL) cases without a positive smear are considered multi-bacillary. All other patients are considered pauci-bacillary.

The practical importance of this classification is that multi-bacillary patients are infectious to others, while pauci-bacillary patients are not infectious.

5. Management of the individual patient

The major priority in the management of the leprosy patient is prevention:

- Prevent nerve damage;
- Prevent deformity;
- Prevent blindness;
- Prevent defaulting.

Prevention of nerve damage

Most nerve damage is caused by reactions. Anaesthesia and paralysis should not be considered as inevitable consequences of leprosy. Early diagnosis and treatment plus adequate management of reactions should prevent all disabilities. Once a leprosy patient is diagnosed, the possibility of reactions should be explained to him. Reactions are a major cause of preventable damage to nerves, and they often make the patient think that the treatment is making him worse. As a result he may not report to the clinic with the reactional condition. He may instead discontinue his anti-leprosy treatment. The patient must be educated about reactions and advised to report immediately he notices symptoms so that he can receive adequate treatment. This will prevent complications.

Prevention of deformity

All deformity in leprosy is either preventable or correctable. Early treatment is the essence of prevention, but even patients with established nerve damage can be helped. It is essential to break through the cycle of injury-infection-deformity by:

- Proper care of the skin;
- Proper care of minor wounds and skin infections;
- Provision of shoes;
- Physiotherapy;
- Health education.

Skin care: If the skin is dry (loss of sweat), teach general skin care. A dry skin is likely to crack and to allow easy entry of bacteria. This is most likely to occur on the soles of the feet.

- Soak the affected parts in cool or warm soapy water. Avoid hot water because it may burn the skin without the patient realizing it (as a result of anaesthesia). Soak until tissue is soft (10–15 minutes).
- Try to remove the thick skin with a blunt object like a key, rough towel or fingernail. Never remove skin with sharp objects like a knife. Thick skin forms callus and callus causes pressure on the tissues below. Because of anaesthesia, this pressure is not felt by the patient and damage is therefore more likely. Removal of the callus prevents this.
- Cover the damp skin with cooking oil, Vaseline, or any other oils. In this way the water is kept in the skin.
- Treat heel cracks as follows:
  - Soak for some hours in cool or warm soapy water;
  - Dry then apply Vaseline;
  - Put on socks;
  - Do this daily before going to bed.

The above skin-care procedures should be carried out at least three times daily whenever the skin seems dry. Advise the patient to keep oil near the washing bowl and to apply a little each time after washing the hands and feet.

Care of minor wounds and sepsis of hands and feet

Uninfected wounds: All patients must be constantly reminded to report immediately all minor wounds or burns of their hands and feet. These should be treated as soon as possible in the following manner:

(a) Wash with soap and water and a nailbrush;
(b) Soak in soapy water for 20 minutes;

(c) Cover with a simple dressing (avoid flavine, which delays healing);
(d) Splint in the position of function.

Minor burns (uninfected blisters) should merely be covered with adhesive plaster and not opened.

Infected wounds must always be treated seriously. Careful examination is essential to decide whether it is a simple case which can be dealt with locally or whether it should be referred to hospital. It must be remembered that leprosy patients who have lost sensation get very severe infection with gross swelling of the hands and feet to a degree seldom seen in non-leprous patients, merely because of delay in recognizing their trouble. (Frequently medical staff mistake these infections for severe leprosy, with disastrous consequences.)

Management is based on general surgical principles and the golden rules are:

- Free drainage;
- Soaks;
- Splinting.

Antibiotics are the second, not the first, line of defence

Provision of shoes: Feet that cannot feel need protection against injury. Shoes may protect leprosy patients against injury but can also increase damage if not properly fitting. Shoes which are too tight or too big will rub the feet and cause ulcers. Shoes with straps fixed with nails can also cause ulcers on the insensitive feet of a leprosy patient.

Insensitive feet need shoes with microcellular cushioning and a tough undersole

Physiotherapy: Joints which are not used will become stiff. Scar tissue tends to retract resulting in contractures. Muscles which are not used will shrink and result in muscle atrophy. All patients with weak or damaged hands and feet, or weak eyelids, should do suitable exercises.

Check each patient to see if he has normal ranges of motion. Exercises are of benefit in the following ways:

- Exercises can prevent loss of the normal range of motion and prevent stiffness. Stiffness may cause the patient to injure himself and do further damage.
- Exercises can improve or help to restore normal ranges of motion, strengthen weak muscles, and make muscles function more effectively.

