Serious childhood problems in countries with limited resources

Background book on *Management of the Child with a Serious Infection or Severe Malnutrition*

DEPARTMENT OF CHILD AND ADOLESCENT HEALTH AND DEVELOPMENT
WORLD HEALTH ORGANIZATION
GENEVA
2004

WHO Library Cataloguing-in-Publication Data

Serious childhood problems in countries with limited resources.

Companion volume to: Management of the child with a serious infection or severe malnutrition: guidelines for care at the first-referral level in developing countries.

1.Communicable diseases – therapy 2.Child nutrition disorders – therapy 3.Acute disease – therapy 4.Disease management 5.Delivery of health care, Integrated 6.Child 7.Teaching materials 8.Developing countries I.World Health Organization.

ISBN 92 4 156269 2 (NLM classification: WS 366)

Designed by minimum graphics
Printed in Switzerland

Contents

Acknowledgements

This book is the result of an international effort coordinated by the World Health Organization's Department of Child and Adolescent Health and Development. A special debt of gratitude is owed to Dr Harry Campbell (University of Edinburgh, Scotland) and to Professor Nathaniel Pierce (Johns Hopkins University, Baltimore, USA) for coordinating the preparation of the chapters and overall editing.

WHO thanks the following for their contributions to this book: Professor Ann Ashworth (UK); Dr Harry Campbell (UK); Dr Trevor Duke (Papua New Guinea/Australia); Professor Greg Hussey (South Africa); Dr Michael English (Kenya); Dr Stephen Graham (Malawi); Professor Elizabeth Molyneux (Malawi); Professor Malcolm Molyneux (Malawi); Professor Nathaniel Pierce (USA); Professor Barbara Stoll (USA); and Dr Giorgio Tamburlini (Italy).

WHO is grateful to the following for reviewing the manuscript at different stages:

Dr A. M. Aguilar, La Paz, Bolivia; Dr S. Allen, Oxford, UK; Dr P. Angos, Cebu City, Philippines; Dr C. Bernal Parra, Medellin, Colombia; Dr R. Bahl, New Delhi, India; Dr M.K. Bhan, New Delhi, India; Dr L. Bravo, Manila, Philippines; Dr D. Brewster, Casuarina, Australia; Dr J. Bunn, Liverpool, UK; Dr M. A. Flores-Verschoor, Buenos Aires, Argentina; Dr M. Funk, Battor, Ghana; Dr L. Gothefors, Umea, Sweden; Dr N. Kesaree, Karnataka, India; Dr M. Krawinkel, Giessen, Germany; Dr K.J. Nathoo, Avondale, Zimbabwe; Dr K. Pagava, Tbilissi, Georgia; Dr A. Palmer, Banjul, The Gambia; Dr H.P.S. Sachdev, New Delhi, India; Dr E. Salazar-Lindo, Lima, Peru; Dr P. Sharma, Kathmandu, Nepal; Dr D. Silimperi, Bethesda, USA; Dr G. Sperotto, Campinas, Brazil; Dr D. Woods, Observatory, South Africa; Dr D. Yaohua, Beijing, China.

Valuable inputs were provided by the WHO Clusters of Communicable Diseases and of Non-Communicable Diseases, Evidence and Information for Policy, and HIV/AIDS, Tuberculosis and Malaria, the WHO Departments of Blood Safety and Clinical Technology, Disability/Injury Prevention and Rehabilitation, Essential Drugs and other Medicines, Nutrition for Health and Development, Reproductive Health and Research, and Vaccines and Biologicals, and by the WHO Regional Offices.

Foreword

This book – part of a series of documents and tools supporting the IMCI (Integrated Management of Childhood Illness) strategy – has been prepared as a companion to the WHO manual entitled *Management of the Child with a Serious Infection or Severe Malnutrition: Guidelines for Care at the First-Referral Level in Developing Countries* (WHO/CAH/00.1, ISBN 92 4 154 531 3), which presents practical guidelines for the care of sick children in small hospitals that have basic laboratory facilities and a supply of essential drugs and inexpensive medicines.

The book is aimed at medical, nursing and other health care students, and presents a summary of the technical background and the evidence-base underlying the clinical guidelines. The book should also be useful for teachers of undergraduates in paediatrics and child health, and for workers in child health as part of their initial training or continuing professional development. It focuses on the major causes of childhood mortality – acute respiratory infections, diarrhoeal disease, malaria, measles, meningitis, HIV, severe malnutrition and neonatal problems. Each section deals with disease definition, burden of disease, aetiology, pathophysiology, clinical features, and management, and ends with suggestions for further reading. The manual also presents details of the general aims of hospital management of sick children – to identify and give priority attention to very sick children, to maintain adequate nutrition, to detect and correct anaemia, to detect and correct hypoxaemia, to control fever, and to monitor the progress of the child. In each of these areas, it presents details of the diagnosis and management of the problem. In addition, it summarizes the evidence linking these factors to a good/poor outcome and the evidence that intervention can control the factor and/or improve the outcome. At the end of each section there are a number of questions to check the learning and understanding of key concepts. A key to answers linking the responses to the text is found at the end of the book, page 59 ff.

It is hoped that this book will be a useful companion study guide to complement undergraduate education in paediatrics in medical and nursing schools. Presentation of the underlying patho-physiological processes should aid in the understanding of clinical signs and treatment regimens; details of the burden of disease should help students place the importance of each condition that is a threat to child health in an appropriate context; and the focus on the evidence base of the guidelines should encourage students to adopt a critical attitude when considering current and proposed new clinical practice. The principles and guidelines presented in this manual should be appropriate in most countries with limited healthcare resources. However, discussion and local adaptation of some sections may be appropriate before use in undergraduate teaching or health worker training.

The current book will best be studied in conjunction with the manual, *Management of the Child with a Serious Infection or Severe Malnutrition: Guidelines for Care at the First-Referral Level in Developing Countries* (WHO/CAH/00.1). At the beginning of each chapter, reference is made to the corresponding chapter. Both books are available on the WHO/CAH website for students who find it easier to look up the treatment recommendations under:

URL: http://www.who.int/child-adolescent-health/publications/CHILD_HEALTH/WHO_FCH_CAH_00.1.htm

1. Introduction

Global childhood mortality

Nearly eleven million children die each year before reaching their fifth birthday. About two thirds of these deaths are due to pneumonia, diarrhoea, measles, malaria, neonatal causes, or malnutrition. Death often results from a combination of these conditions, which typically account for three out of four sick children seeking care at a health facility.

About 22% of deaths are estimated to be due to conditions arising in the neonatal period. The remaining deaths are caused by acute respiratory infections (ARI), mostly pneumonia (about 20%), as well as diarrhoea (about 12%), malaria (8%), measles (5%), congenital anomalies (5%), HIV (4%), pertussis (3%) and tetanus (2%). WHO has estimated that childhood malnutrition is associated with about 54% of all child deaths.

Fig. 1-1 Major causes of death in children <5 years old worldwide, 2000 (WHO)

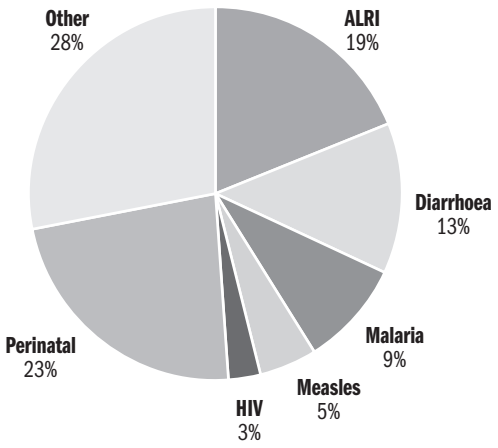
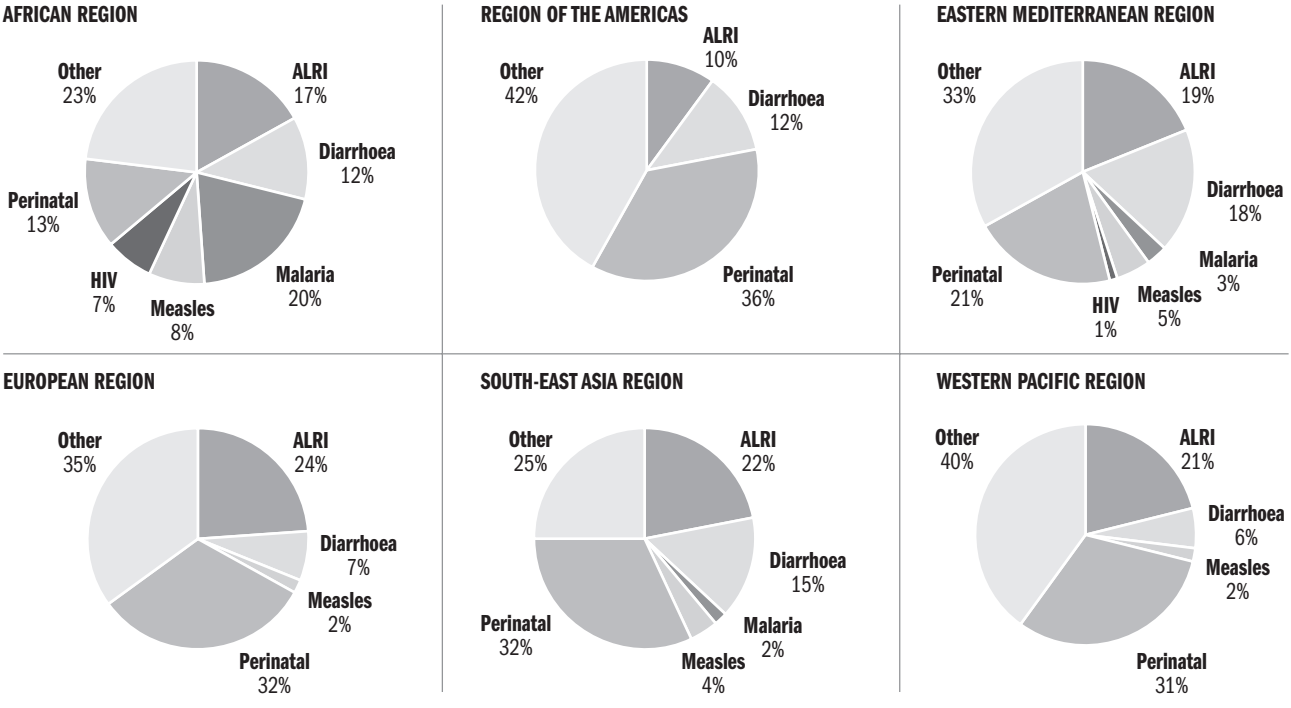


Fig. 1-2 Major causes of death in children <5 years old by Region, 2000 (WHO)



Some diseases play a larger role in some regions of the world. For example, malaria is more important in the African Region, and neonatal problems take a relatively larger share in the Americas, where overall child mortality has declined. The figures show the proportionate child mortality estimated for the year 2000 of the 6 major causes of death by region.

Low birth weight, suboptimal breastfeeding practices, malnutrition, and maternal child care practices increase the incidence and severity of these diseases. Important environmental risk factors include indoor air pollution (for pneumonia) and inadequate water supply and sanitation (for diarrhoeal diseases). HIV infection is an additional risk factor of growing importance.

An integrated approach to management of sick children and its benefits

Most developing countries have reported reductions in child mortality in the last 10 years. United Nations estimates show that deaths among under-five-year-olds in the developing world numbered 13.3 million in 1985, 12.2 million in 1993, and 10.9 million in 2000.

Much has been learned in recent years from disease-specific control programmes, such as those tackling diarrhoeal disease and ARI. However, children presenting with severe illness often have multiple disorders. A more comprehensive approach to assessment and management that ensures prompt recognition of other serious problems, such as septicaemia, anaemia, malnutrition and malaria, is therefore needed. The World Health Organization (WHO) and the United Nations Children's Fund (UNICEF) responded to this challenge by combining the successful approaches to ARI and diarrhoeal disease case management, and adding to them the clinical management of malaria, measles, meningitis and malnutrition. Integrated Management of Childhood Illness (IMCI) is the name given to this combined approach.

IMCI uses an evidence-based approach to development of case management guidelines and an integrated approach to training of health professionals in the management of important childhood illnesses. Priority in training is given to developing essential skills (including communication skills) and to key related issues, such as management of essential drugs and supplies, and efforts to improve timely care-seeking by families. The case management approach, which focuses on simple yet valid clinical signs, can be taught to health workers in peripheral health centres and health posts, and should greatly increase the access of families in developing countries to life-saving care for the most common and serious childhood illnesses.

According to the World Bank's 1993 report, this approach to management of common childhood infections and malnutrition is, out of all possible interventions, likely to have the greatest impact in reducing the global burden of disease. It is estimated that this approach alone can potentially prevent 14% of that burden in low-income countries and is among the most cost-effective health interventions in both low-income and middle-income countries.

Integrated case management guidelines

Integrated guidelines for management of the sick child have been developed by reviewing existing disease-specific guidelines, systematic literature reviews, clinical and health systems research, and field testing. Treatment guidelines are based on evidence from community-based intervention trials (e.g. ARI case management, vitamin A administration, and measles immunization), clinical trials (e.g. vitamin A treatment in measles), and observations of decreased case-fatality rates following the implementation of standard guidelines (e.g. treatment of severe malaria and diarrhoeal disease). Discussion and adaptation of these generic guidelines will be required before adoption by individual countries. The approach to case management is appropriate for first-level outpatient facilities, such as peripheral health centres. Diagnosis is based solely on valid, yet simple, clinical symptoms and signs that health workers can be trained to recognize accurately; laboratory studies are not required. Experience in several countries has shown that health workers trained in this approach make clinical decisions about case management that accord closely with the independent assessments of experienced paediatricians.

IMCI guidelines and training materials include:

- Guidelines for the management of important childhood infections and malnutrition at health centres and small hospitals
- Teaching materials for use in medical schools
- Materials for workshops on the management of drug supplies at health facilities
- Guidelines for monitoring and reinforcing skills during visits to facilities where health workers have completed IMCI training
- A guide to interventions to improve household management of childhood illness, including timely care-seeking.

Case management guidelines

Case management at the first-level health facility (health centre)

These guidelines describe the following basic steps:

- The health worker first ***assesses*** the child by asking questions, examining the child, and checking the immunization status.
- The health worker then ***classifies*** the child's illnesses, using a colour-coded triage system; each illness is classified according to whether it requires urgent referral, specific medical treatment and advice, or simple advice on home management.
- Specific ***treatments*** are then identified; if the child is to be referred urgently, the health worker gives only essential treatment before the child is transferred.
- The ***mother is taught*** how to treat her child at home, including how to give oral drugs, to increase fluid intake during diarrhoea, and to treat local infections.
- The ***mother is advised*** on how to recognize the signs which indicate that the child should immediately be brought to the clinic and is given the dates for routine follow-up; feeding practices are assessed and the mother is advised on how best to feed her child.
- Finally, any necessary ***follow-up instructions*** are given when the child returns to the clinic.

Case management at the first referral level (small hospital)

These guidelines are for use by doctors, senior nurses and other senior health workers who are responsible for the inpatient care of young children. They deliberately focus on management of the major causes of childhood mortality: pneumonia, diarrhoea, severe malnutrition, malaria, meningitis and measles. They complement standard paediatric textbooks, which are more comprehensive in their coverage and should be consulted for details of the management of other, less common, conditions or complications.

The guidelines are up-to-date, authoritative, and designed for small hospitals with limited laboratory capability but having a supply of inexpensive essential drugs. They may also be appropriate for large health centres that can admit a few sick children for inpatient care. They assume a minimum capacity for laboratory investigation: blood film examination for malaria parasites, estimation of haemoglobin or packed cell volume, blood glucose, and basic microscopy of CSF and urine. They avoid, however, expensive treatment options, such as use of newer antibiotics or mechanical ventilation, which are unlikely to be available.

Overview of the book

This background manual is a companion to the document entitled *Management of the Child with a Serious Infection or Severe Malnutrition: Guidelines for Care at the First Referral Level in Developing Countries*. It summarizes the key points in management of seriously ill children in hospital and describes the principles underlying the guidelines. It also presents **revision questions** to guide and direct student learning. The manual includes sections on triage and emergency treatment of sick children, on the assessment and management of children with the most common serious illnesses, including sick young infants, children with HIV infection, and children with severe malnutrition, as well as children on supportive care, monitoring the progress of the sick child and preparing the child to return home.

Further reading

Brady JP, Awan FB, Wafula EM, Onyango FE. Recognition of illness in very young infants by inexperienced health workers. *Ann Trop Paeds* 1993; **13**: 401–7.

Feachem RG. Preventing diarrhoea: what are the policy options? *Hlth Policy Plan* 1989; **1**: 109–17.

Garenne M, Ronsmans C, Campbell H. The magnitude of mortality from acute respiratory infections in children under 5 years in developing countries. *Wld Hlth Statist Quart* 1992: **45**: 180–91.

Hussey GD, Klein M. A randomised, controlled trial of vitamin A in children with severe measles. *New Engl J Med* 1990; **323**: 160–4.

Kirkwood B, Gove S, Rogers L, Levyt J, Arthur P, Campbell H. Potential interventions for the prevention of childhood pneumonia in developing countries: a systematic review. *Bull WHO*, 1995; **73**: 793–8.

Lee LA, Dogore R, Redd SC, Dogore E, Metcholk B, Diabata J, Assendelft OW, deCock K, Patrick E, Herrington J. Severe illness in African children with diarrhoea: implications for case management strategies. *Bull WHO* 1995; **73**: 779–85.

Mulholland EK, Simoes EAF, Costales MOD, McGrath EJ, Manalac EM, Gove S. Standardized diagnosis of pneumonia in developing countries. *Pediatr Infect Dis J* 1991; **11**: 77–81.

Sazawal S, Black RE. Effect of pneumonia case management on mortality in neonates, infants, and preschool children: a meta-analysis of community-based trials. *Lancet Infect Dis* 2003; **3**(9):547–556.

WHO Division of Diarrhoeal and Acute Respiratory Disease Control. Integrated management of the sick child. *Bull WHO* 1995; **73**: 735–40.

WHO. CAH: Child health epidemiology. http://www.who.int/child-adolescent-health/OVERVIEW/CHILD_HEALTH/child_epidemiology.htm

WHO Global Programme on Evidence for Health Policy. *The Global Burden of Disease 2000: project aims, methods and data sources*. WHO Discussion Paper 36. Geneva: WHO, 2001. http://www.who.int/evidence

World Bank: *World development report 1993: Investing in health*. Washington 1993

Questions

1.1 What are the five main causes of death in young children in developing countries and what percentage of deaths do they account for?

1.2 List the main risk factors which increase the incidence and severity of these diseases in childhood.

2. Emergency Triage, Assessment and Treatment (ETAT)

Deaths in hospital often occur within 24 hours of admission. Many of these deaths could be prevented if very sick children are identified soon after their arrival in the health facility and treatment is started immediately. This section outlines a process of rapid triage to determine whether any emergency/priority signs are present.

See also: ***Management of the Child with a Serious Infection or Severe Malnutrition*****, Chapter 1, Page 1.**

Background

In developing countries, the most common illnesses for which children are brought to hospital are malaria (in endemic areas), pneumonia, diarrhoea (often with dehydration), conditions that cause altered consciousness (including cerebral malaria, seizures, and meningitis), malnutrition, and, in young infants, sepsis. Many children with these problems are seriously ill and at risk of dying unless they are rapidly identified by systematic triage, adequately assessed, and given appropriate emergency treatment without delay. However, a WHO study of hospital care in seven developing countries showed that triage and emergency care were poorly performed, especially in non-teaching hospitals. Common deficiencies included: no formal system of triage or standard assessment, few or no standard treatment guidelines, long delays in providing emergency care, poor training of staff to provide triage and emergency care, understaffing (especially at night), inadequate organization of facilities for triage, and inadequate supplies of drugs and other essential materials. It was concluded that improving the quality of triage and emergency care should contribute substantially to reducing morbidity and mortality in sick infants and children who are brought to hospital for care.

To improve this situation calls for the development of a system of triage, assessment and emergency treatment, which suits the needs and limited resources of developing countries. Nurses, paramedical and non-medical staff, as well as doctors, each plays an important role and must be trained in triage. Competent round-the-clock coverage must be ensured in all hospital locations where sick children present for care. Training of nurses and paramedics is especially important in settings where a doctor may only be on the premises during part of the day. Facilities must be organized to support this approach. Essential drugs must be available immediately without the family having to purchase them before treatment is given; other necessary supplies and equipment must also be on hand.

Development of a system for triage and emergency care in developing countries

Efforts to improve the emergency care of children began in the USA and UK in the 1980s and led to development of the Advanced Paediatric Life Support (APLS) system, which is now widely used in developed countries. The system proposes the assessment and treatment of the most urgent problems in the following order of priority:

Airway (obstruction)
Breathing (respiratory arrest)
Circulation (cardiac arrest, arrhythmia, shock), and
Disability (altered consciousness, other neurological deficit).

Assessment and treatment of other urgent problems follow this "ABCD" sequence, which health workers will remember easily as the priority order in assessment.

The principles of triage and emergency care embodied in APLS also apply to the developing countries. However, the guidelines had to be revised to match the needs and resources of those countries. The ETAT (**e**mergency **t**riage, **a**ssessment and **t**reatment) guidelines, developed by WHO, focus primarily on the most important illnesses of young children in developing countries. They use simple screening and assessment criteria to identify and classify the most seriously ill children, and proven therapies that are cost-effective and easily delivered by trained nurses, paramedics as well as doctors.

Triage and emergency treatment

The objective of triage is rapid identification of children with signs of serious illness so that they may be treated without delay. Each child must be seen immediately on arrival in the hospital and rapidly classified in one of the following groups:

(i) ***emergency signs*** present – start emergency treatment immediately;

(ii) ***priority signs*** present – start full assessment and treatment promptly;

(iii) ***a non-urgent case*** – can await his/her turn for assessment and treatment.