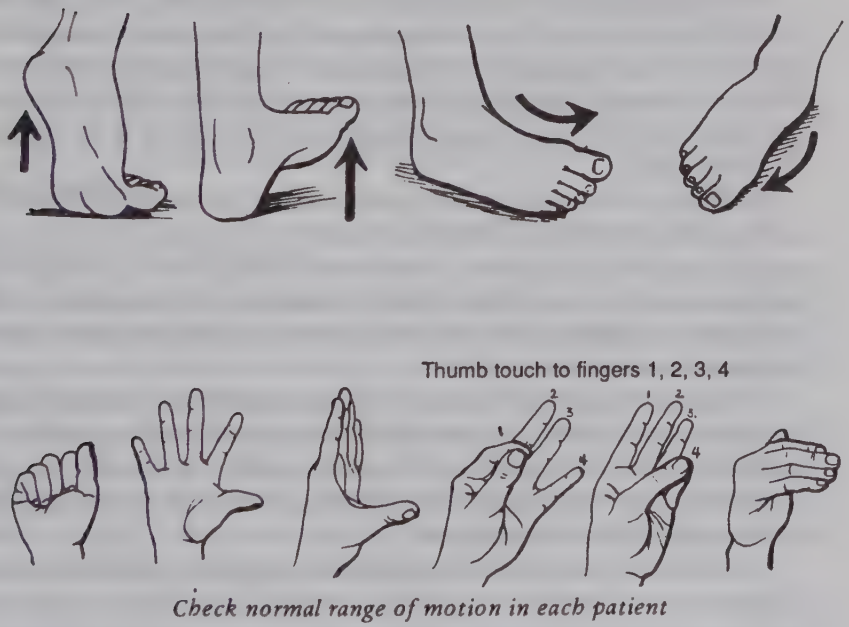


Figure 9.26 **Exercises for feet and hands**

There are two types of exercises: active and passive.

Active exercises are done to strengthen muscles which are weak. Only weak muscles can be strengthened by active exercises. Completely paralysed muscles cannot be strengthened by any type of exercise.

Passive exercises are done with the other hand or by another person. They are useful because they can lengthen ligaments and thus loosen stiff joints and can lengthen skin and muscles.

Patients should learn to make passive movements of joints if active movements are difficult because of stiffness or weakness.

Do passively what cannot be done actively

Exercises should not be done when there are injuries, infection, or cracks in the skin, and when nerves are painful. If the skin cracks when a patient is exercising, splint the hand or foot as straight as possible. This will prevent shortening of the scar when the skin heals and the resulting scar will then be long enough to allow a much wider range of movements.

If the aim of the exercise is to prevent or stop further stiffness of a joint, each movement should be made 5–10 times daily. This is enough provided the joint is exercised through its full range of movements. If, on the other hand, the aim of the exercise is to strengthen and improve the function of weak muscles, the more often the exercises are done the better.

These exercises must, however, be done carefully so the skin does not split or crack. Start with very gentle exercises, and then gradually introduce more strenuous ones. Let the patient decide the most convenient time for his exercises each day. This will ensure that he comes regularly for his exercises.

If the patient learns to do his exercises properly and does them regularly he can usually expect good results. Learning to do some hand exercises while walking about can be very rewarding for the patient. In addition, it is very useful for the patient to learn to do some exercises at home.

Health education: All leprosy patients with loss of sensation should be taught how to prevent further damage by avoiding injuries and infection.

Hands:

- Teach the patient to look and think before acting: hot or cold, sharp or blunt, dangerous or safe?
- Teach the patient not to grip tools too tightly;
- Teach home exercises.

Feet:

- Teach the patient to wash and inspect his feet daily for minor injuries and ulcers;
- Teach him to wear shoes and not to walk long distances;
- Teach first aid for small injuries;
- Explain the dangers of closed injury to the feet;
- Teach the patient not to stand for too long on one foot.

Teach personal hygiene and awareness of infectiousness of nasal discharges.

Prevention of blindness

Test for lagophthalmos during every visit by asking the patient to close his eyes and observing whether they close properly or not. In this way you will be able to detect cases of lagophthalmos early and you can refer the patients for proper treatment at an early stage.

Test the sensation of the cornea in every patient with patches on the face. If the cornea is insensitive, the patient will not feel foreign bodies in the eye and scars from corneal ulceration will lead to serious loss of sight. Constant awareness of the possibility of eye damage is essential. Refer patients with red eyes immediately.

Check the vision and always look for dilated ciliary vessels in all patients with multi-bacillary leprosy

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Prevention of defaulting

There are a number of reasons why a patient may give up his treatment. Examples and suggestions on how to overcome these problems are given on the following page.

No confidence in treatment	Explain to every new patient that leprosy is curable.
Shame	Explain to every new patient that leprosy is a disease like any other.
Thinks he has not got leprosy	Explain to every new patient diagnosed what it means to have leprosy and what can be done to cure him.
Thinks he is cured	Explain to all patients that macules may disappear, and they may feel better, but that treatment must not be stopped until the patient is declared cured by the doctor.
Treatment too long	Explain to all patients why treatment has to be continued and what the dangers are of stopping treatment.
Clinic too far	Supply drugs for 2 or 3 months if patients are living far away.
Irritations over treatment	Order your drugs in time to avoid running out of stock; organize clinic in such a way that waiting time is as short as possible. Treat leprosy patients yourself. Give proper advice on ulcer care. Give adequate treatment for tender nerves.
Disability	Provide adequate ulcer care. Refer patients with reactions for adequate treatment.
Ignorance	Explain to every new patient for the first three or four visits that leprosy is curable provided the patients take their drugs regularly until they are declared cured.

One of the reasons patients default, or do not turn up for treatment, is that health workers are often rude and ill-tempered. Abusing patients for having missed an appointment may only discourage them from coming back to the clinic. It is far more useful to patiently explain to them the need for regular visits to the clinic. Irregular attendances can be minimized by:

- Fixing a day for a well-organized clinic;
- Giving health education to all new patients;
- Home-visiting all patients living nearby;
- Tracing patients through their village chiefs if they live far away;
- Tracing all non-attenders immediately.

Outpatient care

It is the task of every hospital, health centre, and dispensary to take care of all patients with chronic diseases in their catchment area. This is best achieved by holding a special clinic for chronic diseases on a fixed day of the week. Such a clinic can take care of patients with leprosy, TB, and other chronic diseases (e.g., epilepsy).

The patients attending such a clinic should be given priority on that day. But if, for one reason or another, they come on a day other than the fixed clinic day, do not refuse to see them. Give them treatment, and advise them to come next time on the clinic day. To turn away a patient who has probably walked a long way to the health centre (and to tell him to come back tomorrow or the day after) is very demoralizing to him. It will ruin the doctor-patient relationship.

If it is possible, it is even better to run a daily clinic in which the most senior staff member sees all cases referred from OPD and MCH clinics, and patients with chronic diseases such as leprosy and TB. This will remove the stigma of attending special clinics.

The work should be divided among all staff. The normal health centre routine can be used — dressing ulcers in the dressing room, dispensing tablets at the dispensary, etc. The attendance register should, however, be filled in by the most senior member of the team, i.e., the person examining the patient.

Outpatient care has many advantages. Patients prefer it to long inpatient treatment, after which they would have to readjust to home life. They are more likely to report earlier for treatment if they know that they need not be admitted.

Isolation in hospital is not necessary because a few months of treatment is all that is needed to render the patient non-infectious as an outpatient. Hospital treatment is only necessary if there are complications, and it should be as short as possible. There is no need for special leprosy wards or hospitals because these contribute to the

idea that leprosy is something mysterious, thus making it more difficult for the patient to continue a normal life in the community.

Drug treatment

The traditional drug for leprosy has, until recently, been diamino-diphenyl-sulphone (DDS) or dapsone given for many years. Two major problems emerged with this form of treatment. The first was that, because the drug had to be taken for so many years, patient compliance became poor and many patients defaulted after some time. Secondly, the leprosy mycobacteria began to show resistance to DDS in many countries. Thus it became necessary to introduce new drugs and to formulate new treatment regimens which could solve these problems.

The new leprosy treatment regimens now combine several drugs which are taken at the same time. This form of treatment is referred to as multi-drug therapy (MDT). The regimen chosen depends on the classification of the patient. Multi-bacillary patients will require longer treatment schedules. Research has shown that using a combination of two or three drugs, including the powerful drug Rifampicin®, shortens the duration of treatment considerably, and at the same time minimizes the risk of the mycobacteria developing resistance to any of the drugs.