Triage may be done in 15–20 seconds by medical staff or by non-medical staff (after appropriate training) as soon as the child arrives, and no special equipment is needed for this. If the first person the child encounters is a gatekeeper or clerk, these persons should also be trained to recognize the emergency signs and ensure that a child with these signs is seen, assessed and treated *immediately* by a doctor, nurse or paramedic. No child who has been assessed and found to have emergency or priority signs should be kept in the queue to wait for his or her turn.

Emergency signs in the ETAT guidelines identify children with immediately life-threatening conditions which are most frequently seen in developing countries, such as obstruction of the airway, shock, severely altered CNS function (coma or convulsions), and severe dehydration. The signs selected are both highly sensitive and sufficiently specific to be practical, and they can be readily and reliably detected by any level of trained staff. The **chart** opposite lists these signs and also the priority signs. Note that the emergency signs in ETAT differ from those in APLS.

The chart also shows the required emergency treatments. These treatments, which must be given by a trained health worker, are intended to stabilize the child and reduce the immediate risk of death, so that the child may be more thoroughly assessed and treated appropriately.

How well does ETAT work?

ETAT guidelines have been formally evaluated in two developing countries, Brazil and Malawi. The goal was to determine how well they detected "emergency" and "priority" conditions, and how well the nurses performed when screening patients and giving emergency treatment. In these studies ill children, mostly aged 2 months to 5 years, seen in the hospital paediatric outpatient clinic were evaluated by nurses trained to use the ETAT guidelines and separately by paediatricians trained in the APLS system. **Table 2-1** shows the distribution and ETAT classification of children in the Brazil study.

Table 2-1 ETAT classification, hospital admission and early death among 3 837 children in Brazil
(data from Tamburlini et al. 1999)

ETAT classification	% of all children assessed	% of all children admitted to hospital	% of all children dying in first 24 hours
Group 1 (emergency)	3	37	71
Group 2 (priority)	17	49	29
Group 3 (not emergency or priority)	80	14	–

A total of 20% of the children were classified as having emergency or priority conditions, and the majority who were admitted to hospital or who died during the first 24 hours came from these groups. This shows that the system correctly identified children who were very seriously ill and in need of urgent treatment in hospital. The two studies concluded that nurses – after only 20 hours of training in the use of ETAT – could identify seriously ill children with a high level of sensitivity and specificity when compared with APLS-trained paediatricians.

Nurses also performed well in choosing and promptly giving the correct emergency treatment. During the first week of the Brazil study, 89% of emergency conditions were correctly treated and this increased to 96% in the second week, which suggests that practice improved the quality of the nurses' performance.

Conclusions

A substantial proportion of children in developing countries who arrive in hospital for treatment are seriously ill and at risk of dying. This risk increases when assessment and effective treatment are delayed, as happens in many hospitals. The emergency triage, assessment and treatment (ETAT) guidelines focus on prompt, round-the-clock screening of all sick children and rapid provision of effective treatment for those who are found to be seriously ill. The guidelines have been adapted to meet the problems and resources in developing countries. Nurses trained to follow the ETAT guidelines have performed well in both assessment and treatment, compared with paediatricians. ETAT is a valuable tool to strengthen the initial assessment

Triage of all sick children

EMERGENCY SIGNS

If any sign positive: give treatment(s), call for help, draw blood for emergency laboratory investigations (glucose, malaria smear, Hb)

			TREAT
1. ASSESS: AIRWAY AND BREATHING	■ Obstructed breathing *or* ■ Central cyanosis *or* ■ Severe respiratory distress	**ANY SIGN POSITIVE***	IF FOREIGN BODY ASPIRATION ➥ Manage airway in choking child IF NO FOREIGN BODY ASPIRATION ➥ Manage airway ➥ Give oxygen ➥ Make sure child is warm
2. ASSESS: CIRCULATION	Cold hands with: ■ Capillary refill longer than 3 seconds, *and* ■ Weak and fast pulse	**ANY SIGN POSITIVE*** Check for severe malnutrition	➥ Stop any bleeding ➥ Give oxygen ➥ Make sure child is warm IF NO SEVERE MALNUTRITION: ➥ Insert IV and begin giving fluids rapidly If not able to insert peripheral IV, insert an external jugular or intraosseous line IF SEVERE MALNUTRITION: *If lethargic or unconscious:* ➥ Give IV glucose ➥ Insert IV line and give fluids *If not lethargic or unconscious:* ➥ Give glucose orally or by NG tube ➥ Proceed immediately to full assessment and treatment
COMA CONVULSING	■ Coma *or* ■ Convulsing (now)	**IF COMA OR CONVULSING***	➥ Manage airway ➥ If convulsing, give diazepam or paraldehyde rectally ➥ Position the unconscious child (if head or neck trauma is suspected, stabilize the neck first) ➥ Give IV glucose
SEVERE DEHYDRATION (only in child with diarrhoea)	Diarrhoea plus any two of these: ■ Lethargy ■ Sunken eyes ■ Very slow skin pinch	**DIARRHOEA plus TWO SIGNS POSITIVE*** Check for severe malnutrition	➥ Make sure child is warm. IF NO SEVERE MALNUTRITION: ➥ Insert IV line and begin giving fluids rapidly IF SEVERE MALNUTRITION, ➥ Do **not** insert IV ➥ Proceed immediately to full assessment and treatment

PRIORITY SIGNS **These children need prompt assessment and treatment**

- ■ Visible severe wasting
- ■ Oedema of both feet
- ■ Severe palmar pallor
- ■ Any sick young infant (< 2 months of age)
- ■ Lethargy
- ■ Continually irritable and restless
- ■ Major burn
- ■ Any respiratory distress or
- ■ An urgent referral note from another facility

Note: If a child has trauma or other surgical problems, get surgical help or follow surgical guidelines*

** Check for head/neck trauma before treating child—do not move neck if cervical spine injury possible.*

NON-URGENT

Proceed with assessment and further treatment according to the child's priority

and management of seriously ill children in developing countries, thereby reducing the risk of death.

Further reading

Gove S, Tamburlini G, Molyneux E, Whitesell P, Campbell H. Development and technical basis of simplified guidelines for emergency triage assessment and treatment in developing countries. WHO Integrated Management of Childhood Illness (IMCI) Referral Care Project. *Arch Dis Child* 1999; **81**:473–7.

Nolan T, Angos P, Cunha AJ, Muhe L, Qazi S, Simoes EA, Tamburlini G, Weber M, Pierce NF. Quality of hospital care for seriously ill children in less developed countries. *Lancet* 2001; **357**:106–10.

Robertson MA, Molyneux EM. Triage in the developing world – can it be done? *Arch Dis Child* 2001; **85**:208–13.

Robertson, MA, Molyneux EM. Description of cause of serious illness and outcome inpatients identified using ETAT guidelines in urban Malawi. *Arch Dis Child* 2001a; **85**:214–7.

Tamburlini G, Di Mario S, Maggi RS, Vilarium MD, Gove S. Evaluation of guidelines for emergency triage assessment and treatment in developing countries. *Arch Dis Child* 1999; **81**: 478–82.

Questions

2.1 List some of the common problems in the emergency care of sick children in developing countries.

2.2 Explain the meaning of A,B,C and D in the Advanced Paediatric Life Support (APLS) system. Describe the equivalent system in the WHO Emergency Triage and Assessment and Treatment (ETAT) approach.

3. Cough or difficult breathing

Cough and difficult breathing are common problems in young children. The causes range from a mild, self-limited illness to severe, life-threatening disease. This section focuses on the most important causes of acute cough (common cold and pneumonia), chronic cough (tuberculosis), and wheeze (asthma).

See also: *Management of the Child with a Serious Infection or Severe Malnutrition*, Chapter 3, Page 29.

Definitions

Cough. The major cause of cough in young children is a respiratory infection. Acute respiratory infections present with cough of less than 30 days' duration. Chronic cough is defined as cough lasting continuously for 30 days or more. **Difficult breathing** can present as lower chest wall indrawing (i.e. an inward movement of the lower chest on inspiration) or respiratory distress (i.e. inability to eat, drink or speak due to breathlessness). **Wheeze** is a musical noise when breathing out (expiration) and stridor is a harsh noise when breathing in (inspiration).

Very severe pneumonia is defined as a condition in a child with cough or difficult breathing with at least one of the following: central cyanosis, severe respiratory distress, inability to breastfeed or drink, vomiting everything, convulsions, lethargy, or unconsciousness

Severe pneumonia is defined as a condition in a child with cough or difficult breathing with at least one of the following: lower chest wall indrawing, nasal flaring or grunting (in young infants) but with no signs of very severe pneumonia.

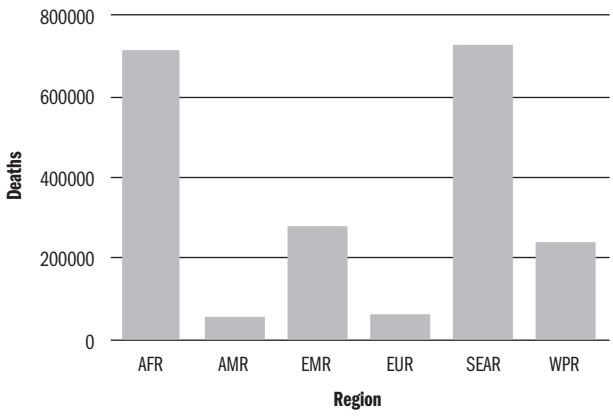
Pneumonia is defined as a condition in a child with cough or difficult breathing and with signs of fast breathing (60 breaths or more per minute in young infants under 2 months old, 50 breaths per minute in a child aged 2 months up to 12 months, 40 breaths or more per minute in a child aged 12 months up to 5 years) with no signs of severe or very severe pneumonia. On auscultation with a stethoscope, crepitations or signs of consolidation such as bronchial breathing might be heard, but this is not used for the classification of severity.

Burden of disease

The common cold is the commonest infection in childhood and children experience about 4–8 episodes per year. The incidence of pneumonia among under-5-year-olds in developing countries is about 0.3 new episodes per child per year, or about 150 million new episodes each year globally. Pneumonia is the most important cause of death in young children globally. It is either the first or second largest cause of death in most developing countries. The relative importance of pneumonia as a cause of death is greatest in those countries with the highest infant mortality rates. The case fatality rate is particularly high in the first 6 months of life.

The most important causes of *wheeze* in the first 5 years of life are bronchiolitis, asthma, and wheeze associated with

Fig. 3-1 Number of deaths from acute lower respiratory tract infections (mainly pneumonia) in children under 5 years of age, estimated for the year 2000, by region (WHO)



AFR African Region, AMR American Region, EMR Eastern Mediterranean Region, EUR European Region, SEAR South-East Asian Region, WPR Western Pacific Region

the common cold. Asthma is less common in developing than in industrialized countries, and it appears to increase as countries undergo economic development. There are therefore widespread geographical differences in the prevalence of asthma and the most likely cause of wheeze in any setting will depend on the relative importance of bronchiolitis and asthma in that setting. *Stridor* is not a common presentation in most developing countries where diphtheria has been controlled by immunization.

Common cold

Aetiology. The common cold is a self-limiting infection caused by a wide variety of respiratory viruses including rhinovirus, coronavirus, influenza virus, adenovirus and respiratory syncytial virus.

Pathophysiology. The common cold is an acute infection of the nose, pharynx and major bronchi. The inflammation may extend up the Eustachian tube to the tympanic membrane in the middle ear. It does not involve the smaller airways in the lung.

Clinical presentation. Children present with a cough or the parents report difficult breathing that is not associated with signs of pneumonia (see below). Nasal discharge which can vary from clear to purulent is common. Fever may be present.

Management. There is no curative treatment. In particular, there is no evidence that an antibiotic reduces the duration of symptoms or the severity of symptoms, or prevents complications such as otitis media or pneumonia from occurring. A purulent nasal discharge does not imply bacterial infection and the need for antibiotic treatment. The common cold can include bronchitis, but again this is viral in origin and antibiotic treatment is not indicated. Treatment of high fever with an antipyretic may be helpful. The majority of cough and cold remedies are ineffective in young children and some may be harmful or expensive. The treatment of the common cold with antibiotics is an important cause of their over-use which drives the development of drug resistance in populations. Unnecessary use of antibiotics for the common cold in an individual child may increase the risk of more severe forms of pneumonia (such as staphylococcal pneumonia) or pneumonia with drug-resistant bacteria.

Complications include otitis media, poor feeding, and pneumonia and these should be treated appropriately.

Pneumonia

Aetiology. Research studies using lung aspiration to obtain a sample of lung fluid have accurately identified the main causes of pneumonia as bacterial rather than viral, as in the industrialized countries. The main causes in children aged 2 months to 5 years are *Streptococcus pneumoniae* (pneumococcus), *Haemophilus influenzae* type b (and to a lesser extent *Staphylococcus aureus*) in almost all countries where this has been studied. Pneumonia in young infants under 2 months of age is associated with a wider range of bacterial agents including *Staphylococcus aureus* and gram-negative bacteria such as *E.coli* and *Klebsiella*.

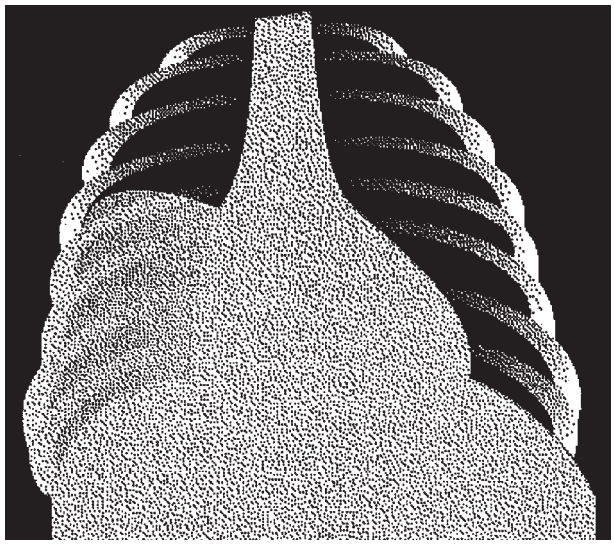
The most important viral pneumonia is caused by respiratory syncytial virus (RSV). This virus can also cause a severe illness called bronchiolitis.

HIV infection increases the risk of bacterial pneumonia. In the first 6 months of life, HIV-infected children can develop pneumocystis pneumonia, an opportunistic infection (see Ch. 10 on HIV/AIDS).

Pathophysiology. The infection and inflammation in the alveoli (pneumonia) and small airways (bronchiolitis) lead to decreased compliance (increased stiffness) of the lungs, an increase in the respiratory rate, and lower chest wall indrawing. A combination of inflammatory fluid in the alveoli and a mismatch between ventilation and perfusion in the lungs can interfere with the transfer of oxygen into the blood and cause hypoxia.

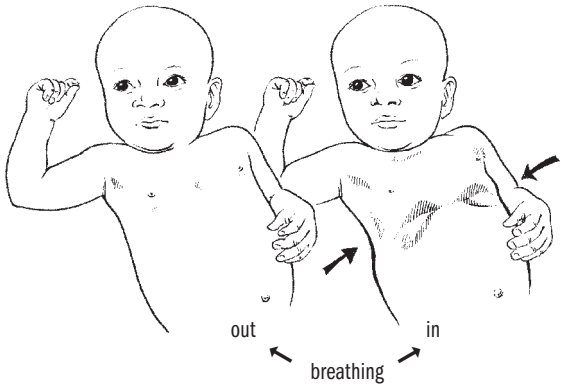
Clinical presentation. Chest X-ray appearances of pneumonia can be confined to one lobe or segment or be diffuse throughout the lungs of the child. However, the clinical

Fig. 3-2 Schematic representation of the X-ray finding of a lobar consolidation of the right lower zone



presentation in both cases is fast breathing and/or lower chest wall indrawing. Fast breathing detects a higher proportion of pneumonia cases in young children than auscultatory signs such as reduced air entry, bronchial breathing or coarse crepitations. Cut-off rates for the diagnosis of fast breathing have been set and balance the need to detect most cases of pneumonia (high sensitivity) against the need not to label too many children with the common cold as having pneumonia (high specificity or low false-positive rate). The respiratory rates vary with age (≥60 for young infants under 2 months, ≥50 in children 2 months to 1 year, ≥40 in children 12 months up to 5 years) since normal respiratory rate falls with age.

Fig. 3-3 Lower chest wall indrawing – with inspiration, the lower chest wall moves in



Children with RSV bronchiolitis also present with fast breathing and lower chest wall indrawing, as in pneumonia, but in addition have wheeze and hyperinflation of the chest.

Hypoxia is best identified by use of a pulse oximeter (which measures reduced oxygen saturation in the blood) but can be recognized clinically by signs such as central cyanosis, respiratory distress causing inability to drink, severe lower chest wall indrawing, very fast breathing (≥70 per minute), grunting with every breath or head nodding. However, the sensitivity of these clinical signs is lower than that of pulse oximeter and so their use will detect a lower proportion of hypoxic children.

Management. *Antimicrobial treatment*. Clinical tests to identify the cause of pneumonia perform poorly, so the treatment has to be based empirically on clinical signs. Most cases of pneumonia in children aged over 2 months can be treated at home with simple, inexpensive oral antibiotics such as cotrimoxazole or amoxicillin. Activity of penicillin V (phenoxymethyl penicillin) is not good against *Haemophilus influenzae* type b and so is not recommended. Children with severe pneumonia have a poorer prognosis and so should be admitted to hospital for treatment with parenteral antibiotics. Benzyl penicillin is the first-line treatment as it is effective against the two major bacterial pathogens. Chloramphenicol should be restricted to the few children who have very severe pneumonia in order to limit the development of drug resistance, and because its use is sometimes associated with rare but serious adverse effects. It is active against a wider range of bacterial pathogens including *Staphylococcus aureus* and Gram-negative organisms. A second-line treatment would be benzyl penicillin plus gentamicin, which also has broad-spectrum activity against bacteria.

Pneumonia in young infants under 2 months of age is associated with a much higher case-fatality rate, so all cases should be admitted to hospital for parenteral treatment. In addition, since the range of bacterial agents causing pneumonia is much wider, the first-line treatment is benzyl penicillin and gentamicin.

Oxygen therapy. Children with hypoxia have a higher mortality. Trials have shown that low-flow methods (1–2 litres per minute) of oxygen delivery are effective at treating hypoxia, and that nasal prongs and the nasal catheter perform as well as the nasopharyngeal catheter and do not cause the serious side-effect of gastric distension. Face masks are not tolerated well by children and so tend not to be effective, and head boxes can be dangerous and require large flows of oxygen. These methods are not recommended for these reasons.

The management of pneumonia in children with HIV infection follows the same principles. *Streptococcus pneumoniae* is the commonest cause of pneumonia in these children and responds well to the antibiotics described above. However, in countries where the prevalence of HIV infection is very high, infants in the first 6 months of life who present with signs of hypoxia are commonly found to have *Pneumocystis carinii* pneumonia (PCP) and require treatment with high-dose cotrimoxazole, steroids and prolonged oxygen therapy.

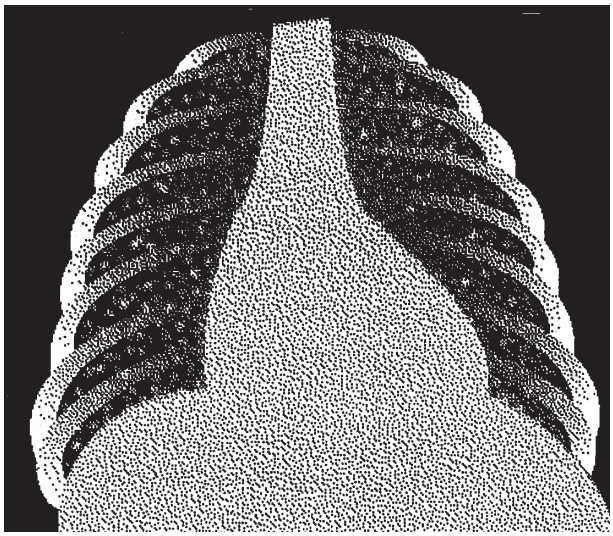
Tuberculosis

Aetiology. Tuberculosis is an infection usually caused by the bacterium *Mycobacterium tuberculosis*.

Pathophysiology. Pulmonary tuberculosis starts with infection in the peripheral lung, with associated infection in a draining lymph node. Subsequent natural history depends on the immune status of the child. In a severely malnourished or otherwise immunocompromised child, progression to miliary tuberculosis can occur with dissemination of infection throughout the lung. Spread by

the bloodstream may lead to infection in other sites (e.g. meninges, vertebrae, joints or kidneys). In a child with good immune responsiveness, the infection will be contained and then regress or may result in a post-primary infection of the lung. Primary infections lead to sensitivity to tubercular protein, as shown by the response to purified protein derivative (PPD), for example by a positive Mantoux test.

Fig. 3-4 Appearance of miliary tuberculosis: widespread small patchy infiltrates throughout both lungs: "snow storm appearance" (X-ray)



Clinical presentation. The diagnosis of tuberculosis in young children remains very problematic. Sputum is usually not present and gastric aspirates and laryngeal swabs are often negative in children with TB. In addition, the Mantoux test can be difficult to interpret in children who have been immunized with BCG (a reaction less than 10 mm can be due to BCG) or who have miliary TB, severe malnutrition or recent measles; the Mantoux test may be negative in a child with TB. Nevertheless, it is important to attempt to obtain early morning gastric aspirates, CSF (if clinically indicated), or pleural fluid for ZN (Ziehl-Neelsen) staining, followed by microscopic examination for acid-fast bacilli, and for culture.

It is important to distinguish tuberculosis from other causes of chronic cough either by history (foreign body, asthma, pertussis, or HIV) or by the findings on clinical or X-ray examination of the chest (bronchiectasis or lung abscess). HIV makes the diagnosis of TB even more difficult because other HIV-related diseases, e.g. lymphocytic interstitial pneumonitis, may present in a similar way to pulmonary TB and because the interpretation of tuberculin skin testing and chest X-rays is less reliable.

In summary, the three key clinical features which may point towards a diagnosis of TB in children are:

1. Contact with a pulmonary TB case.
2. Lack of response of respiratory symptoms and signs to broad-spectrum antibiotics.
3. Weight loss or failure to thrive.