Duration of treatment for pauci-bacillary leprosy: The patients are treated for a period of 9 months, during which time they are closely supervised at least every 4 weeks.

Duration of treatment for multi-bacillary leprosy: Patients are treated for at least 2–3 years.

Treatment can be stopped once the course has been satisfactorily completed. In some countries, treatment is continued until the patient is smear negative. There is now enough evidence to show that the bacilli that are found in the skin smear after completing a full course of treatment are in fact the remains of dead mycobacteria. It takes some time before the body clears all the tissues of these dead bacilli. It is therefore not necessary to continue treatment for longer than the recommended period just because of the presence of dead bacilli in the skin smears. Note that by using the new MDT regimens the treatment periods have been considerably reduced. MDT must, however, be very strictly implemented and supervised.

Table 9.7 **WHO recommended multi-drug therapy for pauci-bacillary leprosy**

MDT	0–5 years	6–14 years	15 years and over
Dapsone daily self-administered (or by mother) at home	25 mg	50 mg	100 mg
Rifampicin 4 weekly supervised in health centre	300 mg	300 mg	600 mg

Table 9.8 **WHO recommended multi-drug therapy for multi-bacillary leprosy**

MDT	0–5 years	6–14 years	15 years and over
Dapsone daily self-administered	25 mg	50 mg	100 mg
Rifampicin 4 weekly supervised	300 mg	300 mg	600 mg
Clofazamine 4 weekly supervised	100 mg	200 mg	300 mg
Clofazamine self-administered	50 mg alternating days	50 mg daily	50 mg daily

Treatment of complications

Reactions

- Type I reversal reactions: refer to hospital immediately.
- Type II or ENL: mild reactions can be treated in the health centre with anti-inflammatory drugs: aspirin 300 mg three times a day and chloroquine 300 mg twice a day for 7 days and 300 mg once a day thereafter.

Rest is a very important for healing of neuritis.

Severe reactions must be treated with corticosteroids and other drugs: patients must therefore be referred to hospital.

Do not stop MDT because of reactions. Dapsone is no longer thought to be the cause of reactions, as in the past

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Indications for referral

- Mild reaction not getting better after 4 weeks' treatment at the health centre;
- Mild reaction getting worse after 1 or 2 weeks' treatment at the health centre;
- All patients with newly swollen and painful nerves (neuritis);
- All patients with newly swollen hands, feet, or face (oedema);
- All patients with new paralysis;
- All patients with fever for more than 3 days, not improving on chloroquine and aspirin.

Eye complications

Iritis: Refer urgently; give atropine eye ointment or drops if available.
Corneal ulcer: Chloramphenicol eye ointment, refer urgently.
Conjunctivitis: Chloramphenicol eye ointment 4-hourly, refer if no improvement after 2 days.
Lagophthalmos: Exercises: frequent forced closure of eyelids while the hands are holding the skin of the forehead. This exercise should be done 20 times each time, and three times daily.

Patients who cannot close their eyes completely should do the following exercise: place the hands on the cheek and forehead to fix

the muscles. Then close the eyes as tightly as possible. Keep them closed and count slowly to 10. Do this at least 40 times twice a day. The more often it is done, the better the results.

Prevention of drying of the cornea: put drops of saline or water in the eyes frequently and use Vaseline at night. Refer for surgical correction if insufficient or no improvement results from the exercises.

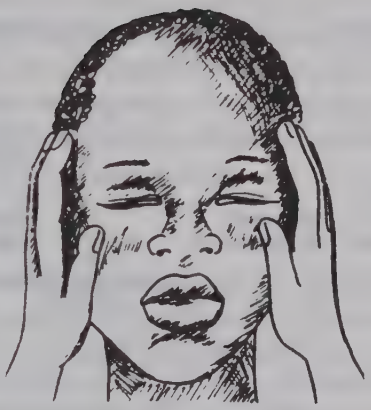


Figure 9.27 **Eye exercise for lagophthalmos**

Plantar ulcers

- In the acute stage rest in bed, preferably with the leg splinted on a pillow. In the pre-ulcer stage complete recovery usually takes 10 days; once an ulcer has formed recovery will take about 6 weeks.
- Antibiotics may be necessary, but only if there are systemic symptoms.
- Soaking twice daily and simple dressings are usually sufficient.
- Osteomyelitis must be treated using general surgical principles, but one should be as conservative as possible with sequestrectomy and amputation.
- When all this has been done, a walking plaster cast can be applied inside which the ulcer will heal spontaneously after 6 weeks.