Management. *Antimicrobial treatment*. Randomized controlled trials have compared various treatment regimens and have defined the most effective treatment schedules. It is essential to follow exactly the national TB guidelines, and to register a child with TB with the national TB control programme. Failure to do so leads to a risk that the treatment will be less effective and there will be more drug resistance. Drug-resistant (or multidrug-resistant) TB is becoming a major problem in some developing countries since second-line drugs are much more expensive and may not be easily affordable. Monitoring of treatment is also essential for the same reasons. Directly observed treatment strategy (DOTS) has many advantages and been adopted by many countries. Public health measures such as contact tracing are an essential part of the management of a case of TB.

Other treatment. It is important to pay attention to the nutrition of these children. Attempts should be made to identify the index case and then to screen all household contacts of the index case (usually an adult with pulmonary TB). All such household contacts (if necessary, school contacts as well) should be checked for undetected cases of tuberculosis and should be treated, where appropriate, or receive anti-TB prophylaxis according to national TB control guidelines.

Asthma

Aetiology. Asthma is an "immune" disorder whose precise cause is not known. It is commoner in "atopic" families where eczema, hay fever and allergic rhinitis are commonly found. Triggers to asthmatic attacks vary widely and can include animal dander, pollen, house fomites (house dust mite exposure), exposure to tobacco smoke, and viral respiratory infections.

Pathophysiology. Asthma is an inflammatory condition of the small airways which involves smooth muscle spasm, mucosal oedema and hyper-secretion of mucus.

Clinical presentation. Asthma presents with bronchial hyper-reactivity and reversible airways obstruction leading to recurrent episodes of cough, wheeze and breathlessness. Diagnosis can be very difficult as asthma may not be apparent until the child is older and had several episodes of wheeze.

Management. *Reliever therapy*. Reliever drugs should be used to reverse airways obstruction. The main agents are active on the beta-adrenergic receptor in the airways, such as salbutamol. Aminophylline and corticosteroids are also used.

Preventive therapy. The main preventive drugs are inhaled corticosteroids and sodium cromoglycate, although – due to the expense and limited availability – these are at present not widely used in developing countries. This treatment aims to decrease bronchial hyper-reactivity and airway inflammation.

It is important for young children to be given the correct method of administration of treatment. In general, inhaler devices cannot be used properly by children under the age of 5 years and so they are ineffective. However, the use of simple spacer devices are an effective and low-cost solution. Two puffs from the inhaler are discharged into the spacer chamber. The propellant gas is allowed to evaporate leaving very small particles of drug (less than 5 microns in size), which can be inhaled deep into the small airways of the child through a face mask or breathing tube. It is important that the drug reaches the small airways in order to be effective. This approach is just as effective in creating very small particle sizes as the use of a nebulizer.

Oxygen therapy. Children who have acute severe asthma and show signs of hypoxia may require oxygen therapy (see above).

Further reading

Campbell H, Byass P, Forgie IM, O'Neill KP, Lloyd-Evans N, Greenwood BM. Trial of co-trimoxazole versus procaine penicillin with ampicillin in treatment of community-acquired pneumonia in young Gambian children. *Lancet* 1988; **2**:1182–4.

Cherian T, John TJ, Simoes E, Steinhoff MC, John M. Evaluation of simple clinical signs for the diagnosis of acute lower respiratory tract infection. *Lancet* 1988; **16**:125–8.

Forgie IM, O'Neill KP, Lloyd-Evans N, Leinonen M, Campbell H, Whittle HC, Greenwood BM. Etiology of acute lower respiratory tract infections in Gambian children: I and II. Acute lower respiratory tract infection in children aged one to nine years presenting at the hospital. *Pediatr Infect Dis J* 1991; **10**:33–47.

Garenne M, Ronsmans C, Campbell H. The magnitude of mortality from acute respiratory infections in children under 5 years in developing countries. *World Health Stat Qly* 1992; **45**:180–91.

Harari M, Shann F, Spooner V, Meisner S, Carney M, de Campo J. Clinical signs of pneumonia in children. *Lancet* 1991; **338**:928–30.

Muhe L, Degefu H, Worku B, Oljira B, Mulholland EK. Comparison of nasal prongs with nasal catheters in the delivery of oxygen to children with hypoxia. *J Trop Pediatr* 1998; **44**:365–8.

Mulholland EK, Olinsky A, Shann FA. Clinical findings and severity of acute bronchiolitis. *Lancet* 1990; **335**:1259–61.

Mulholland EK, Ogunlesi OO, Adegbola RA, Weber M, Sam BE, Palmer A, Manary MJ, Secka O, Aidoo M, Hazlett D, Whittle H, Greenwood BM. Aetiology of serious infections in young Gambian infants. *Pediatr Infect Dis J* 1999; **18**(10 Suppl):S35–41.

Sazawal S, Black RE. Meta-analysis of intervention trials on case-management of pneumonia in community settings. *Lancet* 1992; **340**:528–33.

Shann F, Linnemann V, Gratten M. Serum concentrations of penicillin after intramuscular administration of procaine, benzyl, and benethamine penicillin in children with pneumonia. *J Pediatr* 1987; **110**:299–302.

Shann F, Barker J, Poore P. Chloramphenicol alone versus chloramphenicol plus penicillin for severe pneumonia in children. *Lancet* 1985; **2**:684–6.

Shann F. Aetiology of severe pneumonia in children in developing countries. *Pediatr Infect Dis J* 1986; **5**:247–52.

Usen S, Weber M, Mulholland K, Jaffar S, Oparaugo A, Omosigho C, Adegbola R, Greenwood B. Clinical predictors of hypoxaemia in Gambian children with acute lower respiratory tract infection: prospective cohort study. *Brit Med J* 1999; **318**:86–91.

Weber MW, Palmer A, Oparaugo A, Mulholland EK. Comparison of nasal prongs and nasopharyngeal catheter for the delivery of oxygen in children with hypoxemia because of a lower respiratory tract infection. *J Pediatr* 1995; **127**:378–83.

Wellcome Trust. *Topics in International Health. Acute respiratory infection*. ISBN 0 85 199 495 4. London 2001.

Williams BG, Gouws E, Boschi-Pinto C, Bryce J, Dye C. Estimates of world-wide distribution of child deaths from acute respiratory infections. *Lancet Infect Dis* 2002; **2**(1):25–32.

World Health Organization. *Treatment of tuberculosis: guidelines for national programmes*. 3rd edition. Geneva, 2003 (WHO/CDS/TB/2003.313).

World Health Organization: http://www.who.int/child-adolescent-health/data_stat.htm

Questions

3.1 Give definitions for the following conditions: chronic cough, pneumonia, severe pneumonia, very severe pneumonia

3.2 Describe the burden of disease due to respiratory infections in children in developing countries.

3.3 List the commonest causes of pneumonia and of wheeze in young children.

3.4 Describe an effective way to give oxygen therapy to children with hypoxia.

3.5 Describe the pathophysiology of pulmonary tuberculosis in children.

4. Diarrhoea

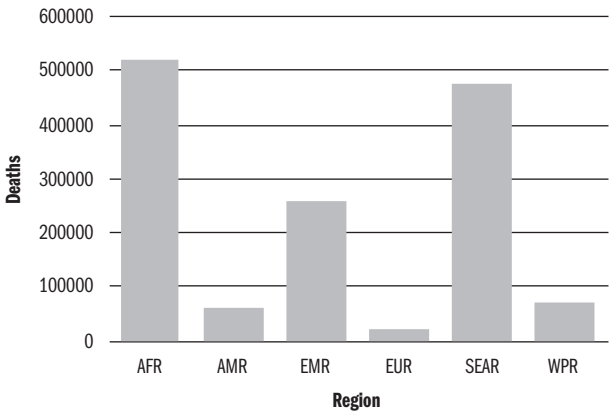
Diarrhoea is an important cause of illness and death in young children. Most deaths are due to dehydration resulting from acute diarrhoea and are treated by correct rehydration of the child. Persistent diarrhoea and dysentery are the other major types of diarrhoeal illness and require different management. Attention to the child's nutritional status and correct feeding are important aspects of the management of diarrhoea.

See also: ***Management of the Child with a Serious Infection or Severe Malnutrition*, Chapter 3, Page 45**

Definitions

Diarrhoea is the passage of loose or watery stools at least 3 times in 24 hours. In *acute diarrhoea* the illness lasts less than 14 days and the stools do not contain visible blood. *Dysentery* is diarrhoea with visible blood in the stool. *Persistent diarrhoea* is diarrhoea that begins acutely and continues for at least 14 days. These conditions differ with regard to pathogenesis, treatment, and risk of death.

Fig. 4-1 Number of deaths from diarrhoea in children under 5 years of age, estimated for the year 2000, by region (WHO)



AFR African Region, AMR American Region, EMR Eastern Mediterranean Region, EUR European Region, SEAR South-East Asian Region, WPR Western Pacific Region

Burden of diarrhoeal disease

In developing countries, children below the age of 3 years experience 3–10 episodes of diarrhoea each year and may spend 10–15% of their days with diarrhoea. About 1.3 million children aged between 2 months and 5 years die each year from diarrhoea. Acute diarrhoea is responsible for about 80% of diarrhoea episodes and 50% of deaths; dysentery causes about 10% of diarrhoea episodes and 15% of deaths; persistent diarrhoea causes about 10% of episodes and 35% of deaths.

Acute diarrhoea

Aetiology. Most episodes of acute diarrhoea are caused by intestinal infection; the most common causes are shown in **Table 4-1**.

Table 4-1 Important diarrhoea pathogens

Viruses	Rotavirus
Bacteria	Enterotoxigenic *E. coli*
	Enteropathogenic *E. coli*
	Enteroinvasive *E. coli* *
	Campylobacter jejuni *
	Shigella *
	Vibrio cholerae 01 and 0139
Protozoa	Cryptosporidium

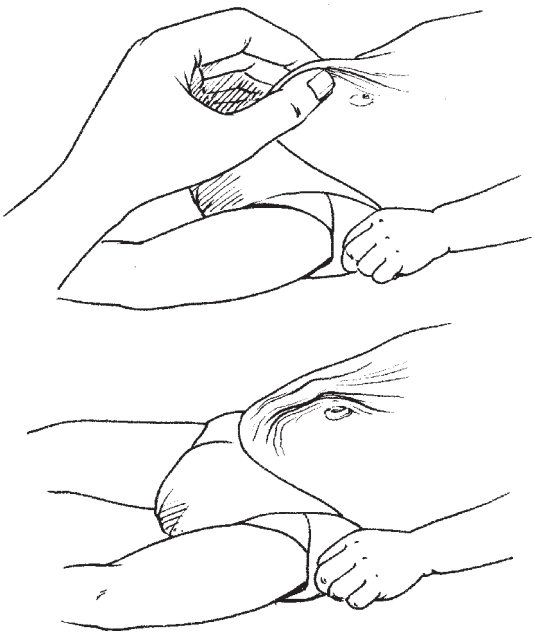
* Also causes dysentery

Pathophysiology. Diarrhoea causes increased loss of water and salts (sodium, potassium, chloride, and bicarbonate) in liquid stools (**Table 4-2**). This occurs because intestinal infection blocks the normal absorption of water and salts in the small intestine and causes them, instead, to be secreted into the intestine from the blood. These secretions cause the stools to become watery. The loss of water and salts produces isotonic dehydration with reduced blood volume, potassium depletion, and base-deficit acidosis.

Clinical features. A fluid deficit equal to 5% of the body weight (50 ml/kg) causes few physical signs or symptoms, other

than thirst. When losses equal 7–8% of body weight (70–80 ml/kg) the infants become restless and irritable, thirst increases, the eyes become sunken, and skin turgor is diminished. When losses approach 10% of body weight (100 ml/kg) dehydration is severe, the blood volume is reduced, and hypovolaemic shock develops. The signs and symptoms include lethargy or coma, very poor skin turgor, and inability to drink normally. Death from cardiovascular collapse occurs if the deficit of water and salts is not replaced rapidly.

Fig. 4-2 Decreased skin turgor.



Management. Treatment of diarrhoea has three components: (i) *rehydration therapy*, to correct dehydration and prevent its recurrence until diarrhoea stops; (ii) *feeding an appropriate diet*, to sustain nutrition, and (iii) in certain specific situations, use of an effective *antimicrobial* to shorten the illness.

Rehydration therapy. Rehydration therapy treats dehydration by restoring the deficit of water and electrolytes, and prevents its recurrence by fully replacing any further losses as they occur until the diarrhoea stops. Most patients can be treated by oral rehydration; when dehydration is severe, rapid intravenous rehydration is needed. In either case, the fluid given must contain sufficient salt and, if possible, potassium and bicarbonate (HCO_3) to correct the deficits and replace ongoing losses (**Table 4-2**).

(i) *IV rehydration*. When dehydration is *severe*, fluids must be given intravenously to correct shock rapidly. Ringer's lactate is the preferred solution because it provides the required salt and bicarbonate. If it is not available, normal saline may be given. Glucose solution (5% or 10%) is not effective because it contains no salt; it should not be given. The objective of initial IV therapy is rapidly to restore an effective blood volume. This can be done by infusing 30 ml/kg over one hour for infants, and within 30 minutes for all others. The remaining deficit of 70 ml/kg is replaced more slowly: 5 hours in infants and $2^1/_2$ hours in older children and adults.

Table 4-2 Average electrolyte content of diarrhoeal stool

	mmol/l			
	Sodium Na+	**Potassium K+**	**Chloride Cl^-**	**Bicarbonate HCO_3^-**
Cholera stool	101	27	92	32
Non-cholera stool	56	25	55	14
ORS solution*	75	20	65	30**
Ringer's lactate	130	4	109	28***
Normal saline	154	0	154	0

* Revised formulation recommended by WHO and UNICEF in 2002. Also contains glucose, 75 mmol/l.
** ORS contains 10 mmol/l sodium citrate, which is converted to HCO_3^-.
*** Lactate is converted to HCO_3^-.

(ii) *Oral rehydration*. When dehydration is not severe, or after shock has been corrected, rehydration may be carried out orally. ORS solution is designed efficiently to replace faecal losses. ORS solution does not cause the stool output to increase or decrease. It contains salt, potassium and citrate (which is metabolized to bicarbonate) to replace faecal losses, and glucose (**Table 4-2**). It is effective because glucose continues to be absorbed in the small intestine, even during diarrhoea, and this causes the simultaneous absorption of sodium, other electrolytes and water. A solution of salt and water without glucose, however, is not absorbed and will not treat or prevent dehydration. ORS solution containing 50 g/l of cooked rice powder in place of glucose is also effective because rice is converted to glucose in the small intestine, but it is not superior, compared to normal ORS, in the management of diarrhoea of children. Thirst is a valuable guide for oral rehydration therapy. Most children, when offered ORS solution, will drink the amount required to maintain adequate hydration and then stop. However, in children with severe malnutrition, rehydration needs to be more cautious (see Ch. 9)

Feeding. The objective of feeding during diarrhoea is to maintain or improve nutrition, prevent weight loss and sustain growth. This requires continued undiluted feeding. Following rehydration, most children regain their appetite and will eat.

During acute diarrhoea the digestion and absorption of nutrients is only slightly reduced. Overall, 80–90% of nutrients continue to be absorbed. Clinically important malabsorption of lactose is unusual and malabsorption of glucose is rare. A child given a nutritious diet during acute diarrhoea, including the child's usual milk, continues to grow. If food is reduced or withheld, growth will slow or stop, weight is lost, and the diarrhoea may be prolonged. Feeding during diarrhoea does not increase stool volume; continued breastfeeding actually reduces stool volume.

Use of antimicrobials. For children with acute watery diarrhoea, the cause cannot be determined when the child is seen and many episodes are due to agents for which antimicrobials are ineffective, such as rotavirus. Routine antimicrobials are not useful for children with acute watery diarrhoea and they should not be given.

When cholera is suspected, an oral antimicrobial to which local strains of *V. cholerae* are sensitive should be given. If the cause is cholera, diarrhoea will end within 48–72 hours. Failure to improve suggests resistance of *V. cholerae* to the antimicrobial that was given or the case is not cholera. Use of an antimicrobial does not reduce the need for careful rehydration therapy.

"Anti-diarrhoeal" drugs. Many products are claimed to reduce or stop diarrhoea. These include drugs, such as loperamide, diphenoxylate, tincture of opium, paregoric and codeine. Also included are adsorbents, such as kaolin, attapulgite, smectite and activated charcoal. Many have been studied in well designed clinical trials. None has proven effective and none should be given, either alone or in combination. Some are dangerous. Loperamide has caused fatal intestinal paralysis in infants.

Dysentery

Aetiology. Most episodes of dysentery are caused by intestinal infection with *Shigella*, *Campylobacter jejuni*, or enteroinvasive *E. coli*. *Entamoeba histolytica* is a rare cause in infants and children. The aetiology of dysentery can only be determined by stool culture and microscopic examination.

Pathophysiology. Bloody diarrhoea results when the intestinal mucosa is damaged by invasive bacteria or protozoa. Important complications of dysentery due to *Shigella* and enteroinvasive *E. coli* include haemolytic-uraemic syndrome, renal failure and rectal prolapse. Bacteria that cause dysentery may also cause acute non-bloody diarrhoea (**Table 4-1**). Children with dysentery may also develop dehydration due to excessive loss of water and electrolytes in liquid stool.

Clinical features. Stools contain visible blood; they may be large and watery or frequent and small. The child often appears listless, irritable and toxic, and refuses to eat. Fever, abdominal pain, and pain or crying when stool is passed are common. A single convulsion may occur, even without fever, and sometimes before diarrhoea begins.

Management. *Use of antimicrobials*. The most common cause of dysentery is *Shigella*. All children with dysentery should be treated for 5 days with an oral antimicrobial to which local strains of *Shigella* are known to be sensitive. If *Shigella* is the cause, and an effective oral antimicrobial is given, definite improvement will occur within 48 hours (less fever, less blood in the stool, better appetite, more active) and the child will recover fully within 4–5 days.

Other treatment. Rehydration and feeding guidelines follow the same principles as for acute watery diarrhoea (see above). Children with dysentery, however, often remain anorexic and must be encouraged to eat. "Anti-diarrhoeal" drugs should never be given.

Persistent diarrhoea

Aetiology. Persistent diarrhoea begins with intestinal infection (see **Table 4-1**), but malnutrition and malabsorption, often in combination, cause diarrhoea to be prolonged. If the stool contains visible blood, *Shigella* is the likely cause.

Pathophysiology. The underlying causes of persistent diarrhoea include protein-calorie malnutrition, concurrent extra-intestinal infection, such as pneumonia, and zinc deficiency, often in combination. The intestinal epithelium may be markedly abnormal, the villi are shortened or flat and the crypts are deepened. Disaccharidase enzymes are reduced and disaccharides may not be hydrolysed efficiently; clinically important malabsorption of lactose is common. When the diet contains animal milk, unabsorbed lactose is fermented in the ileum and colon, an osmotic effect causes diarrhoea to worsen, and stools become acidic. Mother's milk, however, is well tolerated. A few children with persistent diarrhoea also show malabsorption of starch and other complex carbohydrates. Reduced feeding, or continued feeding of an inappropriate diet, usually one containing animal milk, causes further weight loss. This is worsening malnutrition, and eventually causing death, usually due to severe infection.

Dehydration can occur, as described above for children with acute diarrhoea.

Clinical features. Liquid stools are often passed after eating, sometimes explosively. Occasionally stools contain visible blood. Weight loss is often evident and signs of malnutrition are often present; malnutrition varies in degree and may be severe. In children with marasmus, the loss of subcutaneous fat gives the appearance of decreased skin turgor and causes the eyes to appear sunken, making these sign useless for detecting dehydration.

Management. Treatment of persistent diarrhoea includes: (i) *rehydration therapy*, to correct dehydration and prevent its recurrence; (ii) *feeding an appropriate diet*, to sustain nutrition and support the recovery of intestinal function, and (iii) *treatment of serious infections* with an appropriate antimicrobial.

Rehydration therapy. This follows the principles described for acute watery diarrhoea (see above). If, however, the child is severely malnourished, there is an increased risk that IV or oral rehydration would cause heart failure. In such children, rehydration should be done slowly by mouth using the low-sodium solution, ReSoMal, as described in Chapter 9.

Feeding. Children with persistent diarrhoea require diets that provide full caloric intake, but with reduced lactose; some also require reduced starch. Supplemental multivitamins and minerals, including zinc, are also essential. Stabilization of weight followed by weight gain is the best indicator that the treatment is effective. Reduced stool output is not the objective, but diarrhoea will subside as the child improves.

Use of antimicrobials. Many children with persistent diarrhoea also have other infections, such as pneumonia, urinary infection or skin infection. In such children the persistent diarrhoea will not improve until the concomitant infection is also treated. A child with persistent diarrhoea should be examined carefully for these infections. When an extra-intestinal infection is diagnosed, an appropriate antimicrobial should be given. If the stool contains visible blood, an antimicrobial effective for *Shigella* should be given, as described above for children with dysentery. The routine use of antimicrobials for persistent diarrhoea, however, is not effective and these should not be given.

"Anti-diarrhoeal" drugs. None of these is effective and none should be given, either alone or in combination.

Further reading

Bennish ML, Salam MA, Haider R, Barza M. Therapy for shigellosis: II. Randomized, double-blind comparison of ciprofloxacin and ampicillin. *J Infect Dis* 1990; **162**:711–6.

Bhan MK, Bhandari N, Bhatnagar S, Bahl R. Epidemiology and management of persistent diarrhoea in children of developing countries. *Indian J Med Res* 1996; **104**:103–14.

Black RE, Brown KH, Becker S, Alim AR, Huq I. Longitudinal studies of infectious diseases and physical growth of children in rural Bangladesh. II. Incidence of diarrhoea and association with known pathogens. *Am J Epidemiol* 1982; **115**: 315–24.

Brown KH, Gastanaduy AS, Saavedra J et al. Effect of continued feeding on clinical and nutritional outcomes of acute diarrhoea in children. *J Paediatr* 1988; **112**: 191–200.