6. Prevention and control

Reduce the number of infective cases and interrupt transmission with effective treatment of all the infective cases. These are the patients with B and L types of leprosy. After a few months of treatment the number of viable bacilli is reduced to nil.

It is essential to continue treatment until the course is completed. Active case-holding is required. This can only be achieved by proper registration of patients and proper follow-up of non-attenders by home visiting. If you have a health auxiliary attached to the health

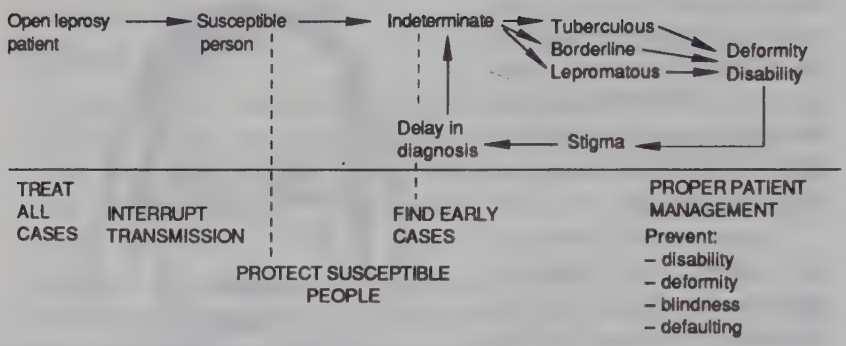


Figure 9.28 **Control cycle**

centre or dispensary, it is his duty to look for the non-attenders, to politely find out why they did not attend, and to encourage them to collect their drugs.

If you do not have a health auxiliary, other methods must be devised. If there are village health workers in your area it is their duty to visit such patients. This should be easy for them as they do not need to travel far since they live in the community themselves. When these arrangements are not possible, try to motivate the village chairmen to help in tracing non-attenders. Inform them about patients who have been lost sight of and keep on until answers are obtained from them.

Reduce opportunity for contact

Leprosy transmission is favoured by overcrowding, poor housing and low socio-economic conditions. The incidence of leprosy will go down when standards of living improve, i.e. better houses and correction of overcrowding.

Multi bacillary (B and L) patients must be taught personal hygiene with regard to their nasal secretions.

Reduce the number of susceptible people

There is no effective immunization against leprosy, but BCG immunization is of slight value. It stimulates the production of cell-mediated immunity in those individuals who are able to produce this

immunity. These are the people who would get tuberculoid or self-healing indeterminate leprosy when exposed to the *M. leprae* itself.

BCG has no protective value in individuals who will develop borderline or lepromatous leprosy on exposure. Drug prophylaxis with DDS is effective, but it is impractical to use chemoprophylaxis on a country-wide scale.

Early diagnosis and treatment

It is impossible to raise socio-economic standards rapidly. Therefore leprosy control is based primarily on the early treatment of as many cases as possible. The aim should, of course, be 100% of the cases. It is generally accepted that control only becomes effective when at least 75% of all infective cases have been found and are taking their drugs at least 75% of the time.

This cannot be achieved by offering treatment only to those who themselves report for treatment. About 25–50% of all cases report spontaneously — on average 2 years after clinical signs of the disease are recognizable.

To increase the number who come spontaneously for treatment extensive health education to the public is necessary. To increase the total number of diagnosed cases active case-finding is necessary:

- Examine all contacts of known multi-bacillary cases (B and L);
- Organize school surveys and village surveys;
- Be constantly aware of the possibility of leprosy when examining patients.

Only when all health workers diagnose and treat leprosy in all health institutions can the disease be eradicated. Treatment of leprosy should be given as close as possible to the place where the patients are living, that is, in the communities by Community Health Workers, in dispensaries and in health centres.

7. Action

Find out whether the community recognizes the need for a control programme. If not, start a careful education programme.

- Get preliminary information about leprosy:
  – observe people in your area for leprosy;
  – review previous reports and statistics about leprosy;
  – find out the attitude of your people towards leprosy.