Heymann DL, Mbvundula M, Macheso A, McFarland DA, Hawkins RV. Oral rehydration therapy in Malawi: impact on the severity of disease and hospital admissions, treatment practices, and recurrent costs. *Bull WHO* 1990; **68**: 193–7.

International Working Group on Persistent Diarrhoea. Evaluation of an algorithm for the treatment of persistent diarrhoea: a multicentre study. *Bull WHO* 1996; **74**: 479–89.

Wellcome Trust. *Topics in International Health. Diarrhoeal Disease*. ISBN 0 85 199 246 3 London 1998

World Health Organization. *Management of severe malnutrition: a manual for physicians and other senior health workers*. Geneva, 1999.

World Health Organization. *The treatment of diarrhoea. A manual for physicians and other senior health workers*. Geneva, 1995 (WHO/CDR/95.3).

Questions

4.1 Give definitions for the following conditions: acute diarrhoea, persistent diarrhoea, and dysentery.

4.2 List the most important diarrhoeal pathogens in children.

4.3 Describe the clinical signs associated with increasing levels of fluid deficit.

4.4 Explain the mechanism by which oral rehydration works.

4.5. Describe the underlying causes and pathological findings in persistent diarrhoea.

5. Malaria

In areas where malaria transmission is intense, malaria is often the commonest cause of fever in young children. The disease may occur year-round or seasonally. Chloroquine used to be the first-line antimalarial in all countries, but resistance is now very common. A knowledge of local resistance patterns is therefore important.

See also: ***Management of the Child with a Serious Infection or Severe Malnutrition*, Chapter 3, Page 57**

Definition

Malaria is an acute febrile illness caused by mosquito-borne infection with one of the four species of *Plasmodium*: *P. falciparum*, *P. vivax*, *P. ovale* and *P. malariae*. Non-severe (uncomplicated) malaria is characterized by fever without another apparent cause and with a positive blood smear showing malaria parasites within red blood cells. Symptoms or signs that indicate *severe malaria* are given below. *P. falciparum* is responsible for most cases of severe malaria and almost all deaths due to malaria.

Fig. 5-1 Thin blood film showing malaria parasites. Ring-shaped parasites are seen within the red blood cells

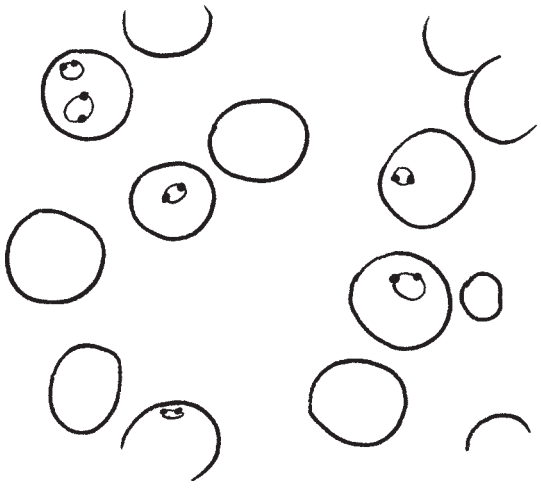
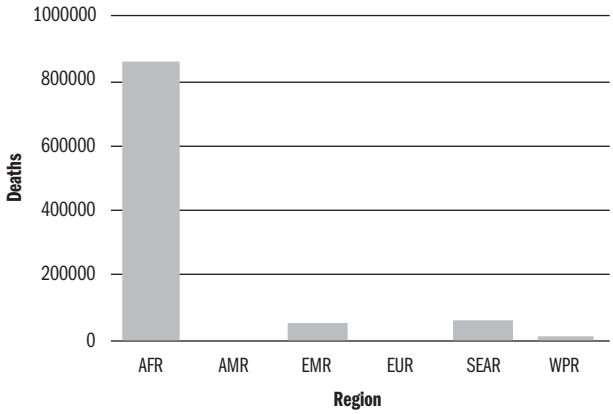


Fig. 5-2 Number of deaths from malaria in children under 5 years of age, estimated for the year 2000, by region (WHO)



AFR African Region, AMR American Region, EMR Eastern Mediterranean Region, EUR European Region, SEAR South-East Asian Region, WPR Western Pacific Region

Burden of disease

There are an estimated 300–500 million cases of malaria annually, and approximately 1.2 million deaths, among 2.3 billion persons at risk globally. Most cases of malaria occur in sub-Saharan Africa, where the estimated rate of infection is 700 cases/1000/year. In contrast, in Asia and South America the rate of infection is 4–5 cases/1000/year. Malaria may be seasonal or, in areas of intense transmission, occur all year round. It is the most common cause of fever in young children in areas where transmission is high. It is less common in older children and adults owing to acquired partial immunity. Non-immune travellers and recent immigrants to endemic areas are also vulnerable to severe infections. The use of bednets treated with a long-acting insecticide markedly reduces the risk of infection.

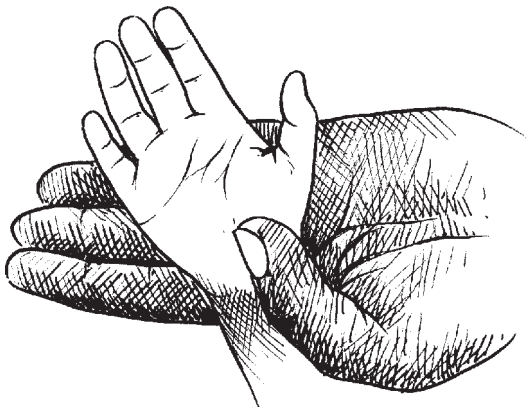
Severe malaria affects mainly children from a few months of age to 5 years, but may also occur in older children and adults in areas of low endemicity. WHO has estimated that 900,000 young children globally died from malaria in the year 2000. Over 90% of all cases of life-threatening malaria are in African children.

Pathophysiology

Severe falciparum malaria is characterized by sequestration of infected erythrocytes in the deep vascular beds of vital organs. In *cerebral malaria*, for example, red cell sequestration occurs predominantly in the brain. The mechanism for sequestration, why it occurs unevenly in vital organs, and how it contributes to symptoms of severe malaria are unclear. Various hypotheses propose that red cell sequestration is caused by mechanical obstruction of capillaries by poorly deformable red cells or by increased adherence of parasitized cells to the capillary endothelium, either of which eventually obstructs capillary blood flow and alters cerebral function. Another hypothesis is that sequestration leads to increased capillary permeability and cerebral oedema. Recent studies suggest that pro-inflammatory cytokines, in particular tumour necrosis factor, may mediate some of the pathological changes seen in malaria.

Anaemia is caused by disruption of red blood cells and by bone marrow suppression. Immune-mediated haemolysis may also contribute. *Blackwater fever* is the passing of dark brown urine (haemoglobinuria). This occurs when children with G6PD deficiency develop malaria and are treated with an oxidant drug, quinine or an artemesinin-derivative drug. It can also occur in severe malaria without G6PD deficiency.

Fig. 5-3 Palmar pallor: a sign of anaemia.



Clinical features

Non-severe malaria. Fever may be the only symptom. The probability that a febrile child has malaria is increased when the child has pallor or splenomegaly, cough is absent, or there is a history of previous malaria. These findings are especially helpful when assessing a febrile child where malaria transmission is low.

Severe malaria. The clinical manifestations of severe malaria in children include prostration (weakness with inability to sit up unassisted), altered consciousness (assessed by a coma score such the AVPU score), multiple convulsions, deep rapid breathing (evidence of acidosis), hypoglycaemia, circulatory collapse, pulmonary oedema, severe anaemia, abnormal bleeding, jaundice and haemoglobinuria. Severe anaemia appears to be most common in young children in areas of high endemicity; cerebral malaria occurs in older children in areas of moderate or seasonal endemicity.

Detection of parasitaemia. A diagnosis of malaria is confirmed by finding parasites in a thick blood film stained with Field's stain or Giemsa stain. A thin film is used to determine the species of *Plasmodium* involved.

The relationship of parasitaemia to severity of illness differs in various age groups and populations. In non-immune children a peripheral blood smear showing 5%, or more, of red cells containing parasites is compatible with a diagnosis of severe malaria. In endemic areas, however, where exposure is frequent, there is little correlation between parasite density and disease severity.

Other laboratory studies. Clinically suspected *anaemia* should be confirmed by measuring the haematocrit. *Hypoglycaemia* should be assessed by measuring the blood glucose level. A *lumbar puncture* to assess for bacterial meningitis should be done on all children who have altered consciousness or convulsions – unless there is a clear contraindication. If a lumbar puncture cannot be done, antibiotic therapy must be given to such children until meningitis is confirmed or excluded, or a course of treatment is completed

Management

Management includes treatment with antimalarial drugs to eliminate malarial parasites and treatment of complications, such as severe anaemia and convulsions . *It is essential that the choice of antimalarial drug follows the guidelines of the national malaria programme.*

Severe malaria. Antimalarial treatment must be started as soon as possible. If reading of the blood smear will be delayed, treatment should be started without that information. In endemic areas, when clinical findings suggest severe malaria, but no parasites are seen, it is still imperative to treat the child for malaria. Drugs for treatment of severe malaria include:

- *Quinine*. This is the drug of choice in Africa and most other countries, except the Amazon region and South-East Asia. It is given either IV or by intramuscular

injection. If an IV infusion cannot be carefully monitored, quinine should be diluted and given IM. Dilution improves the absorption of IM quinine, and makes it less painful and less likely to cause a sterile abscess. Severe toxicity is rare; it includes hypotension, cardiac dysrythmias, blindness, deafness and coma. Quinine stimulates pancreatic insulin secretion and can cause hypoglycaemia. Oral quinine is well absorbed, but bitter to taste; children may refuse to swallow it. Quinine should be given parenterally until the child can swallow oral medication. Then, either continue quinine orally for a total of 7 days or, in areas where sulfadoxine/pyrimethamine (SP) is still effective, give a single oral dose of SP.

- *IV artesunate, IM artemether, and IV quinidine*. Artesunate and its derivatives are trioxane compounds originally extracted from the qinghaosu plant (*Artemisia annua*). Artemesins have been used in China for many years. *Artemether* and *artesunate* are semi-synthetic derivatives with potent antimalarial activity. They are rapidly effective and have few side-effects. Parasite clearance times and fever reduction times are shorter than with other antimalarials. They are short-acting drugs and are often given in combination with other longer-acting agents (e.g. SP). Artemesinin can be given orally or rectally; artesunate is available in parenteral, oral and rectal form; artemether is usually given IM.
- *Chloroquine* is effective for severe malaria *only* in the limited areas where *P. falciparum* remains fully sensitive to it. These are: Haiti, parts of the Middle East, East Asia, North Africa, and Central America north of the Panama Canal. Absorption may be very rapid after intramuscular injection (5–20 minutes to reach peak serum levels); there is a potential risk for hypotension and cardiac arrhythmias.

  (It is important to remember that chloroquine and quinine doses refer to the amount of *base* drug to be given. The drugs are provided, however, as hydrochloride or sulfate salts (quinine), or the phosphate salt (chloroquine). This must be considered when determining the appropriate dosage.)

Non-severe malaria. Febrile children who are not vomiting and have none of the signs or symptoms of severe malaria should be treated at home with an oral antimalarial drug that is effective in the area. Children who are pale, but do not have severe anaemia, should be given oral iron for 3–4 months to correct the anaemia and replenish iron stores. Iron should not be given, however, to children in the acute phase of severe malnutrition because it increases the risk of serious bacterial infection. The child's mother should be advised to return at once if the child's condition deteriorates, or the child refuses to drink, vomits the medication, or becomes listless and weak. Some drugs for treatment of non-severe malaria are:

- *Chloroquine*. This is the most important of the 4-aminoquinoline compounds. It is rapidly absorbed after an oral dose and maintains curative levels for 6–10 days (terminal elimination time is 1–2 months). Minor side-effects such as nausea, headache and pruritus are fairly common. Resistance of *P. falciparum* to chloroquine is widespread. *P. vivax* remains sensitive in most areas.
- *Amodiaquine*. This is a 4-aminoquinoline that is given orally and is similar in structure to chloroquine. After absorption, it is rapidly metabolised to its active metabolite, desethylamodiaquine, which has a half-life of 18 hours. Adverse reactions to standard treatment courses are similar to those of chloroquine. Resistance to amodiaquine is parallel to that of chloroquine.
- *Sulfadoxine/pyrimethamine* (SP). This antagonises parasite folic acid synthesis. It is well absorbed orally with peak plasma concentrations in 2–6 hours and a sustained action (terminal elimination time is 90 hours). It is very well tolerated. Resistance of *P. falciparum* occurs in Brazil, Colombia and South-East Asia.

Treatment of complications. A child with severe malaria should be monitored for coma level, convulsions, increasing pallor and anaemia, and fluid input and output. An unconscious child needs careful nursing to avoid aspiration pneumonia. Treatment for complications is as follows:

- *Convulsions*. Emergency measures may be required to control convulsions. Hypoglycaemia must always be sought for, treated or excluded in any child with altered consciousness or who is convulsing. The ideal anticonvulsive drug of choice is rapid-acting with few side-effects. There is no drug that fulfils all these criteria and diazepam or paraldehyde are used for their rapid action, though diazepam may cause respiratory depression. Both drugs can be given rectally. Diazepam can be given intravenously and paraldehyde by IM injection. Paraldehyde should not be left in a plastic syringe for a long time as it can dissolve the plastic. However, in practice when paraldehyde is drawn up and used straightaway it causes no problems.
- *Anaemia*. Because of the increasing danger of blood transfusions, these should be given only to children with a haemoglobin concentration of 4–6 g/dl and one of the following clinical features: respiratory distress (acidosis), impaired consciousness, shock, heart failure or hyper-

parasitaemia (>10% of RBCs parasitized). If the haematocrit has fallen to 12% or less, or the haemoglobin is 4 g/dl or less, a blood transfusion should be given regardless of the patient's condition. Whole blood is transfused at 20 ml/kg (or packed cells at 10 ml/kg) over 3 to 4 hours. This may need to be repeated if the haematocrit is very low and the clinical signs are not improving. Furosemide is only needed if there is evidence of heart failure.

- *Fever.* Antipyretic treatment, e.g. with paracetamol, is advisable if a child's body temperature is >38.5 °C. Fanning and tepid sponging may be required if the temperature is >39 °C. Febrile children are miserable, difficult to encourage to drink and may convulse.
- *Fluids.* A child with fever, vomiting and poor oral intake may become dehydrated and require extra fluids. If fluid is given intravenously it is important to look for signs of fluid overload and check the urine output. The fluid of choice is 5% glucose infusion as hypoglycaemia is a known complication of malaria, quinine administration and poor feeding.
- *Acidosis.* Fluid replacement and correction of anaemia will usually correct any acidosis present.

Further reading

Barat LM, Bloland PB. Drug resistance among malaria and other parasites. *Infect Dis Clin North Am* 1997; **11**:969–87.

Berkley J, Mwarumba S, Bramham K, Lowe B, Marsh K. Bacteraemia complicating severe malaria in children. *Trans R Soc Trop Med Hyg* 1999; **93**:283–6.

Bloland PB, Boriga DA, Ruebush TK, McCormick JB, Roberts JM, Oloo AJ, Hawley W, Lal A, Nahlen B, Campbell CC. Longitudinal cohort study of the epidemiology of malaria infections in an area of intense malaria transmission II. Descriptive epidemiology of malaria infection and disease among children. *Am J Trop Med Hyg* 1999; **60**:641–8.

Bloland PB, Ettling M. Making malaria-treatment policy in the face of drug resistance. *Ann Trop Med Parasitol* 1999; **93**:5–23.

Breman JG. The ears of the hippopotamus: manifestations, determinants, and estimates of the malaria burden. *Am J Trop Med* 2001; **64**(1–2 Suppl):1–11.

English M, Punt J, Mwangi I, McHugh, Marsh K. Clinical overlap between malaria and severe pneumonia in Africa children in hospital. *Trans R Soc Trop Med Hyg* 1996; **90**:658–62.

Winstanley P. Modern chemotherapeutic options for malaria. *Lancet Infect Dis* 2001; **1**:242–50.

Wellcome Trust. *Topics in International Health. Malaria*. 2nd edition. ISBN 0 85 199 494 6 London 2000

World Health Organization. Severe falciparum malaria (Severe and complicated malaria, third edition). *Transactions of the Royal Society of Tropical Medicine and Hygiene* 2000; **94**(Suppl 1): 1–55.

Questions

5.1 Describe the burden of disease due to malaria in young children.

5.2 Explain the mechanism by which malaria can cause severe pathology in vital organs.

5.3 Describe the signs of severe malaria.

5.4 Discuss the role of chloroquine, quinine and artesunate in the management of malaria in children.

5.5 Describe the management of anaemia in severe malaria.

6. Meningitis

Meningitis is a less important cause of child mortality than the other conditions described in this manual but the risk of death in individual children with meningitis is very high. Management should be guided by an understanding of the common bacterial and viral causes of meningitis and, where possible, by the findings from a lumbar puncture.

See also: ***Management of the Child with a Serious Infection or Severe Malnutrition*, Chapter 5, Page 60**

Definition

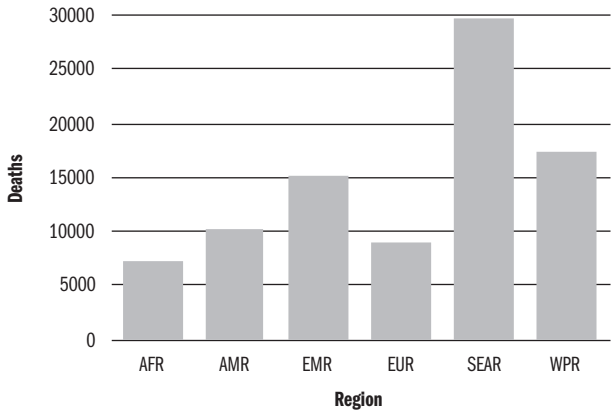
Meningitis is inflammation of the meninges. It is usually caused by viruses or bacteria, and occasionally fungi, that penetrate the blood-brain barrier and enter the cerebrospinal fluid (CSF) where immunological protection against infection is relatively poor. The three main types of meningitis due to infection are: *viral*, *pyogenic bacterial* and *tuberculous*.

Aetiology and burden of disease

Viral meningitis. This is a disease of children and young adults. It is due mostly to enteroviruses (ECHO, coxsackie and polio viruses), and occasionally others, such as mumps or herpes simplex virus. Spread is by the faecal-oral or respiratory routes. The global incidence of viral meningitis is not accurately reported. Poliomyelitis is a type of viral meningitis that can cause permanent paralysis; it has been almost globally eradicated through the use of polio vaccine.

Bacterial meningitis (pyogenic). This causes an estimated 77,000 deaths annually among 426,000 cases in children aged 0–4 years. Globally more than half of the episodes are caused by *H. influenzae* type b, with *S. pneumoniae* and *N. meningitidis* each causing about 11%. *N. meningitidis*, however, causes a much higher proportion of cases during periodic outbreaks in the "meningitis belt" of Africa, which stretches from Ethiopia to Senegal. Pneumococcal and *H. influenzae* meningitis occur most frequently in children aged 0–2 years; meningococcal meningitis occurs most frequently in older children and young adults. Mortality rates vary considerably depending on the quality of available care, but average 12–40% in developing countries compared to <5% in developed countries. Many survivors have significant sequelae, such as deafness or a motor handicap. The risk of mortality is greatest with pneumococcal meningitis.

Fig. 6-1 Number of deaths from meningitis in children under 5 years of age, estimated for the year 2000, by region (WHO)



AFR African Region, AMR American Region, EMR Eastern Mediterranean Region, EUR European Region, SEAR South-East Asian Region, WPR Western Pacific Region

Important causes of meningitis in neonates include *S. pneumoniae*, Gram-negative bacteria, such as *E. coli*, Klebsiella or Salmonella, and rarely group B streptococci or *Streptococcus faecalis*.

The bacteria that cause meningitis are spread by the respiratory route, colonizing the nasopharynx from where they have the potential to invade and cause meningitis while being protected by their polysaccharide capsules. Immunity develops naturally during childhood as a result of this colonization. Pneumococcal meningitis is often a complication of pneumococcal pneumonia, mastoiditis or skull fracture. Highly effective vaccines are available for *H. influenzae* type b, meningococcus types A and C, and for the most important pneumococcal serotypes.

Tuberculous meningitis. In children this is usually a complication of primary tuberculous pneumonia or miliary tuberculosis. The infection is often acquired from an older household member. Less than 1% of children with primary tuberculosis develop tuberculous meningitis. Neonatal immunization with BCG vaccine provides significant protection against tuberculous meningitis. Its value in preventing other forms of tuberculosis, however, is limited.

Clinical features

All three types of meningitis present with symptoms of headache, fever and neck stiffness. They differ, however, in other clinical features, treatment and outcome, including the risk of long-term disability or death.

Viral meningitis. This is an acute, self-limited illness characterized by fever, headache and neck stiffness. Some children develop a fine petechial or measles-like rash; photophobia also occurs. Except for polio, neurological damage is rare and recovery is usually complete in a few days without treatment.

Bacterial meningitis (pyogenic). This is a serious acute illness that begins with abrupt onset of fever, irritability, headache and neck stiffness, and progresses rapidly to include seizures, inability to feed, coma and, if untreated, death. Bacterial meningitis should be suspected in any infant or young child with unexplained fever and irritability, repeated seizures or altered consciousness. Important physical findings include neck stiffness, bulging fontanelle in infants, a petechial or purpuric rash, and cranial nerve paralysis. In neonates, the signs are less specific, resembling sepsis. Most frequent are fever or hypothermia, irritability, poor feeding or vomiting, seizures and respiratory distress or apnoeic episodes. A bulging fontanelle may occur; neck stiffness is unusual.

The clinical features of bacterial meningitis are similar to those of cerebral malaria. Moreover, the two conditions may be present at the same time. A child with signs suggesting meningitis should have a lumbar puncture even when a blood smear is positive for malaria parasites. If CSF findings suggest meningitis, treatment should be given for *both* conditions.

Tuberculous meningitis. TB meningitis develops over days or weeks, beginning with fever, headache, and irritability or listlessness. Stiff neck develops slowly and is followed by disorientation, coma and, if untreated, death. Paralysis of cranial nerves is common.

Fig. 6-2 Looking and feeling for neck stiffness in a child

Diagnosis

Analysis of CSF is the *only* way to confirm a diagnosis of meningitis. The findings also help to determine the likely cause and to guide treatment. Where possible, a lumbar puncture (LP) should be done on all children with suspected meningitis, unless there are signs of increased intracranial pressure or a skin infection at the LP site. **Table 6-1** shows typical findings in CSF in various types of meningitis.

In untreated bacterial meningitis a Gram stain of centrifuged CSF often reveals the causative agent: Gram-positive diplococci (*S. pneumoniae*), Gram-negative coccobacilli (*H. influenzae*), or Gram-negative diplococci (meningococci). In tuberculous meningitis, a Ziehl-Neelsen stain of centrifuged CSF may reveal rare acid-fast bacilli, but is often negative. Where culture is possible, CSF and blood should be cultured for bacteria, and CSF should be cultured for TB.

When an LP is not possible, treatment should be based on clinical suspicion, recognizing that early treatment of bacterial and tuberculous meningitis is essential to avoid serious complications or death.

Management

Viral meningitis. No specific treatment is required. Supportive care is sufficient. The illness is self-limited, usually resolving in a few days.

Bacterial meningitis (pyogenic). Treatment includes the prompt administration of antibiotics, management of complications and provision of supportive care.

Antibiotics. Antibiotics should be given parenterally and be effective against the most common causes of bacterial meningitis, the objective being to achieve bactericidal levels

Table 6-1 CSF findings in various types of meningitis

	Appearance	Cells	Protein	Glucose
Normal	Crystal clear	<6, all mononuclear cells; neonates: 1–3 polymorphonuclear cells	<40 mg/dl	50–80 mg/dl; >2/3 of blood glucos
Bacterial, untreated	Cloudy or purulent *	100s to 1000s, all polymorphonuclear cells	Increased, up to 100s mg/dl	Decreased or none
Bacterial, partly treated	Clear or slightly cloudy	Increased; mostly polymorphonuclear cells; later becoming mononuclear cells	Increased	Decreased or normal
Viral	Clear or slightly opalescent	0 to a few hundred mononuclear cells	20–125 mg/dl	Normal
Tuberculous	Straw coloured or slightly cloudy	250–500,mononuclear cells	45–500 mg/dl	Decreased or none

* May be clear during the first few hours of illness

in the CSF. If the causative organism is isolated from CSF or blood, antibiotic therapy should be based on its sensitivity determined in the laboratory.

In infants and young children, chloramphenicol plus ampicillin, or chloramphenicol plus benzylpenicillin, are usually effective against *S. pneumoniae*, *H. influenzae* and *N. meningitidis*. However, increasing resistance to these antibiotics, especially among *S. pneumoniae* and *H. influenzae*, may require the use of other agents. Third-generation cephalosporins, such as ceftriaxone or cefotaxime, are effective alternatives. Both are bactericidal and rapidly achieve high concentrations in the CSF. They are, however, more expensive and are not always available. It is important to know the local bacterial resistance patterns and to follow national guidelines as to which antibiotics to use.

In neonates, the most effective antibiotic is ceftriaxone or cefotaxime. Alternatives are gentamicin plus ampicillin or chlopramphenicol plus ampicillin. Both combinations, however, have shortcomings. Gentamicin does not penetrate well into CSF and chloramphenicol is ineffective for many *E. coli* and some *Salmonella*. Chloramphenicol should not be given to premature infants or during the first week of life.

Fluids. Some children with bacterial meningitis have hypovolaemia because of vomiting, poor feeding, or systemic sepsis; others have mild fluid overload due to increased antidiuretic hormone activity. Fluid restriction has been found to be harmful in children with meningitis. Children should therefore receive maintenance fluids, avoiding overhydration. Some of this can be as enteral feeds, when it is safe to do so.

Steroids. The role of steroids in bacterial meningitis remains controversial. Their routine use is not recommended at the present time.

Nutrition. It is often possible to start very small amounts of enteral nutrition from the beginning of treatment. If a child is unable to feed by mouth a nasogastric tube should be passed after 48 hours.

Fever. A temperature >39 °C should be treated with paracetamol. If the child is still febrile after 72 hours of antibiotic treatment a repeat LP may show that the infection has not responded to the treatment. Fever in a patient who is not improving, or who has focal neurological signs, may indicate a subdural effusion or cerebral abscess. It is important to exclude an infection at the drip site.

Convulsions. These must be controlled promptly as convulsions lead to increased cerebral oedema, acidosis and greatly increase the energy and oxygen requirements of the brain. A fast-acting, short-lasting anticonvulsant such as paraldehyde or diazepam should be used to control the convulsion. If the fits continue, a longer-acting drug such as phenobarbital is required.

Hypoglycaemia. This is common in any sick child who is not eating or drinking. It may be symptomless, or cause convulsions, irritability and peripheral vascular shutdown. This requires urgent correction with 10% glucose solution given intravenously. All children with an altered level of consciousness or convulsions should have a blood glucose checked to exclude hypoglycaemia.

Long-term sequelae:

- *Sensorineural hearing loss* occurs in up to 30% of survivors in developing countries. Hearing should be evaluated at discharge and after 1–3 months. As hearing loss is permanent, the parents of affected children should be counselled on how best to support their child.
- *Neurological sequelae* are common and vary in severity. Physiotherapy may be needed to aid motor function

and to advise about feeding the child. Epilepsy needs anticonvulsant therapy for control. Learning difficulties may require special assistance in school.

- *Hydrocephalus* may only be evident several weeks or months after the illness. It is important to measure the head circumference on discharge from hospital and on review after 1 month. If the head size is increasing the child should be referred for neurosurgical evaluation and advice.

Tuberculous meningitis. Treatment for tuberculous meningitis should follow national guidelines that are based on the sensitivity of isolates of *M. tuberculosis* in the country. Where resistance is not a problem, combination therapy with isoniazid, rifampicin and pyrazinamide is optimal. Pyrazinamide is given for two months, and the other two drugs for at least six months.

Where it is impossible to distinguish between partially treated bacterial meningitis and tuberculous meningitis, it is advisable to treat for both conditions and repeat a lumbar puncture 2 weeks later. In cases where the cause was bacterial, the CSF should have become virtually clear, while in TB meningitis no change will be noted.

HIV-related meningitis. Children who are immunosuppressed by the HI virus are at increased risk of serious bacterial infections, including meningitis caused by *S. pneumoniae* and *Salmonella* species. Cryptococcal meningitis, or another fungal meningitis, should raise suspicion of HIV infection. Such children may have severe headaches, but no neck stiffness. Treatment of bacterial meningitis in children with HIV is the same as for any other child, but relapses are more common and treatment time may need to be lengthened.

Further reading

Berkley JA, Mwangi I, Mellington F, Mwarumba S, Marsh K. Cerebral malaria versus bacterial meningitis in children with impaired consciousness. *Quart J Med* 1999; **92**:151–7.

Goetghebuer T, West TE, Wermenbol V, Cadbury AL, Milligan P, Lloyd-Evans N, Adegbola RA, Mulholland EK, Greenwood BM, Weber MW. Outcome of meningitis caused by *Streptococcus pneumoniae* and *Haemophilus influenzae* type b in children in The Gambia. *Trop Med Int Hlth* 2000; **5**:207–13.

Lehmann D, Yeka W, Rongap T, Javati A, Saleu G, Clegg A, Michael A, Lupiwa T, Omena M, Alpers MP. Etiology and clinical signs of bacterial meningitis in children admited to Garoka Base Hospital, Papua New Guinea, 1989–1992. *Ann Trop Paediatr* 1999; **19**:21–32.

Martin E, Hohl P, Guggi T, Kayser FH, Fernex M. Short course single daily ceftriaxone monotherapy for acute bacterial meningitis in children: results of a Swiss multicenter study. Part I: Clinical results. *Infection* 1990; **18**:70–7.

Molyneux EM, Walsh AL, Forsyth H, Tembo M, Mwenechanya J, Kayira K, Bwanaisa L, Njobvu A, Rogerson S, Malenga G. Dexamethasone treatment in childhood bacterial meningitis in Malawi: a randomised controlled trial. *Lancet* 2002; **360**:211–8.

Molyneux E, Walsh A, Phiri A, Molyneux M. Acute bacterial meningitis in children admitted to Queen Elizabeth Central Hopsital, Balntyre, Malawi in 1996–97. *Trop Med Int Hlth* 1998: **3**:610–8.

Salih MA, Khaleefa OH, Bushara M, Taha ZB, Musa ZA, Kamil I, Hofvander Y, Olcen P. Long-term sequelae of childhood acute bacterial meningitis in a developing country. A study from the Sudan. *Scand J Infect Dis* 1991; **23**:175–82.

Thwaites G, Chau TTH, Mai NTH, Drobniewski F, McAdam K, Farrar J. Tuberculous meningitis. *J Neurol Neurosurg Psychiatry* 2000; **68**:289–99.

Weber MW, Herman J, Usen S, Jaffar S, Oparaugo A, Omosigho C et al. Clinical predictors of bacterial meningitis in infants and young children in The Gambia. *Trop Med Int Hlth* 2002; **7**(9):722-731.

Questions

6.1 Describe the three main types of meningitis and the major microbiological causes in each category.

6.2 Describe the main CSF findings (appearance, cell count, protein content and glucose level) in each type of meningitis.

6.3 Explain why correct fluid management is important in children with meningitis and describe the main elements of fluid management.

6.4 List three long-term sequelae of meningitis and describe how they may present.

6.5 List the main causes of meningitis in children with HIV infection and describe how the treatment differs in these children.

7. Measles

Measles is a highly contagious viral disease with serious complications. It has been a major cause of death globally in young children although this has been reduced due to measles vaccination delivered through national child immunization programmes. A knowledge of how to recognize and treat the complications of measles is important for the successful management of children with measles. Vitamin A is now an important aspect of the treatment.

See also: ***Management of the Child with a Serious Infection or Severe Malnutrition*****, Chapter 5, Page 64**

Definition

Measles is a highly communicable acute infection of young children that is caused by an RNA paramyxovirus. The disease is characterized by a generalized maculo-papular erythematous rash, high fever and coryza, conjunctivitis, cough and stomatitis. Life-long immunity follows recovery.

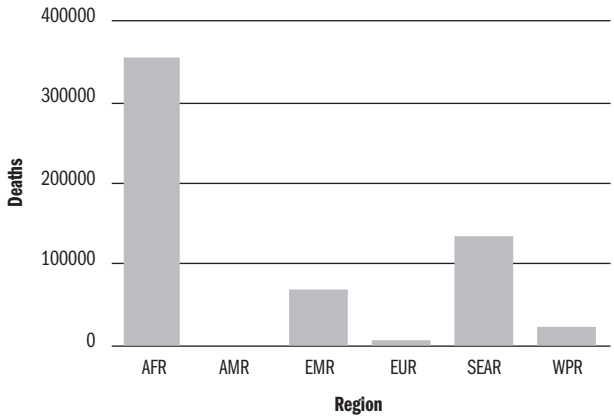
Burden of disease

Despite the availability of a cheap and effective vaccine, measles remains a problem in many developing countries. It is a leading childhood killer, accounting for more deaths than any other vaccine-preventable disease. WHO has estimated that in 2000 there were approximately 587,000 deaths from measles, mostly occurring in Africa and Asia. Most deaths result from complications such as pneumonia, diarrhoea and malnutrition. In addition, countless thousands are disabled by chronic lung disease, malnutrition, blindness, deafness, and recurrent infections that follow measles.

Pathophysiology

Measles virus causes immune suppression, epithelial damage and vitamin A deficiency. These result in increased susceptibility to secondary viral and bacterial infections. The epithelium of the conjunctiva, respiratory and gastrointestinal tracts are particularly vulnerable; diarrhoea, pneumonia and eye damage are, thus, major complications. The reason for the dramatic decline in serum vitamin A levels during acute measles is not fully understood. Contributing factors are likely to include increases in breakdown, use and urinary excretion of the vitamin, and altered homoeostasis of retinol-binding protein.

Fig. 7-1 Number of deaths from measles in children under 5 years of age, estimated for the year 2000, by region (WHO)



AFR African Region, AMR American Region, EMR Eastern Mediterranean Region, EUR European Region, SEAR South-East Asian Region, WPR Western Pacific Region

Diagnosis and clinical features

Measles virus is transmitted by droplets from, or direct contact with, an infected individual. The incubation period is 8–12 days. The illness starts with a prodrome characterized by fever, cough, conjunctivitis and coryza that lasts for 2–4 days. Grey-white Koplik spots are often visible on the buccal mucosa during this period; they are diagnostic for measles. The maculopapular erythematous rash then appears, starting in the head and neck, and spreading to the face and the rest of the body. The child

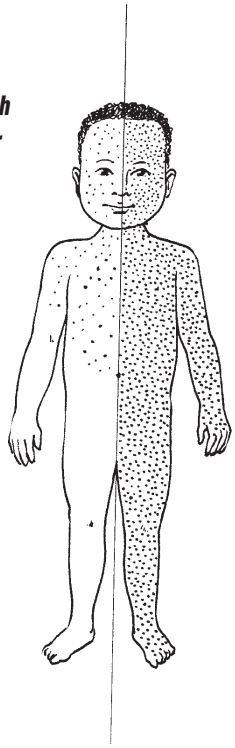


Fig. 7-2 Distribution of the measles rash. The left side of the drawing shows the early rash covering the head and upper part of the body, the right side shows the later rash covering the whole body

has a high fever, is irritable, photophobic and usually has conjunctivitis, cough and stomatitis. The illness lasts for about 4–5 days. The rash then darkens or fades. Desquamation often follows and may persist for weeks.

The diagnosis of measles is based on the typical progression of these signs and symptoms and is seldom difficult. The rash of measles can sometimes be confused with rubella, infectious mononucleosis, scarlet fever, Rocky Mountain spotted fever and enteroviral infections.

Significant complications occur in 15–30% of all cases. These include pneumonia, diarrhoea, malnutrition, otitis media, severe mouth ulcers and severe conjunctivitis. The case fatality rate of children hospitalized with measles varies between 5% and 25%. Measles greatly accelerates the development of xerophthalmia (dryness of the conjunctiva and cornea) in children already deficient in vitamin A. If treatment with vitamin A is not given promptly, blindness may result and mortality is extremely high. Uncommon complications include encephalitis, myocarditis, nephritis and pneumothorax .

Children who have had measles have significantly increased morbidity and mortality in the ensuing months. This is likely to be due to prolonged immunosuppression and is characterized by failure to thrive, and persistent or repeated episodes of pneumonia, diarrhoea and dysentery, and other recurrent infections. Vitamin A deficiency, if present, will aggravate these problems.

Management

There is no specific therapy for measles. Nevertheless, its adverse consequences can be reduced by early treatment with vitamin A, by maintaining nutrition, and by early recognition and careful treatment of complications in accordance with standard guidelines.

Vitamin A. All children with measles should receive vitamin A, unless supplemental vitamin A was given in the past month. The dosage and schedule recommended by WHO are shown in **Table 7-1**.

*Table 7-1 Vitamin A dosage for measles**

Age	Immediately	Next day
Up to 6 months	50,000 IU	50,000 IU
6–11 months	100,000 IU	100,000 IU
12 months or older	200,000 IU	200,000 IU

* If the child is severely malnourished, or has signs of xerophthalmia, give a third dose after 2–4 weeks.

Nutritional support. Measles is a major catabolic event that can cause significant weight loss and accelerate the development of malnutrition. To avoid these effects, adequate nutritional intake must be sustained. The mother should be encouraged to feed the child, even if diarrhoea is present. Breastfeeding must be maintained. For children who are not breastfed, the energy content of the food should be increased by adding a teaspoon of vegetable oil and a teaspoon of sugar to the milk or cereal. The child should be given more fluids than usual to prevent dehydration. If dehydration develops, oral rehydration should be given (see Chapter 4). A multi-vitamin syrup should be given, if available. If a child refuses to take feeds, check carefully for mouth ulcers and treat any that are found. If necessary, give liquid feeds and fluids through a nasogastric tube.

Treatment of complications. Children should be checked carefully and frequently for the complications listed above. This is especially important for children who remain febrile, refuse to eat or continue to lose weight. Treatment of complications should follow standard guidelines. Antibiotics should only be given if there is a specific indication such as pneumonia, otitis media, dysentery or purulent conjunctivitis. There is no evidence that prophylactic antibiotics are of benefit. The indiscriminate use of antibiotics may lead to complications such as antibiotic-

associated diarrhoea, severe drug reactions, and the emergence of drug-resistant organisms.

Since failure to thrive and recurrent infections are common in the months following measles, it is essential that children be closely monitored. Regular clinic attendance is recommended for at least six months following recovery from acute measles.

Measles in HIV-infected children

In children with HIV the prodrome and rash of measles are usually typical. There is, however, a high rate of complications, including giant-cell pneumonia and encephalitis. Measles infection may persist or recur, and mortality rates frequently exceed 50%.

Prevention. Measles vaccine is a live attenuated vaccine. WHO recommends a single dose at 9 months in developing countries. Children who are HIV-positive should be vaccinated. However, those with severe manifestations of AIDS or who are seriously immunocompromised for other reasons should not be vaccinated. High coverage (>85–90%) with the vaccine is required to provide adequate herd immunity and to avoid the epidemics of measles that occur typically every 3–5 years in some developing countries.

Further reading

Coovadia HM, Wesley A, Brain P. Immunological events in acute measles influencing outcome. *Arch Dis Child* 1978; **53**: 861–7.

Hussey GD, Clements CJ. Clinical problems in measles case management. *Ann Trop Paeds* 1996; **16**:307–17.

Hussey GD, Klein M. A randomized, controlled trial of vitamin A in children with severe measles. *N Engl J Med* 1990; **323**:160–4.

Hussey GD. Managing measles. Integrated management reduces disease severity. *Br Med J* 1997; **314**:316–7.

Markowitz LE, Nzilambi N, Driskell WJ et al. Vitamin A levels and mortality among hospitalized measles patients. *J Trop Paediatr* 1989; **35**:109–12.

Morley D. Severe measles in the tropics I and II. *Br Med J* 1969; **1**: 297–300; 363–5.

Moss WJ, Monze M, Ryon JJ, Quinn TC, Griffin DE, Cutts F. Prospective study of measles in hospitalized, human immunodeficiency virus (HIV)-infected and HIV-uninfected children in Zambia. *Clin Infect Dis* 2002 15; **35**:189–96.

Palumbo P, Hoyt L, Demasio K, Oleske J, Connor E. Population-based study of measles and measles immunization in human immunodeficiency virus-infected children. *Pediatr Infect Dis* 1992; **11**: 1008–14.

Tomkins A, Hussey G. Vitamin A, immunity and infection. *Nutr Res Rev* 1989; **2**:17–28.

Questions

7.1 Describe the pathological effects of measles virus infection.

7.2 Describe the time sequence of the course of infection – from infection with the virus through to the disappearance of the rash.

7.3 List the main complications of measles.

7.4 Describe the key elements of nutritional support in the management of children with measles.

7.5 Describe the role of antibiotics in the management of the child with measles and explain the rationale for this approach.

8. Infections in young infants

Infections are an important cause of both morbidity and mortality in young infants up to 2 months of age. Infections are especially important because the deaths they cause are potentially preventable, either by prophylaxis (e.g. tetanus immunization during pregnancy) or early diagnosis and appropriate treatment (e.g. for pneumonia or sepsis). Some infections, such as ophthalmia neonatarum, cause little mortality, but have substantial morbidity and serious long-term sequelae.

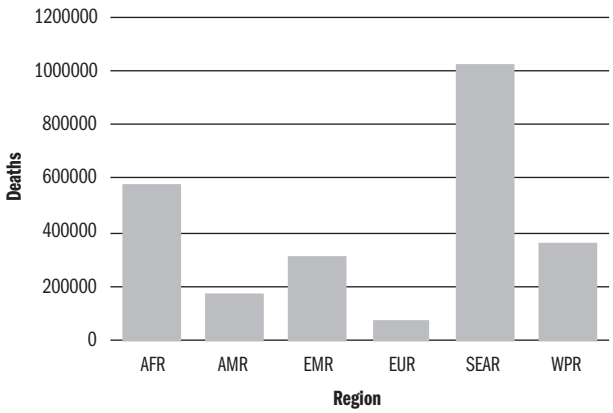
See also: ***Management of the Child with a Serious Infection or Severe Malnutrition*, Chapter 6, Page 74**

Burden of disease

According to WHO estimates, conditions arising in the neonatal period accounted for 2.5 million deaths in the year 2000, or about 23% of all child deaths. Infections in the neonatal period are estimated to cause about 42% of deaths that occur from birth up to 28 days of age in developing countries, and an even higher proportion of those in the second month of life. The most important, potentially lethal, infections during the first 28 days of life are pneumonia (20% of neonatal deaths), septicaemia and meningitis (9%), tetanus (9%) and diarrhoea (4%). With the exception of tetanus, which occurs mostly during the first 2 weeks of life, the same infections are also the most important causes of mortality for infants aged 28 days to 2 months. Other important infections during the first month involve the umbilical stump and skin (omphalitis and pustular dermatitis) and eyes (ophthalmia neonatarum).

These infections are acquired either from a colonized maternal genital tract during labour and delivery (typically with onset during the first week of life) or from contact after birth with organisms in the newborn's environment (typically with onset after the first week of life). The estimated number of episodes and proportion of all neonatal deaths for the most severe of these infections are shown in **Table 8-1**.

Fig. 8-1 Number of deaths from perinatal causes in children under 5 years of age, estimated for the year 2000, by region (WHO)



AFR African Region, AMR American Region, EMR Eastern Mediterranean Region, EUR European Region, SEAR South-East Asian Region, WPR Western Pacific Region

Table 8-1 Neonatal infections and deaths in developing countries
(Stoll 1997)

Type of infection	Number of neonatal episodes	Proportion of all neonatal deaths (%)
Pneumonia	2,650,000	20
Sepsis/meningitis	876,000	8.8
Tetanus	400,000	8.5
Diarrhoea	25,000,000	3.7

Definitions

In this chapter "young infant" refers to infants aged up to 2 months. The diagnoses listed in **Table 8-1** are based on clinical features that are described below. Among these, pneumonia, septicaemia and meningitis are often indistinguishable clinically and may occur together as part of a single acute illness. For this reason they are considered together as *serious bacterial infection*. Other serious infections,

for which accurate clinical diagnosis is usually possible, are considered separately.