- Prepare what you need to run a leprosy clinic:
  – discuss with your DMO and local leprosy officer;
  – order drugs, case record cards, attendance register;
  – enquire where you can send your complicated cases.
- Coach your staff:
  – how to deal with leprosy patients;
  – how to spread education and change attitudes.
- Start a health education programme (after consultation and in co-operation with the local leaders):
  – that leprosy can be cured;
  – that you will treat leprosy on an outpatient basis.
- Register and treat all known cases:
  – establish a regular leprosy treatment service;
  – provide health education to each case and family;
  – help patients with late complications;
  – follow-up all defaulters.
- Search for unknown cases:
  – get co-operation from local leaders to find suspicious cases;
  – organize school and village surveys once yearly;
  – encourage patients to bring their family members for a check-up.
- Organize a contact person (Community Health Worker, teacher, village chairman) in every village. They can distribute treatment to the patients, so that no patient need walk more than one hour to a treatment centre.
- Evaluate your service.

8. Summary

Leprosy is a chronic infectious disease of man caused by *M. leprae* and the tissue reaction against the leprosy bacillus. It mainly affects the peripheral nerves and skin. The disease is transmitted by close contact with an infectious multi-bacillary case (B or L). Paralysis, anaesthesia and the consequent cycle of injury and infection cause deformity and disability. Reactions and eye complications may hasten this disabling process. Diagnosis is either clinical, as in T cases (i.e., patches with anaesthesia, nerve enlargement) or clinical, plus skin smear, as in B and L cases. Management should concentrate on prevention of disability by regular multi-drug treatment. Control is mainly by case-finding and case-holding. Protection by BCG immunization is minimal.

Appendix: WHO Diarrhoea Treatment Chart

HOW TO ASSESS YOUR PATIENT

For dehydration

For other problems

	A	B	C	
1. Ask about: **Diarrhoea**	**Less than 4 liquid stools/day**	4 to 10 liquid stools/day	More than 10 liquid stools/day	**Longer than 14 days duration** **Blood in the stool**
Vomiting	None or a small amount	Some	Very frequent	
Thirst	Normal	Greater than normal	Unable to drink	
Urine	Normal	A small amount, dark	No urine for 6 hours	
2. Look at: **Condition**	Well, alert	Unwell, sleepy or irritable	Very sleepy, unconscious, floppy or having fits	**Severe undernutrition**
Tears	Present	Absent	Absent	
Eyes	Normal	Sunken	Very dry and sunken	
Mouth, tongue	Wet	Dry	Very dry	
Breathing	Normal	Faster than normal	Very fast and deep	
3. Feel: **Skin**	A pinch goes back quickly	A pinch goes back slowly	A pinch goes back very slowly	
Pulse	Normal	Faster than normal	Very fast, weak or can't be felt	
Fontanelle (in infants)	Normal	Sunken	Very sunken	
4. Take temperature				**Fever—38.5°C (101°F) or more**
5. Weigh if possible	Loss of less than 25 g for each kg of weight	Loss of 25–100 g for each kg of weight	Loss of more than 100 g for each kg of weight	
6. Decide	The patient has no signs of **dehydration**	If the patient has 2 or more of these signs, he has **some dehydration**	If the patient has 2 or more of these danger signs, he has **severe dehydration**	**If your patient has:** / **Then:**
	Use Plan A	**Use Plan B**	**Use Plan C**	Blood in the stool and diarrhoea for less than 14 days — Treat with an appropriate oral antibiotic for shigella dysentery. If the child is also – dehydrated – severely undernourished *or* – less than 1 year of age, reassess child's progress in 24–48 h. For the severely undernourished child, also refer for treatment of severe undernutrition.
				Diarrhoea for more than 14 days with or without blood; Severe undernutrition — Continue feeding and refer for treatment

For other problems (*Continued*)

Fever of 38.5°C (101°F) or greater — Show the mother how to cool the child with a wet cloth and fanning. Look for and treat other causes (e.g., pneumonia, malaria).

← *Continued on the left*

TREATMENT PLAN A TO TREAT DIARRHOEA

EXPLAIN THE THREE RULES FOR TREATING DIARRHOEA AT HOME:

1. GIVE YOUR CHILD MORE FLUIDS THAN USUAL TO PREVENT DEHYDRATION.
   SUITABLE FLUIDS INCLUDE:
   - The recommended home fluid or food-based fluids, such as gruel, soup, or rice water.
   - Breast milk or milk feeds prepared with twice the usual amount of water.
2. GIVE YOUR CHILD FOOD:
   - Give freshly prepared foods. Recommended foods are mixes of cereal and beans, or cereal and meat or fish. Add a few drops of oil to the food, if possible.
   - Give fresh fruit juices or bananas to provide potassium.
   - Offer food every 3 or 4 hours (6 times a day) or more often for very young children.
   - Encourage the child to eat as much as he or she wants.
   - Cook and mash or grind food well so it will be easier to digest.
   - After the diarrhoea stops, give one extra meal each day for a week, or until the child has regained normal weight.
3. TAKE YOUR CHILD TO THE HEALTH WORKER IF THE CHILD HAS ANY OF THE FOLLOWING:
   - passes many stools
   - is very thirsty
   - has sunken eyes

   } These 3 signs suggest your child is dehydrated.
   - has a fever
   - does not eat or drink normally
   - seems not to be getting better.

TEACH THE MOTHER HOW TO USE ORS SOLUTION AT HOME, IF:

- The mother cannot come back if the diarrhoea gets worse.
- It is national policy to give ORS to all children who see a health worker for diarrhoea treatment, or
- Her child has been on Plan B, to prevent dehydration from coming back.

SHOW HER HOW TO MIX AND GIVE ORS.

SHOW HER HOW MUCH TO GIVE:

- 50-100 ml (¼ to ½ large cup) of ORS solution after each stool for a child under 2 years old.
- 100-200 ml (½ to 1 large cup) for older children.
- Adults should drink as much as they want.

TELL HER IF THE CHILD VOMITS, wait 10 minutes. Then continue giving the solution but more slowly — a spoonful every 2-3 minutes.

GIVE HER ENOUGH PACKETS FOR 2 DAYS.

Note: While a child is getting ORS, he or she should be given breast milk or dilute milk feeds and should be offered food. Food-based fluids or a salt and sugar solution should *NOT* be given in addition to ORS.

EXPLAIN HOW SHE CAN PREVENT DIARRHOEA BY:

Giving only breast milk for the first 4-6 months and continuing to breast-feed for at least the first year.

Introducing clean, nutritious weaning foods at 4-6 months.

Giving her child freshly prepared and well-cooked food and clean drinking-water.

Having all family members wash their hands with soap after defecating, and before eating or preparing food.

Having all family members use a latrine.

Quickly disposing of the stool of a young child by putting it into a latrine or by burying it.

TREATMENT PLAN B
TO TREAT DEHYDRATION

1. AMOUNT OF ORS SOLUTION TO GIVE IN FIRST 4 TO 6 HOURS

Patient's age	2 4 6 8 10 12 18 2 3 4 6 8 15 adult months → years →					
Patient's weight in kilograms	3 5 7 9 11 13 15 20 30 40 50					
Give this much solution for 4-6 hours — in ml	200-400	400-600	600-800	800-1000	1000-2000	2000-4000
Give this much solution for 4-6 hours — in local unit of measure						

- Use the patient's age only when you do not know the weight.

NOTE: ENCOURAGE THE MOTHER TO CONTINUE BREAST-FEEDING.

If the patient wants more ORS, give more

If the eyelids become puffy, stop ORS and give other fluids. If diarrhoea continues, use ORS again when the puffiness is gone.

If the child vomits, wait 10 minutes and then continue giving ORS, but more slowly.

2. IF THE MOTHER CAN REMAIN AT THE HEALTH CENTRE

- Show her how much solution to give her child.
- Show her how to give it — a spoonful every 1 to 2 minutes.
- Check from time to time to see if she has problems.

3. AFTER 4 TO 6 HOURS, REASSESS THE CHILD USING THE ASSESSMENT CHART. THEN CHOOSE THE SUITABLE TREATMENT PLAN.

Note: If a child will continue on Plan B, tell the mother to offer small amounts of food.

If the child is under 12 months, tell the mother to:

- continue breast-feeding or
- if she does not breast-feed, give 100-200 ml of clean water before continuing ORS.