Serious bacterial infection

Clinical presentation. The clinical features of young infants with septicaemia, meningitis or pneumonia are often non-specific and overlapping. Typically, signs and symptoms develop within a few hours, or 1–2 days, and may include any of the following:

- Temperature >38 °C or <35.5 °C
- Lack of spontaneous movement
- Altered mental state (agitation, lethargy, or coma)
- Poor feeding
- Respiratory rate >60 breaths/minute
- Lower chest wall indrawing
- Grunting
- Cyanosis
- A history of convulsions
- A bulging fontanelle
- Slow digital capillary refill.

Infants with more than one of these signs are likely to have septicaemia or meningitis and should be treated immediately. When only a single sign is present, e.g. fever or feeding poorly, serious bacterial infection is possible, but much less certain. For such infants it may be safest to treat promptly, even though some will be treated unnecessarily. The alternative is to observe the infant carefully for the development of additional signs, or improvement, without giving treatment. This, however, carries a risk of rapid deterioration and death. In no instance should a young infant with one of these signs be sent home before it is certain the infant has improved.

Bacteriology. A multicountry study has provided valuable information on the most frequent causes of serious bacterial infections during the first two months of life. These are summarized in **Table 8-2** and **Table 8-3**.

Diagnosis and management. There is real urgency in recognizing illness in a newborn or young infant and ensuring that the child has access to trained health care workers for assessment and, most importantly, treatment with life-saving antibiotics. Such care is most likely to be available at a well supplied health centre or hospital. It has also been shown that village health workers, specially trained in home-based neonatal care, can provide treatment and reduce deaths from serious bacterial infection in neonates.

Parenteral antibiotics. These should be given as soon as a diagnosis of serious bacterial infection is suspected. Give a combination of ampicillin (or benzyl penicillin) plus gentamicin (or kanamycin). If meningitis is suspected or diagnosed, treat as described in Chapter 6. If blood or CSF cultures are done, the initial antibiotic therapy may need to be modified when the results are available.

Table 8-2 Bacteria isolated from blood of infants with sepsis
(WHO Young Infants Study Group 1999)

	Age 0–7 days n=25	Age 8–60 days n=104
S. aureus	20%	22%
Str. pneumoniae	8%	19%
Str. pyogenes	12%	17%
E. coli	24%	12%
Salmonella spp.	4%	12%
H. influenzae type b	–	4%
Enterobacter spp.	12%	4%
Acinitobacter spp.	4%	3%
Klebsiella spp.	–	3%

Table 8-3 Bacteria isolated from CSF of infants with sepsis
(WHO Young Infants Study Group 1999)

	Age 0–7 days n=6	Age 8–60 days n=28
Str. pneumoniae	–	53%
Str. pyogenes	16%	7%
E. coli	16%	14%
Enterobacter spp.	32%	4%

Supportive care. This includes attention to respiratory status (airway and oxygen, if needed and available), proper maintenance of body temperature (to avoid hypo- or hyperthermia), attention to fluid status, continued nutrition, and prevention or treatment of hypoglycaemia.

- *Oxygen*, if available, should be given to any young infant with respiratory distress or measured oxygen desaturation. Measurement of arterial oxygen saturation by pulse oximeter, if possible, should be used to monitor the adequacy of oxygen therapy.
- *Hypothermia* is dangerous for newborns and young infants, and is frequently a sign of serious infection. It

is important that the infant be kept warm. Using wraps, a cap, and keeping the room warm are helpful. "Kangaroo care", i.e. keeping the infant in close skin to skin contact with the mother at all times, may also help. If the infant is febrile, unwrap or undress the child; do not give antipyretic drugs.

- *Feeding and fluids*. Frequent breastfeeding should be encouraged, if the infant is able to suck. This will maintain fluid and electrolyte balance, prevent hypoglycaemia, and provide needed nutrition. If the infant cannot suck, give expressed breast milk by nasogastric tube. If this is not possible, and the infant can swallow, try giving expressed breast milk by mouth with a cup or spoon, taking care to avoid aspiration.

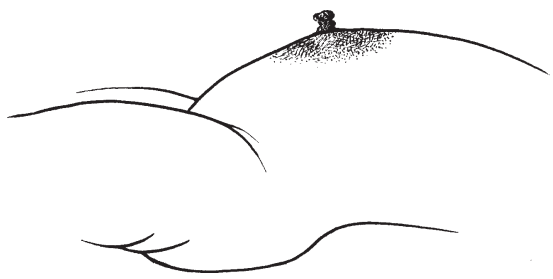
Diarrhoea

When diarrhoea occurs in young infants the risk of death is high. Exclusive breastfeeding for the first six months provides substantial protection against diarrhoea and diarrhoea-associated mortality. Dehydration is the immediate threat to a young infant with acute diarrhoea. Treatment should include continued breastfeeding and oral rehydration with ORS solution (see Chapter 4).

Omphalitis and skin infections

Omphalitis, infection of the umbilical stump, is a problem where aseptic cutting of the umbilical cord and hygienic care of the remaining stump are not universal practices. The necrotic umbilical stump is a particularly good medium for bacterial growth. Although inflammation that is immediately adjacent to the umbilical stump is not life-threatening, the close proximity to the umbilical vessels gives bacteria potential access to the bloodstream, which increases the risk of bacterial septicaemia.

Fig. 8-2 Peri-umbilical flare in umbilical sepsis: the inflammation extends beyond the umbilicus to the abdominal wall



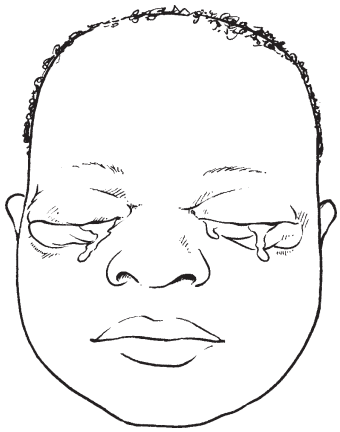
Peri-umbilical skin redness that does not extend to the abdominal wall should be treated with antiseptics applied to the affected area and with an oral antimicrobial, such as cotrimoxazole. The infant with local skin infection can be treated at home. If, however, redness extends to the abdominal wall, induration develops, the umbilicus drains pus, or the infant develops signs of serious bacterial infection, as described above; parenteral antibiotics and treatment in hospital are essential.

Skin infections include pustules and furuncles. When these are few, isolated and with little or no surrounding redness, they may be treated by washing carefully with soap and water, applying a local antiseptic, and giving an oral antimicrobial, such as cotrimoxazole. If, however, there are numerous pustules or furuncles, or they coalesce to form expanding lesions, treatment should be given in hospital with parenteral antibiotics effective against *S. aureus*, *Str. pyogenes* and Gram-negative pathogens, such as the combination of cloxacillin and gentamicin.

Ophthalmia neonatorum

Ophthalmia neonatorum is defined as a purulent conjunctivitis in the first 28 days of life. The most important causes are *Neisseria gonorrhoeae*, *Chlamydia trachomatis* and *Staphylococcus aureus*. Infection with *N. gonorrhoeae* and *C. trachomatis* is acquired from an infected mother during passage of the infant through the birth canal. The risk of neonatal infection is related directly to the prevalence of maternal infection in the area and inversely to the frequency of antimicrobial eye prophylaxis. Gonococcal ophthalmia often develops soon after birth, is more severe than chlamydial ophthalmia, and, if untreated, can lead to corneal scarring and blindness. Because it is a preventable cause of blindness, it is critical that gonococcal ophthalmia be diagnosed and treated promptly. In contrast, chlamydial conjunctivitis rarely causes permanent eye damage. It does, however, correlate with the occurrence of nasopharyngeal colonization with this agent and with an increased risk of chlamydial pneumonia in the first few months of life. Gram stain of conjunctival pus may reveal Gram-negative diplococci (*N. gonorrhoeae*) or Gram-positive cocci (*S. aureus*). In areas where gonorrhoea is prevalent and where laboratory diagnosis (eye culture and/or Gram stain) is not possible, all neonates with ophthalmia should be treated for presumed gonococcal infection, e.g. with parenteral ceftriaxone (or kanamycin or spectinomycin), depending on the resistance pattern of gonococci and the recommended treatment of gonorrhoea in STD clinics in the country. .

Fig. 8-3 Ophthalmia neonatorum



Ophthalmia neonatorum due to gonorrhoea and chlamydia can be controlled by preventing sexually transmitted diseases (STDs) in the mother, antenatal care with screening for, and treatment of, STDs in the mother, or routine eye prophylaxis at birth. A combination of these measures is best. Eye prophylaxis is a cost-effective and feasible strategy, especially in areas where STDs are prevalent. This involves cleaning the eyelids and instilling an antimicrobial agent into the conjunctival sac of all neonates as a routine measure promptly after birth.

Further reading

Bang AT, Bang RA, Baitule SB et al. Effect of home-based neonatal care and management of sepsis on neonatal mortality: field trial in rural India. *Lancet* 1990; **354**:1955–61.

Dragovich D, Tamburlini G, Alisjahbana A et al. Thermal control of the newborn: knowledge and practice of health professional in seven countries. *Acta Paed* 1997; **86**:645–50.

Dragovich D, Tamburlini G, Alisjahbana A et al. Thermal control of the newborn: knowledge and practice of health professional in seven countries. *Acta Paed* 1997; **86**:645–60.

Garner P, Lai D, Baea M et al. Avoiding neonatal death: an intervention study of umbilical cord care. *J Trop Pediatr* 1994; **40**:24–8.

Haider R, Kabir I, Fuchs GJ et al. Neonatal diarrhoea in a diarrhoea treatment center in Bangladesh: clinical presentation, breast-feeding management and outcome. *Indian Paediatr* 2000; **37**:37–43.

Laga M, Meheus A, Piot P. Epidemiology and control of gonococcal ophthalmia neonatorum. *Bull WHO* 1989; **67**:471–8.

Mason WH, Andrews R, Ross LA et al. Omphalitis in the newborn infant. *Pediatr Infect Dis J* 1989; **8**:521–5.

Stoll BJ. The global impact of neonatal infection. *Clinics Perinatol* 1997; **24**:1–21.

Victora CG, Smith PG, Vaughan JP et al. Infant feeding and deaths due to diarrhoea: a case-control study. *Am J Epidemiol* 1989; **129**:1032–41.

Weber MW, Carlin JB, Gatchalian S, Lehmann D, Muhe L, Mulholland EK et al. Predictors of neonatal sepsis in developing countries. *Pediatr Infect Dis J* 2003; **22**(8):711-717.

Whitcher JP, Srinivasan M, Upadhyay MP. Corneal blindness: a global perspective. *Bull WHO* 2001; **79**:214–21.

WHO Young Infants Study Group. Aetiology and clinical signs of serious infections in young infants in developing countries: a WHO collaborative study. *Pediatr Infect Dis J* 1999; Suppl **18**:S4–S69.

Questions

8.1 List the most important neonatal infections, in order of their importance, as causes of death in developing countries.

8.2 List the most important bacterial causes of sepsis in the young infant.

8.3 Describe the three main aspects of supportive care in the sick young infant.

8.4 Describe the management of skin infections in the young infant.

8.5 List the main microbiological causes of and risk factors for ophthalmia neonatorum.

9. Severe Malnutrition

Children with severe malnutrition are at risk of several life-threatening problems like hypoglycaemia, hypothermia, serious infection, and severe electrolyte disturbances. Because of this vulnerability, they need careful assessment, special treatment and management, with regular feeding and monitoring. Their treatment in hospital should be well organized and given by specially trained staff.

See also: ***Management of the Child with a Serious Infection or Severe Malnutrition*****, Chapter 7, Page 80**

Introduction

Many severely malnourished children die from incorrect management. As a result, mortality rates as high as 40–50% are found in many hospitals. With correct treatment, however, mortality rates can be reduced substantially. Therefore, doctors and nurses must be taught best practice during their training, and improve their practice during their professional life. Lives can be saved when treatment guidelines are followed, and the dramatic and rapid transformations of severely malnourished children can be one of the great satisfactions of professional life.

Burden of disease

WHO has estimated that 54% of child deaths are associated with malnutrition.

Definition

Severe malnutrition is defined as the presence of:

- *Severe wasting* (<70% of the median weight-for-height or <–3SD), and/or
- *Oedema of both feet*.

Severe malnutrition includes *marasmus*, marasmic-kwashiorkor and kwashiorkor. Children with *marasmus* have severe wasting without oedema; those with marasmic-*kwashiorkor* have severe wasting + oedema; those with kwashiorkor have oedema. Low weight-for-age is not a criterion for hospital treatment by itself; it could lead to stunted children being hospitalized, which would be inappropriate, as the short stature is not going to be improved in the short term.

Fig. 9-1 Child with marasmus

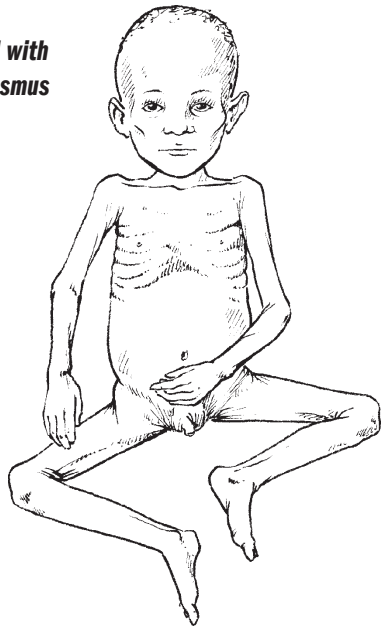
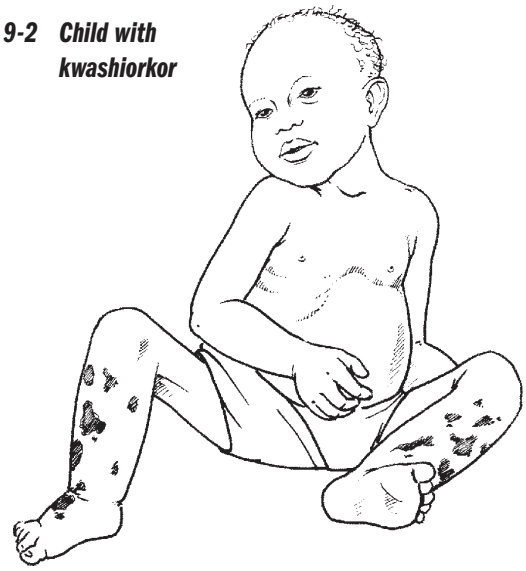


Fig. 9-2 Child with kwashiorkor



Clinical presentation

Severe malnutrition may occur at any age but is most common between 6 and 30 months, except during famine. It often follows a severe illness such as measles or persistent diarrhoea, or repeated infections.

Severe wasting is due to loss of fat and skeletal muscle and is most visible on the buttocks, thighs and upper arms, where the skin hangs loose in folds. The ribs and shoulder blades are clearly visible. The eyes may appear sunken due to wasting of the tissues behind the eye. The wizened face gives an 'old person' appearance. Weakened abdominal muscles and intestinal gas from bacterial overgrowth in the small bowel can lead to a distended belly. Wasted children are often anxious and irritable, and cry easily. Tears, however, may be absent due to atrophy of the lacrymal glands.

In **oedematous malnutrition**, the oedema usually appears first in the feet, and then the lower legs. Gross oedema includes the arms and face and can develop quickly. Skin changes include abnormally dark, crackled, peeling patches (called 'flaky paint' dermatosis) with pale skin underneath that is easily damaged and infected. Hair becomes brittle, is easily pulled out, becomes discoloured, and loses its curl in ethnic groups who would normally have curly hair. The liver may be enlarged with fat. Children with oedema are miserable, dislike being touched, and may have a moaning cry. They are apathetic and often refuse to eat. Children with oedematous malnutrition may appear chubby, but wasting becomes evident when oedema fluid is lost during the first few days of treatment. In the presence of oedema, muscle wasting can be best seen over the upper arms and shoulders.

Aetiology

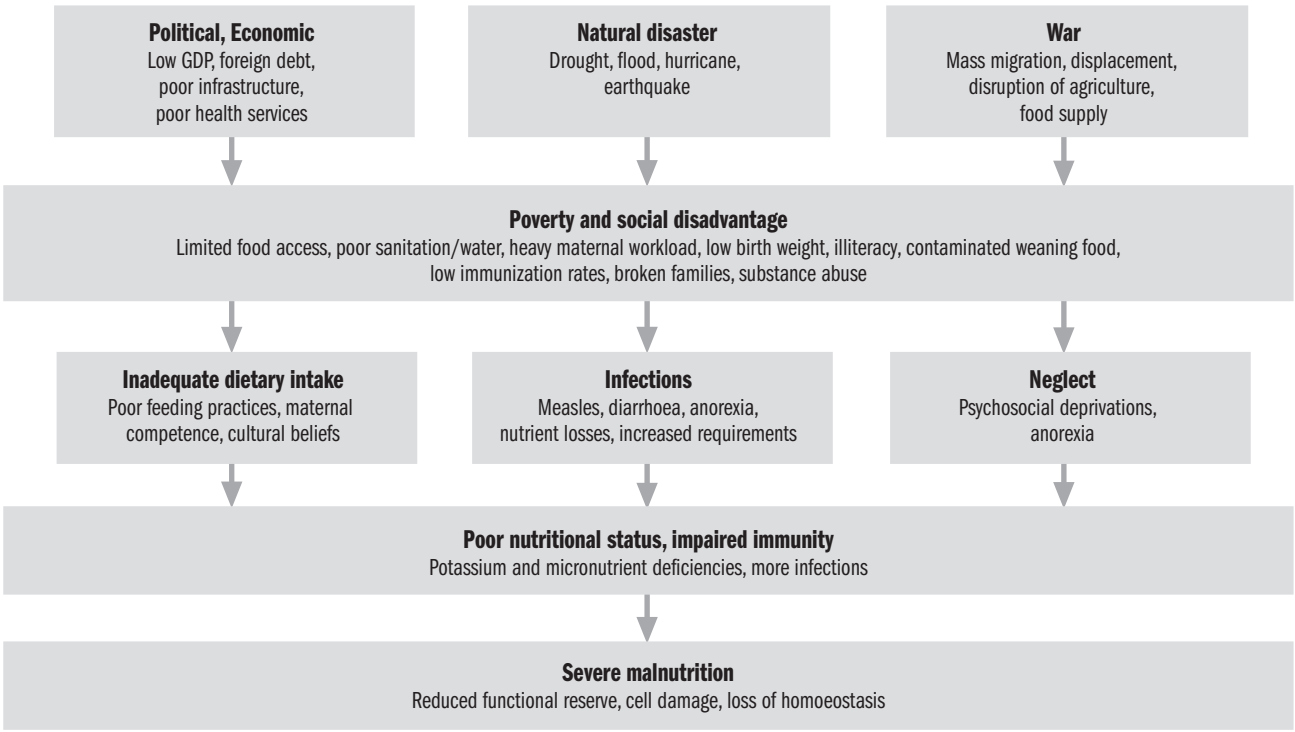
Many factors lead to malnutrition, including crop failure, displacement due to war, lack of food due to poverty, infections, poor weaning practices, and occasionally neglect (see **Fig. 9-3**).

Pathophysiology

When food intake is severely reduced, physiological and metabolic changes begin to take place in an orderly progression to conserve energy and support life for as long as possible. This process is called *reductive adaptation*. Fat stores are mobilized to provide energy and eventually muscle and skin proteins are also used. Energy is conserved by:

- reducing physical activity and growth;

Fig. 9-3 Aetiology of severe malnutrition



- reducing basal metabolism by:
  — slowing the rate of protein turnover,
  — reducing the functional reserve of organs,
  — slowing the Na^+/K^+ pumps in cell membranes, and
  — reducing inflammatory and immune responses.

Consequently:

- less heat is produced;
- loss of muscle leads to associated loss of potassium, magnesium, zinc and copper;
- red cell mass is reduced, liberating iron;
- the heart is smaller and weaker than usual, and has a reduced output; it cannot cope with excess fluid in the circulation, which increases the risk of heart failure;
- the kidneys cannot extract excess sodium or excess fluid from the blood; excess fluid easily builds up in the circulation;
- sodium builds up inside cells due to slower and fewer Na^+/K^+ pumps, leading to excess retention of sodium and water;
- potassium leaks out of cells and is lost in the urine, which contributes to electrolyte imbalance, anorexia, fluid retention and heart failure;
- the liver produces less albumin, transferrin and other transport proteins; gluconeogenesis is reduced; a dysfunctioning liver is less able to excrete toxins, such as aflatoxin, and cannot cope with excess dietary protein;
- the gut produces less gastric acid and enzymes; motility is reduced; bacteria often colonize the stomach and small bowel, damaging the mucosa and deconjugating bile salts; damaged, flattened villi cause impaired digestion and absorption of nutrients; and
- immune function is impaired, particularly cell-mediated immunity; there may be no fever, no redness or swelling, or raised white cell count during an infection.

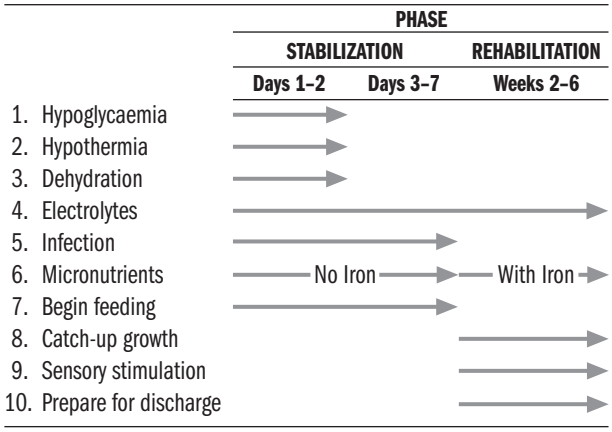
In addition, deficiencies of vitamin A, zinc, selenium and other antioxidant nutrients limit the ability to mop up free radicals that damage cell membranes. Leaky, damaged cells are thought to contribute to oedema formation, and skin and hair changes. Iron liberated from red cells is converted to ferritin and stored, but this process uses scarce glucose and amino acids. Unbound iron may increase if iron supplements are given initially, as there is insufficient transferrin to bind it. Unbound iron exacerbates some infections and promotes the generation of free radicals.

Management

Many children hospitalized with a common illness as the primary diagnosis (e.g. diarrhoea or pneumonia) are severely malnourished. A common and often fatal mistake is to try to treat the illness first and the malnutrition afterwards. This ignores the profound metabolic and physiological changes that exist in severe malnutrition and greatly increases the risk of death. The correct approach is to regard such children as having severe malnutrition with a coexisting infection and to treat both conditions simultaneously.

Severe malnutrition is managed in two phases: a *stabilization phase*, during which homoeostasis is restored and acute life-threatening conditions are prevented or treated, and a longer *rehabilitation phase* of rapid catch-up growth. Treatment procedures are based on a 10-step approach and are similar for both wasted and oedematous children. The approximate time-scale is shown in **Fig. 9-4**.

Fig. 9-4 Time-frame for management of a child with severe malnutrition



Severely malnourished children die in hospital from four main causes: *hypoglycaemia*, *hypothermia*, *heart failure*, and *infection*. Centres with a record of low mortality take immediate steps to prevent death from these causes. Centres where the mortality is high have practices that contribute to these causes of death.

1. **Hypoglycaemia**. Severely malnourished children are at increased risk of hypoglycaemia due to increased demands for, and a limited supply of, glucose. This is because:
   — their liver makes glucose less easily;
   — they have less glycogen in reserve because their muscles have wasted;

— they usually have multiple infections and the immune response requires relatively large amounts of glucose; and
— conversion of liberated red cell iron into ferritin increases the demand for glucose.

Treatment practices that delay feeding (e.g. long admission times) or lead to long gaps between feeds (e.g. no night feeds) contribute to hypoglycaemia and must be avoided. Severely malnourished children need to be given priority in the admissions queue and be sent quickly to the ward. *In the ward they must be fed straightaway and then every 2 or 3 hours by day and night*. Anorexic children should be fed by nasogastric tube. Ordinary meals prepared by hospital kitchens are not suitable for malnourished children. They need a specially prepared diet, such as F-75 (see below), which contains sugar or another readily available source of glucose. If this is not immediately available, give a solution of 10% glucose or sucrose.

2. **Hypothermia**. Severely malnourished children have an increased risk of developing hypothermia due to:

   — reduced heat production from lowered basal metabolism and diminished physical activity;
   — increased heat loss from their relatively larger surface area/kg and loss of the insulation normally provided by subcutaneous fat; and
   — infections, which increase the demand for glucose via the immune system.

   Conditions in the wards may add to the risk of hypothermia as they may be draughty and cold. Children become chilled if they are not fully dressed, if left in wet clothes, or if they are not dried carefully after bathing.

   Hypoglycaemia and hypothermia often occur together and can be signs of severe infection. The combination of hypoglycaemia, hypothermia and infection is a 'deadly triad'. Keeping malnourished children warm and dry, giving frequent feeds by day and night, and treating infections straightaway help to prevent deaths from both hypoglycaemia and hypothermia.

3. **Dehydration**. Severely malnourished children often have diarrhoea. It is difficult to diagnose dehydration in a severely malnourished child because the common signs (slow skin pinch, sunken eyes, dry mouth, absent tears) are similar to the signs of malnutrition itself. All severely malnourished children with watery diarrhoea should be assumed to have some dehydration.

   Many malnourished children die from fluid overload during rehydration. There are three main reasons:

   — the degree of dehydration is overestimated, because of confusion between signs of malnutrition and signs of dehydration;
   — fluids are given intravenously, which increases the risk of overload; and
   — children are not monitored carefully during rehydration, so fluid overload is not diagnosed until it is too late.

   Severely malnourished children with diarrhoea *should not be given IV fluids*, except when there is shock. Instead they should be rehydrated orally with a special rehydration solution for malnutrition (ReSoMal). This is low in sodium (45 mmol/l) and has added potassium, magnesium, and sugar. Rehydration (5 ml/kg every 30 min for 2 h and then 5–10 ml/kg/h for up to 10 h) is slower than for well-nourished children. Pulse and respiration rates should be monitored every 30 min for the first 2 h and then hourly for signs of overhydration.

4. **Electrolytes**. All severely malnourished children have excess body sodium (even though plasma sodium may be low) and deficiencies of potassium and magnesium. Oedema is partly due to these imbalances. All malnourished children should be given extra potassium (4 mmol/kg/d as potassium chloride) and extra magnesium (0.6 mmol/kg/d as magnesium chloride) for at least 2 weeks. A combined solution (e.g. a vitamin-mineral mix) can be added to feeds and to ReSoMal. Sodium should be restricted. Diuretics make potassium deficiency worse and should never be given to treat oedema.

5. **Infection**. Infections are easily missed in severely malnourished children as the normal signs are often absent. *All children should, therefore, be presumed to be infected, even if there are no clinical signs. A broad-spectrum oral antibiotic such as cotrimoxazole should be given straightaway on the first day of admission*. Children who are hypoglycaemic, hypothermic or who appear seriously ill should be given IM or IV ampicillin and gentamicin, or a similar combination that provides Gram-negative cover.

   Malnourished children are very vulnerable to nosocomial infection. Good ward hygiene and handwashing by doctors, nurses and other carers are therefore important. Overcrowding and sharing of cots should be avoided. Measles vaccination should be given to non-immunized children aged >9 months.

6. **Micronutrients**. All severely malnourished children have vitamin and mineral deficiencies which must be corrected. A large dose of vitamin A should be given

on Day 1 to boost immune function and prevent blindness. If there is any sign of xerophthalmia, the dose should be repeated on Day 2 and Day 14. Other deficiencies are treated by giving multivitamins, folic acid, zinc and copper. Provision of iron should be delayed until the child is gaining weight in the rehabilitation phase.

7. **Initial feeding**. During the stabilization phase, the aim is to provide just enough energy and protein to meet basic needs (100 kcal/kg/d and ~1g protein/kg/d) and 130 ml fluid/kg/d (or 100 ml/kg/d if the child has gross oedema). Giving too much food initially stresses the liver, kidneys, heart and gut. Initial feeding should consist of small, frequent feeds of a low osmolar and low lactose diet that contains 75 kcal and 0.9 g protein per 100 ml. Milk-based feeds (such as starter formula F-75) are best for most children and should be started immediately after admission. Breastfeeding should be continued.

8. **Catch-up growth**. Readiness to enter the rehabilitation phase and start catch-up growth is signalled by a return of appetite, usually about one week after admission. Feeding should then be increased. A controlled transition over 3 days is recommended to prevent a child suddenly consuming huge amounts, which can lead to heart failure. Thereafter, unlimited amounts should be given.

   The aim of the rehabilitation phase is to achieve intakes of 150–220 kcal/kg/d and 4–6 g protein/kg/d. The recommended catch-up formula (F-100) contains 100 kcal and 2.9 g protein/100ml. Modified porridges or modified family foods can be used, provided they have comparable energy and protein concentrations. Mothers should be encouraged to continue breastfeeding. Rates of weight gain are considered poor if <5 g/kg/d, moderate if 5–10 g/kg/d, and good if >10 g/kg/d.

9. **Sensory stimulation**. In severe malnutrition there is delayed mental and behavioural development. Loving interactions and structured play help children recover mentally and behaviourally. The ward should provide a cheerful, stimulating environment where daily play and physical activities are led by staff and involve the mother.

10. **Prepare for follow-up**. Continuing care after discharge and back referral letters should be organized before children leave hospital. The parent or caregiver needs to be given knowledge and skills to feed the child at home in ways that will promote good health and continued catch-up growth. This includes what to feed, how much and how often, and using foods that are affordable and culturally acceptable. They also need to know how to provide psychosocial stimulation to promote development. Parents should be advised to take their child for regular follow-up checks and ensure that booster immunizations are given, and also 6-monthly vitamin A doses in areas where vitamin A deficiency and xerophthalmia are common.

Further reading

Ahmed T, Ali M, Ullah MM et al. Mortality in severely malnourished children with diarrhoea and use of a standardised management protocol. *Lancet* 1999; **353**:1919–22.

Ashworth A. Treatment of severe malnutrition. *J Pediatr Gastroenterol Nutr* 2001; **32**:516–8.

Chopra M, Wilkinson D. Treatment of malnutrition. *Lancet* 1995; **345**:788–9.

Deen JL, Funk M, Guevara VC, Saloojee H, Doe JY, Palmer A et al. Implementation of the WHO severe malnutrition guidelines in hospitals in Africa. *Bull World Health Organ* 2003; **81**(4):237-243.

Jackson A. Severe malnutrition. In: Warrell DA, Cox TM, Firth JD, Benz EJ, eds. *Oxford Textbook of Medicine*, Fourth Edition. Oxford: Oxford University Press, 1996.

Pelletier DL, Frongillo EA, Jr., Schroeder DG, Habicht JP. The effects of malnutrition on child mortality in developing countries. *Bull WHO* 1995; **73**(4):443-448.

Schofield C, Ashworth A. Why have mortality rates for severe malnutrition remained so high? *Bull WHO* 1996; **74**:223–9.

Waterlow J C. *Protein–energy malnutrition*. Edward Arnold: London, 1992.

Wellcome Trust. *Topics in International Health*. Nutrition. ISBN 0 85 199 251 X. London 1999

World Health Organization. *Management of severe malnutrition: a manual for physicians and other senior health workers*. Geneva, 1999.

Questions

9.1 Define severe malnutrition and describe the three main types of presentation.

9.2 Describe ways in which poverty and social disadvantage can lead to severe malnutrition.

9.3 Describe the effects of severe reduction in food intake on four tissues or organs (reductive adaptation).

9.4 List the main elements of each of the two phases in the management of severe malnutrition and describe their timing.

9.5 Explain why hypoglycaemia and hypothermia are common in severely malnourished children, and describe hospital practices which place the child at risk of these complications.

10. Children with HIV/AIDS

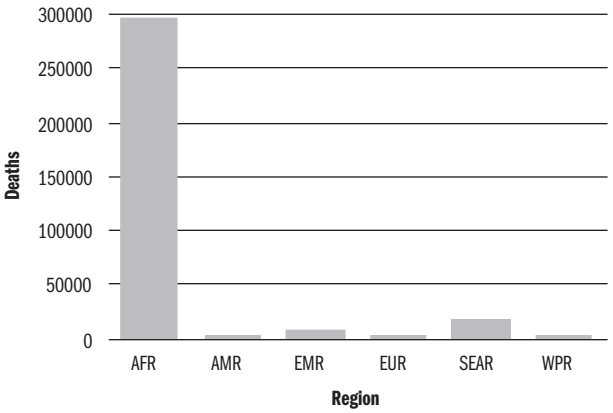
HIV/AIDS is caused by the human immunodeficiency virus which attacks cells of the immune system, so that the affected child is more vulnerable to a wide variety of infections. This section discusses the management of children with HIV/AIDS and prevention of mother-to-child transmission of HIV.

See also: ***Management of the Child with a Serious Infection or Severe Malnutrition*, Chapter 8, Page 92**

Introduction

It is estimated that 40 million people are infected with HIV. In 2003, there were 5 million new infections and 3 million deaths from HIV. The principal forces driving the paediatric HIV epidemic are the adult heterosexual epidemic, the large number of fertile infected women, the high rate of mother-to-child transmission (MTCT), the very high rates of breastfeeding of infants in developing countries, and unsafe transfusion practices.

Fig. 10-1 Number of deaths from HIV/AIDS in children under 5 years of age, estimated for the year 2000, by region (WHO)



AFR African Region, AMR American Region, EMR Eastern Mediterranean Region, EUR European Region, SEAR South-East Asian Region, WPR Western Pacific Region

Burden of disease in children

Of an estimated 1.3 million children infected with HIV, 1.1 million are in Africa. WHO estimated that 400,000 child deaths were due to HIV in the year 2000. The HIV epidemic is reversing the declines in infant and child mortality rates (IMR and CMR) that have occurred in many African countries over the last 25 years. Current IMR and CMR in Southern African countries are estimated to be some 20–30% higher than they would have been without the HIV epidemic. **Figure 10-1** illustrates the predicted loss of life due to HIV/AIDS in children in 2000, by WHO region.

Aetiology

HIV/AIDS is caused by the human immunodeficiency virus (HIV 1 and 2) which attacks cells of the immune system, making the affected child more vulnerable to a wide variety of infections.

HIV infection

Transmission. Over 90% of paediatric HIV infections are maternally acquired. MTCT occurs during pregnancy, delivery or postnatally from breastfeeding. Transmission rates without any intervention vary from 15–25% in developed countries, or up to 30–45% in settings where breastfeeding is common and prolonged. The current MTCT rate in the USA is <2%, indicating that interventions to reduce MTCT can be highly effective. High maternal viral load, prolonged duration of ruptured membranes, vaginal delivery and presence of other sexually transmitted diseases all increase the risk of MTCT. Breastfeeding approximately doubles the overall risk, with the risk increasing with the total duration of breastfeeding. However, the use of breastmilk substitutes is limited by their high cost. In addition, where clean water and sanitation are lacking they cause increased mortality due to diarrhoeal disease.

Diagnosis. Most children in developing countries acquire HIV from their mothers around the time of delivery or during

breastfeeding. However, as all babies born to HIV-infected women have maternal antibodies until 15–18 months of age, HIV antibody tests cannot be used to diagnose HIV infection until around that age. Early definitive diagnosis requires virologic tests (such as DNA or RNA PCR), but these are costly. Where PCR tests are available and antiretroviral therapy (ART) is possible, a reasonable approach is to use the test to confirm HIV infection in any infant with symptoms suggestive of HIV. After the age of 18 months and when breastfeeding has stopped for at least 6 months, an HIV antibody test alone can be performed. In most resource-poor settings, definitive laboratory diagnosis in young children is not currently possible and clinical definitions of HIV infection lack both sensitivity and specificity in this age group. Cheaper reliable laboratory tests for use in infants and young children are urgently required.

Diseases associated with HIV infection. Early cohort studies in developed countries reported that about 20% of HIV-infected infants progressed to AIDS or death by 12 months of age. In Africa, 23–35% of HIV-infected children die in the first year and 57–68% within the first 5 years of life. Children with HIV have more rapidly progressive disease than adults and experience more frequent bacterial and opportunistic infections. It has been estimated that CMR increases by 3 per 1000 for every 1% increase in antenatal HIV seroprevalence (assuming 1/3 of infected women transmit HIV to their babies).

Early HIV immunosuppression results in more frequent, more severe and recurrent forms of infections, such as pneumonia, sepsis, tuberculosis and measles. These illnesses present in a similar fashion to those in HIV-negative children, so that infected and uninfected children often cannot be distinguished. There is no evidence of increased risk of malaria. Most HIV-infected children in developing countries die because of inadequate treatment, rather than from opportunistic infections that occur later as immunosuppression worsens.

Pneumocystis jiroveci (formerly *P. carinii*) pneumonia (PCP) is the commonest opportunistic infection in HIV-infected children. Studies of children in Malawi have shown that PCP is responsible for 15–20% of severe pneumonia cases in HIV-infected children and for 30–50% of HIV-related deaths in infancy. PCP usually presents as very severe pneumonia between 2 and 6 months of age, and is poorly responsive to treatment.

Since many HIV-related diseases are severe, HIV infected children occupy a large proportion of hospital beds in HIV prevalent areas. Because many of these children are orphans no family may be present to provide nursing care. HIV-infected children make up an increasingly high proportion of hospital paediatric deaths in Africa.

Associated social problems

13 million children throughout the world have been orphaned by the death of their parents from HIV and this number is expected to double by 2010. Most orphans have poor access to education and health services. As a vulnerable group they are more likely to be abused, neglected or stigmatized, and to have a higher risk of death in childhood. Despite strong traditions of intra-family fostering, the ability of extended families to cope has become saturated in many African countries and community-based orphan support programmes are being established. In many sub-Saharan countries the limited ability to respond to these problems is further threatened by loss of health workers and teachers to AIDS.

Management of children with HIV infection

HIV-infected children must have good access to basic child health facilities for the prompt management of common childhood infections. In addition, new services, such as palliative care, need to be developed. Full EPI immunization and nutritional interventions, such as vitamin A and zinc supplementation in populations at risk of deficiency, will reduce morbidity and mortality. In addition, attention should be given to specific HIV/AIDS services and provision of anti-retroviral therapy.

Antiretroviral therapy. Children metabolize antiretroviral drugs faster than adults and require higher than adult equivalent doses to achieve appropriate drug levels. The limited studies of Antiretroviral Therapy (ART) in children suggest that results similar to those in adults are seen with many different regimens.

PCP prophylaxis in infants. PCP can be prevented by prophylaxis with cotrimoxazole. PCP is very unusual now in countries where prophylaxis is routine. Cotrimoxazole prophylaxis has been shown to be very effective in HIV-infected South African infants, reducing the proportion of severe pneumonias that are PCP from 15% to 1.7%. Since it is very difficult to confirm HIV infection in infancy, cotrimoxazole prophylaxis to begin from 6 weeks up to the age of 6 months has been recommended for *all* infants born to HIV-infected mothers.

PCP prophylaxis in older children. After 6 months of age, cotrimoxazole prophylaxis has the potential to reduce morbidity due to malaria, invasive bacterial infections and diarrhoea. However, this must be balanced against the serious risk that widespread use of cotrimoxazole for

prophylaxis will accelerate the development of malaria resistance to sulfadoxine/pyrimethamine. Such prophylaxis programmes are therefore not widespread in developing countries. Current UNAIDS recommendations for cotrimoxazole prophylaxis in HIV-infected children are based on studies done in HIV-infected adults in regions where bacterial resistance, and possibly malaria resistance, to cotrimoxazole are much lower than in most African countries.

Prevention of mother-to-child transmission of HIV

WHO and its partner agencies are promoting a four-pronged approach which involves:

- primary prevention of HIV infection;
- prevention of unintended pregnancies among HIV-infected women;
- prevention of HIV transmission from HIV-infected women to their infants; and
- provision of care and support to HIV-infected women, their infants and family.

The main determinant of the rate of childhood infection with HIV is the extent of the epidemic in adults. Thus, reductions of new infections in young women by promoting safer sex and better treatment of sexually transmitted diseases, and ensuring a safe blood supply will reduce the risk to children. A number of strategies to reduce transmission of HIV and HIV-infected women to their infants have been assessed by randomized controlled trials. The following have proven to be effective:

- prophylactic antiretroviral (ARV) treatment, including with various short-course regimens appropriate to resource-constrained settings.
- infant feeding counselling and support for safer options to reduce the risk of transmission through breastfeeding. While breastfeeding can add to the risk of HIV transmission by 5–20%, lack of breastfeeding can expose children to an increased risk of malnutrition or infections diseases other than HIV. When replacement feeding is acceptable, feasible, affordable, sustainable and safe, avoidance of all breastfeeding by HIV-infected mothers is recommended. Otherwise, exclusive breastfeeding is recommended during the first months of life and should then be discontinued as soon as it is feasible; and
- safer delivery practices, including avoidance of invasive obstetrical procedures. Elective caesarian section can reduce the risk of HIV-transmission, but may not be appropriate in resource-constrained settings.

Effective interventions require that a woman knows her HIV status, which means making testing and counselling available in antenatal clinics, or in other places accessible to pregnant women. Many countries have begun implementing programmes to prevent mother-to-child HIV transmission incorporating the above elements, and some, such as Botswana, Brazil and Thailand, have national programmes. The wider availability of ARVs through treatment access initiatives should improve countries' ability to provide services.

Further reading

Duggan C, Fawzi W. Micronutrients and child health: studies in international health and HIV infection. *Nutr Rev* 2001; **59**:358–69.

Gilks CF. HIV care in non-industrialised countries. *Brit Med Bull* 2001; **58**:171–86.

Graham SM, Mtitimila EI, Kamanga HS, Walsh AL, Hart CA, Molyneux ME. The clinical presentation and outcome of Pneumocystis carinii pneumonia in Malawian children. *Lancet* 2000; **355**:369–73.

Mbori-Ngacha D, Nduati R, Reilly M, Richardson B, Mwatha A, Ndinya-Achola J, Bwayo J, Kreiss J. Morbidity and mortality in breastfed and formula-fed infants of HIV-infected women: a randomised controlled trial. *JAMA* 2001; **286**:2413–20.

McIntyre J, Gray G. What can we do to reduce mother to child transmission of HIV? *Brit Med J* 2002; **324**:218–21.

Wellcome Trust. *Topics in International Health. HIV/AIDS*. ISBN 0 85 199 250 1. London 1999.

WHO. New Data on the Prevention of Mother-to-Child Transmission of HIV and their Policy Implications: Technical Consultation, UNFPA/UNICEF/WHO/UNAIDS Inter-Agency Team on Mother-to-Child Transmission of HIV, Geneva, 11–13 October 2000, Conclusions and Recommendations. 2001. WHO, Geneva. WHO/RH/01.28. Available on UNAIDS web site, http://www.unaids.org.).

WHO. Recommendations for the safe and effective use of short-course ZDV prevention of mother to child transmission of HIV. *Wkly Epidemiol Rec* 1998; **73**:313–20.

WHO: Scaling Up Antiretroviral Therapy in Resource-Limited Settings. Geneva 2002. Accessible at http://www.who.int/hiv

WHO, UNICEF, UNFPA and UNAIDS. *HIV and Infant Feeding: Guidelines for decision-makers*. Geneva, 2003 (revised).

WHO, UNICEF, UNFPA and UNAIDS. *HIV and Infant Feeding: A Guide for health-care managers and supervisors*. Geneva, 2003 (revised).