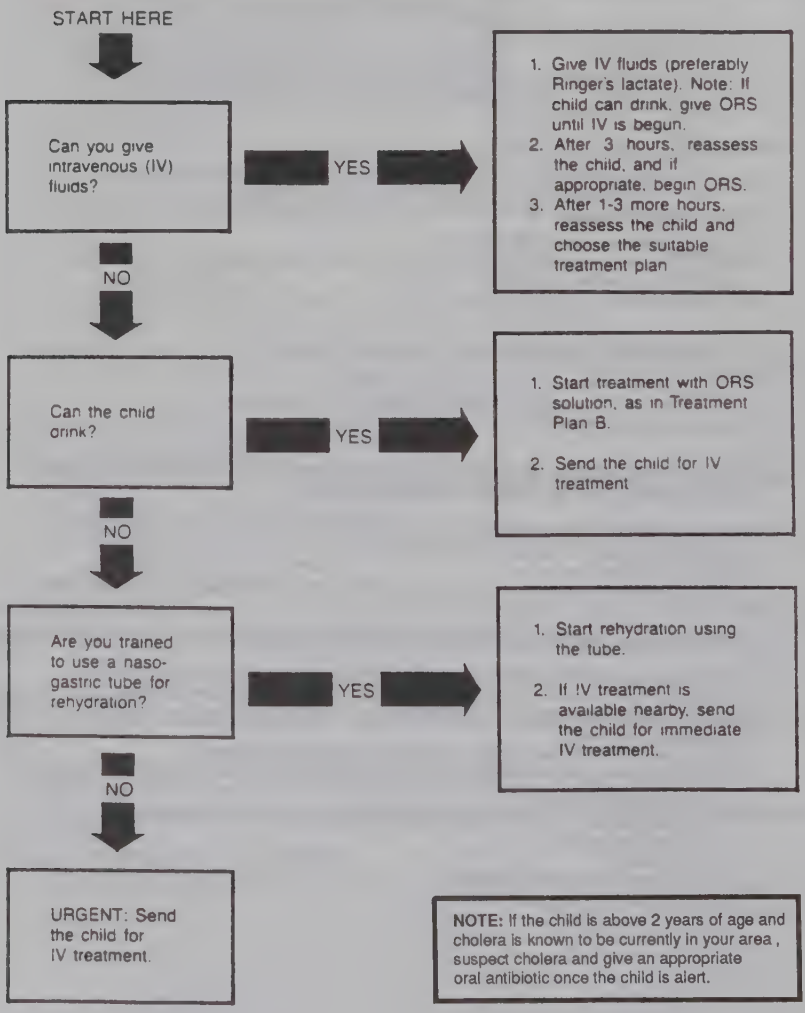
4. IF THE MOTHER MUST LEAVE ANY TIME BEFORE COMPLETING TREATMENT PLAN B

- Give her enough ORS packets for 2 days and show her how to prepare the fluid.
- Show her how much ORS to give to finish the 4-6 hour treatment at home.
- Tell her to give the child as much ORS and other fluids as he or she wants after the 4-6 hour treatment is finished.
- Tell her to offer the child small amounts of food every 3-4 hours.
- Tell her to bring the child back to the health worker if the child has any of the following:
  - passes many stools
  - is very thirsty
  - has sunken eyes

  } These 3 signs suggest the child is dehydrated.
  - has a fever
  - does not eat or drink normally
  - seems not to be getting better.

TREATMENT PLAN C TO TREAT SEVERE DEHYDRATION QUICKLY

Follow the arrows. If the answer to the questions is 'yes', go across. If it is 'no', go down.



NOTE: If the child is above 2 years of age and cholera is known to be currently in your area, suspect cholera and give an appropriate oral antibiotic once the child is alert.

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Since the first edition of this book was published in 1978 there have been many advances in health science, particularly in the prevention of some diseases—in the case of smallpox, leading to complete eradication. But communicable and vector-borne diseases are still the most common killing diseases in Africa, therefore this revised and up-to-date second edition will be as useful as was its predecessor. It combines all the essential information for diagnosing and managing individual patients with the appropriate practical methods for preventing and controlling these diseases, bearing in mind the facilities available at health-centre level.

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Chapter 5. Diseases caused by faecal contamination

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Chapter 6. Helminthic diseases

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Chapter 7. Diseases through contact with animals or their products

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Chapter 9. Tuberculosis and leprosy

Introduction. Tuberculosis. Leprosy.

Appendix: WHO diarrhoea treatment chart.

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