WHO, UNICEF, UNFPA, UNAIDS, World Bank, UNHCR, WFP, FAO, IAEA. 2003. HIV and Infant Feeding: Framework for Priority Action. Geneva, 2003. Available on WHO web site, http://www.who.int/child-adolescent-health.

Zar HJ, Dechaboon A, Hanslo D, Apolles P, Magnus KG, Hussey G. Pneumocystis carinii pneumonia in South African children infected with human immunodeficiency virus. *Pediatr Infect Dis J* 2000; **19**:603–7.

Zwi K, Pettifor J, Soderland N, Meyers T. HIV infection and in-hospital mortality at an academic hospital in South Africa. *Arch Dis Child* 2000; **83**:227–30.

Questions

10.1 Describe when mother-to-child transmission (MTCT) of HIV occurs and the risk factors for MTCT.

10.2 Explain the problems with the interpretation of HIV test results in young children.

10.3 Discuss the role of PCP (pneumocystis pneumonia) prophylaxis in infants and in young children.

10.4 Describe the four main approaches to reducing MTCT, which are promoted by WHO.

10.5 List the main issues to cover in the discussion with a HIV-infected mother to decide on the most appropriate infant feeding method for her and her baby.

11. Supportive care

Management of a sick child involves both specific treatment (such as giving an antibiotic to control a bacterial infection) and supportive care (such as ensuring proper nutrition). Both are essential elements of good case management. Some of the most important aspects of supportive care are described below in the management of nutrition and fluids, fever, anaemia and oxygen therapy.

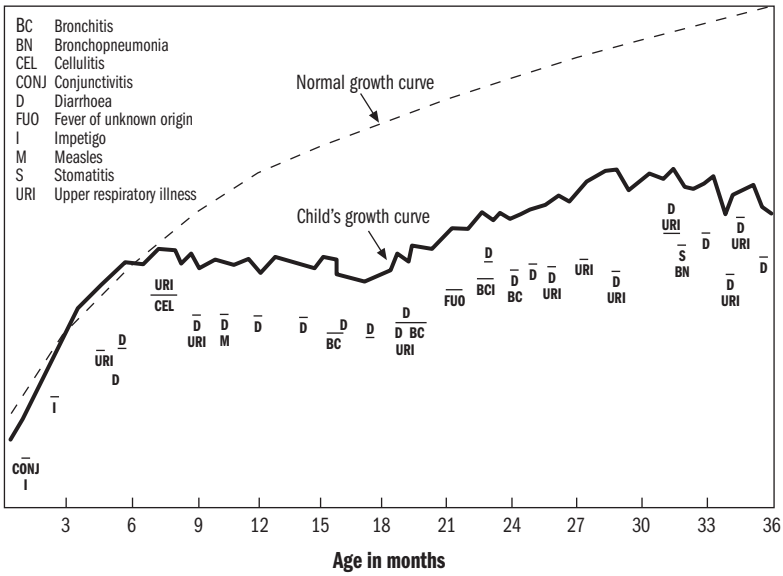
See also: ***Management of the Child with a Serious Infection or Severe Malnutrition*, Chapter 9, Page 99**

Nutritional and fluid management

Feeding the sick child

Weight loss is common during acute illness and is greatest when feeding is reduced due to anorexia, withholding of food, or giving foods of low nutritional value. Moreover, if feeding is not appropriate, the weight lost during illness may not be fully regained and can become an important cause of malnutrition, including both wasting and stunting. **Fig. 11-1** shows how severe growth faltering may be associated with repeated infections.

Fig. 11-1 Growth pattern of a child with frequent episodes of diarrhoea and other infections



Source: Mata, LJ. Nutrition and infection. *Protein Advisory Group bulletin* 1971; **11**:18–21.

Fig. 11-2 Effect of feeding on weight gain in children with acute diarrhoea
(Brown et al. 1988)

Diet	Average weight gain (grams)	
	Day 5	Day 8
Schedule 1		
Days 1–2: fasting		
Days 3–4: half strength diet	–135	+195
Days 5–8: full strength diet		
Schedule 2		
All days: full strength diet	+80	+310

Feeding should always be continued during illness, providing as much nutritious food as the child will accept. The *only* exceptions are conditions in which the food cannot safely pass into and through the intestine, such as intestinal obstruction, paralytic ileus or surgical conditions of the abdomen. *When feeding is continued, weight loss is minimized, recovery from illness is hastened, and long-term health is improved.* Weight that is lost during an acute illness can usually be recovered by ensuring a generous intake of nutritious food during convalescence. **Fig. 11-2** compares the effect of reduced feeding (fasting on days 1–2, half-strength diet on days 3–4, and full-strength diet on days 5–8), or continued full-strength feeding, on weight gain in children with diarrhoea. The benefit of full-strength feeding is clear.

The nutritional management of sick children in hospital has two objectives:

- To sustain growth, or minimize weight loss, by continuing to provide nutritious foods that the child will accept and that are appropriate for the child's age.
- To assess how the child has been fed before becoming sick and to counsel mothers on how best to feed their child at home, *especially if the child is malnourished*.

To achieve these objectives:

- Breastfeeding should always be continued; a sick child will often take breastmilk even when refusing other foods; animal milk or formula should be given only if the child is not normally breastfed.
- Energy-rich soft or solid foods should be included in the diet for children older than 6 months.
- Children should be offered food frequently and encouraged to eat; tube-feeding may be needed for those who are unable to eat for more than 1–2 days.
- Children should be weighed at least twice a week, and more often if possible; continued weight gain is the most important evidence that feeding is adequate.
- Whenever possible, the mother should be actively involved in feeding her child in hospital, giving nutritious foods that are similar to those she can prepare at home.
- When the child nears discharge, confirm that the mother knows how to feed her child appropriately at home; and instruct her to give at least one additional meal per day until the child has regained any lost weight and is growing well.

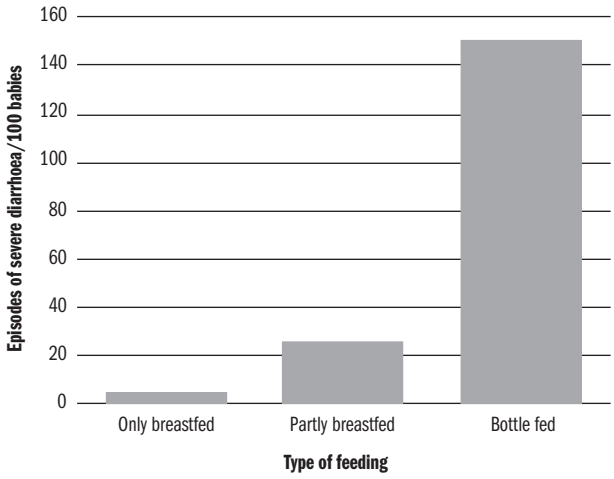
Breastfeeding of infants aged up to 6 months

Breastfeeding should begin within 30 minutes after birth. This provides colostrum, which contains antibodies that protect against infections and may help to prevent the later development of allergies. Early breastfeeding also increases the rate of successful breastfeeding. Newborns with early skin-to-skin contact have better temperature control, higher blood glucose levels, and cry less than those without early contact.

Before milk flows freely, the infant needs *no fluids* other than colostrum. When pre-lacteal fluids are given, the infant may not want to suckle or may not suckle efficiently, and is at increased risk of developing diarrhoea and other infections. Furthermore, the mother may develop engorgement of the breasts or mastitis, her breastmilk will take longer to come in, and she is more likely to fail at breastfeeding.

Exclusive breastfeeding for the first 6 months. Infants should be *exclusively breastfed* from birth until 6 months of age. This means the healthy infant should receive *only breastmilk* and no other fluid or food by mouth. Breastfeeding provides *all* of the fluid and nutrients a healthy baby needs during the first 6 months of life. Moreover, by providing maternal antibodies and avoiding potentially contaminated fluids or foods, breastfeeding protects against diarrhoea (see **Fig. 11–3**), pneumonia and otitis media. The risk of dying from diarrhoea is reduced fourfold, and from pneumonia twofold, in exclusively breastfed infants. Breastfeeding also prolongs amenorrhea in the mother, thereby delaying her fertility and increasing birth interval, which has important benefits for survival of the infant.

Fig. 11-3 Relative risk of diarrhoea during the first 6 months depending on feeding
(Mahmood et al. 1989)



Low birth weight babies. Breastfeeding has many advantages for low-birth-weight (LBW) infants (<2.5 kg at birth). Amino acids and fats present in breastmilk are physiologically adapted to the needs of the infant, with composition changing as the infant ages. Proteins and fats in the breastmilk of a mother with an LBW infant are easy to digest. Breastmilk also provides a low renal solute load and has active enzymes that enhance maturation of the gut. The anti-infective properties of breastmilk are especially important for LBW infants, and there is some evidence that breastmilk protects against necrotizing enterocolitis. LBW babies often have low iron stores and breastmilk has a low content of iron. To avoid iron deficiency, supplemental iron drops should be given from age 2–3 months until complementary foods are introduced.

Breastfeeding positioning and attachment. A baby who is well positioned and correctly attached to the breast will suckle

effectively and get enough milk. As a result of efficient suckling and emptying of the breast, the mother will continue to produce milk. A baby who is poorly attached may injure the nipple and cause pain. If the baby does not remove milk effectively, the breast will become engorged, milk production may diminish, and the baby may be unsatisfied and fail to gain weight. If a mother's milk supply is reduced she should increase it by encouraging the baby to suckle frequently and offering the baby her breast whenever the baby will suckle.

Babies who cannot breastfeed. Infants who cannot breastfeed should be given expressed breast milk or, if this is not possible, safely prepared animal milk or formula. These should be given by cup or spoon. Feeding bottles should never be used because they are easily contaminated with disease-producing bacteria.

A discussion on breastfeeding and HIV transmission is given in Chapter 10 and has been reviewed by a number of international agencies.

Feeding of infants and children aged 6 months or older

Breastfeeding should continue at least until 2 years of age. Significant protection against infections continues for as long as the child is breastfed, even after complementary feeding is begun. Breastfeeding also remains an important source of both macronutrients and micronutrients after complementary foods are added. Continuing breastfeeding has been linked to reduced risk of childhood chronic illnesses and obesity, as well as to improved cognitive function.

The transition from exclusive breastfeeding to feeding only with family foods is progressive, typically beginning at age 6 months and lasting until 24 months of age. Starting complementary foods too early increases unnecessarily the risk of diarrhoea from contaminated feeds. And if the mother breastfeeds less frequently, or stops, she may be at increased risk of becoming pregnant. On the other hand, if complementary feeding is begun too late, the energy and nutrient needs of the child may not be fully met, growth may slow, and the risk of malnutrition and micronutrient deficiency is increased.

The period of greatest risk for developing malnutrition is from 6 to 24 months of age. The immediate causes are inadequate intake of energy and nutrients, and frequent infections, both of which are directly linked with poor complementary feeding. The goal of complementary feeding is to fill the gap between the total nutritional needs of the infant or child and the proportion of those needs provided by breastmilk, so that malnutrition is avoided. To achieve this, infants and young children must receive both a sufficient quantity and variety of energy-rich complementary foods to meet their macronutrient and micronutrient requirements. **Table 11-1** summarizes the feeding recommendations during sickness and health.

Foods must also be prepared in a way that is suited to the infant's level of development. In most infants, oral motor function is adequate for soft foods by age 6 months. By 8 months infants are able to chew and can eat "finger foods". By 12 months most children can eat family foods of a solid consistency. The method of feeding a child (handling a spoon, chewing, using finger foods) should be adapted to the child's psychomotor abilities and should change as these develop. Initially, the caregiver should actively feed the child; as the child develops he should increasingly feed himself.

Table 11-1 Feeding recommendations during sickness and health[a]

Up to 6 months of age	6 months up to 12 months	12 months up to 2 years	2 years and older
• Breastfeed as often as the infant wants, day and night, at least 8 times in 24 hours. • Do not give other food or fluids.	• Breastfeed as often as the infant wants. • Give adequate servings of thick cereal gruel with added oil, cooked beans or lentils, mashed soft vegetables or fruit, or egg. – 3 times per day if breastfed; – 5 times per day if not breastfed.	• Breastfeed as often as the child wants. • Give adequate servings of cereal gruel with oil; rice, beans, or lentils; bread; eggs, cheese, meat or fish; and fruit, or family foods, 5 times per day.	• Give family foods at three meals each day. Also, twice daily, give nutritious snacks between meals, such as bread, milk, fruit, cheese, yoghurt, beans or ground nuts.

[a] The specific foods shown are examples. Diets should be adapted to reflect local practices and available foods, using foods of similar nutritional value.

The recommended number of complementary feeds per day is based on the energy needs and gastric capacity of the infant or young child, and on whether the child is continuing to breastfeed. Infants who do not continue to breastfeed require more frequent meals. In breastfeeding infants, however, an increased meal frequency may adversely affect breastfeeding by reducing the intake of breastmilk.

Complementary foods and drinks are easily contaminated, either during preparation or storage, and are an important cause of diarrhoea, which occurs most frequently between 6 and 12 months of age. The risk of diarrhoea can be reduced by hand washing before preparing or serving food, heating food thoroughly before it is eaten, and using only clean or boiled water for drinks.

Fluid management

Daily fluid requirements are the sum of *normal maintenance requirements* plus the water and salts needed to replace any *unusual losses*. Maintenance fluids are needed to replace normal losses of water and salts in urine and faeces, and evaporative losses from the lungs and skin. *A child who eats and drinks normally, including an exclusively breastfed infant under 6 months of age, requires no added water or salts to meet normal maintenance needs.*

Maintenance requirements for water are based on the child's metabolic rate, an average of 100 ml being required for each 100 calories of energy expended. Normal requirements are proportional to body surface area, which is greater, in relation to weight, in infants than in older children. For example, a healthy infant weighing <10 kg requires 100–120 ml/kg/day of water, whereas a child weighing >20 kg needs only 50–90 ml/kg/day.

Unusual losses of water and salts occur both externally and internally. *External losses* include diarrhoea, increased evaporative loss due to fever or fast breathing, and losses due to sweating, excessive vomiting, gastric drainage or through extensive burns. *Internal losses* occur when vascular permeability is increased and fluid "leaks" from the intravascular space into tissues. This occurs in septic shock and dengue shock syndrome, and also when fluid accumulates within the lumen of an obstructed bowel, or in the peritoneal cavity as ascites. When unusual losses occur, the goal of treatment is to replace the water and salts that have been lost while also meeting normal maintenance needs. The type of fluid required and amount needed depend upon the specific condition being treated. Examples are shown in the **Table 11-2**.

Table 11-2 Water and salt replacement in various conditions

Condition	Treatment objective	Replacement fluid
Normal	Replace normal losses of water and salt (maintenance)	Normal fluids, including breastmilk, milk, or formula, water and food by mouth
Fever, sweating, fast breathing due to serious infection	Replace normal *and* additional losses of water and salt	Increase normal fluids by mouth [a]
Diarrhoea	Replace deficit and continuing losses of water and salts in liquid stools; prevent dehydration	ORS solution by mouth Only for severe dehydration: give IV Ringer's lactate, half-strength Darrow's solution (or 0.9% saline) [b]
Persistent vomiting or gastric suction	Replace lost gastric fluid; prevent metabolic alkalosis	0.9% saline (IV)
Shock or burns	Restore intravascular volume; correct shock	Colloid (5% albumin or dextran), or 0.9% saline or Ringer's lactate (IV) [b]

[a] IV replacement is needed *only* if the child cannot drink or breastfeed normally. Use a *single* solution of 1/2 normal (0.45%) saline combined, if possible, with 5% glucose. Give 100–120 ml/kg if child weighs <10 kg; 90–120 ml/kg if 10-19 kg; and 50–90 ml/kg if >20 kg. Increase these amounts by 10% for each 1 °C of fever. Also use 0.45% saline for giving IV antibiotics.

[b] See further details in section on diarrhoea.

Unusual *external losses* should be replaced as they occur with equal volumes of the appropriate fluid, if possible by mouth. Children should be observed carefully and often to ensure that replacement is sufficient, but not excessive. Persistent or recurring signs of dehydration indicate insufficient fluid replacement; puffy eyelids, distended external jugular veins or signs of heart failure indicate excessive replacement. The risk of excessive replacement is greatest when fluids are given intravenously. When treating shock due to *internal losses*, the objective is rapidly to restore and maintain an effective blood volume as shown by a strong pulse, urine flow and normal blood pressure.

Management of fever

Physiology of fever

Fever is present when the rectal temperature, correctly measured, exceeds 38 °C. Oral and axillary temperatures average 0.5 °C and 0.8 °C lower, respectively.

The core body temperature is fixed by the temperature "set point", which is controlled by the anterior hypothalamus. Core body temperature is maintained at the set level by sweating (to dissipate excess heat) or shivering (to generate and conserve heat). Fever is caused by an upward adjustment of the set point and is usually the result of a bacterial or viral infection, or another inflammatory condition, such as rheumatic fever. These conditions cause the release of interleukins, especially IL-1, and other mediators of inflammation, which stimulate prostaglandin E2 production in the anterior hypothalamus. This, in turn, resets the temperature set point to a higher level. Body temperature is then raised by increased metabolism, increased muscle tone, and diminished blood flow to the skin.

Data from laboratory studies and a limited number of animal studies suggest that fever may improve immune defences against infection. There are, however, few clinical studies on the possible benefits of fever in children or on how these might be affected by antipyretic treatment. In contrast, adverse effects of fever are well described. These include:

- listlessness or irritability, and reduced appetite,
- convulsions in some children aged 6 months to 5 years, especially when the temperature exceeds 40 °C or rises rapidly,
- increased oxygen consumption, which increases the work of the cardiopulmonary system and can be dangerous for children with underlying pulmonary or cardiac problems, such as pneumonia, or heart failure due to severe anaemia, and
- brain damage when the temperature exceeds 42 °C, as can occur in heatstroke, when sweating stops and temperature regulation fails.

Treatment to reduce fever seeks to avoid, or reduce the severity of, these effects.

Management of fever – general principles

A child with fever should be examined carefully to determine the cause and treated appropriately. For infants aged 2 months or more, unexplained fever is not a reason to give an antibiotic. These should be given only when signs of a bacterial infection are present, such as cough and fast breathing (pneumonia) or stiff neck and altered consciousness (meningitis). In malarious areas, however, a child with fever, but without signs of bacterial infection, should be treated with an antimalarial drug.

In infants younger than 2 months, fever (>37.5 °C, axillary), or hypothermia (<35.5 °C), may be the only sign of serious bacterial infection. Febrile or hypothermic young infants should be examined carefully to identify a possible source of infection and treated for sepsis with an appropriate parenteral antibiotic.

Treatment to reduce fever should be given when the temperature is ≥39 °C rectal (≥38.5 °C axillary) and the child is listless or irritable. The treatment will reduce irritability and improve the child's interest in eating. Treatment should also be given to children with cardiac or pulmonary problems, or a history of febrile seizures. A temperature >42 °C suggests possible heat stroke, and must be lowered rapidly to avoid brain damage or death.

Antipyretic treatment

Antipyretic drugs work by inhibiting the production of prostaglandin E2 in the anterior hypothalamus and thereby resetting the temperature set point towards normal. They also have important benefits as analgesics. Where an antipyretic drug is required, paracetamol is recommended because of its superior safety. All, however, can cause serious adverse effects; they should be used only when clearly indicated to relieve fever or pain.

Paracetamol (acetaminophen) is the drug of choice for children over 2 months of age because it is both safe and effective when given in a correct dose. When the dose exceeds 150 mg/kg, however, toxic aryl intermediates may accumulate and cause liver damage. It is important to keep paracetamol tablets out of the reach of children and not to provide 500 mg adult tablets for infants.

Aspirin is **not** recommended for use in children. Its use in viral infections (mainly influenza and varicella) can cause Reye's syndrome, a rare but potentially fatal condition affecting the liver and brain. In addition it damages the gastric mucosa, which may lead to gastric ulcer, haemorrhage and perforation. Aspirin also increases the risk of serious bleeding in children with dengue fever or other haemorrhagic disorders. Overdose causes salicylism, which is characterized by hyperventilation, reduced consciousness and severe metabolic acidosis.

Other measures. Fever causes increased water loss by fast breathing and sweating, which may lead to dehydration. Dehydration should be avoided by encouraging the child to drink extra fluids. Children who are dehydrated from diarrhoea are frequently febrile. They should be rehydrated appropriately, which will often cause the fever to disappear. Sponging with tepid water is effective, especially when combined with paracetamol, but is rarely required.

Sponging with tepid water is appropriate, however, for young infants (<2 months) because their metabolism of antipyretic drugs is prolonged and not entirely predictable. A child with severe hyperthermia (>42 °C) should be cooled initially with a cold water (or ice water) bath to reduce the body temperature rapidly. An antipyretic drug should also be given.

Management of anaemia

Anaemia is a reduction in the number of red blood cells (RBCs) in blood, in their average size, or in their content of haemoglobin, the oxygen-transporting protein. Anaemia causes pallor of the conjunctivae, oral mucous membranes and palms of the hands. In blood, both the haematocrit (packed cell volume) and haemoglobin concentration are reduced. The most important consequence of anaemia is reduced capacity for transport of oxygen by the blood to critical organs, such as the brain and heart. When anaemia is severe it can cause high-output heart failure, lactic acidosis, or both. When either of these occurs the risk of death is high.

Types of anaemia

The most common types of anaemia are shown in **Table 11-3**. Among these the most important are haemolytic anaemia (due to malaria) and iron deficiency (due to blood loss or insufficient dietary intake of iron). These two types of anaemia often occur together.

- **Haemolysis** causes anaemia by the premature destruction of RBCs, so that their half-life is severely shortened and production of new RBCs is not sufficient to replace them. In malaria, haemolysis occurs when the parasites mature within RBCs and destroy them as they re-enter the blood. When a large population of parasites matures simultaneously, many RBCs are lysed and the haemoglobin level may drop rapidly, sometimes to life-threatening levels. *P. falciparum* malaria is most likely to cause severe anaemia.
- **Iron-deficiency** impairs the production of RBCs, causing them to be small (microcytic) and to have a reduced content of haemoglobin (hypochromic). The most common causes are chronic blood loss, as occurs, for example, in hookworm infestation. Insufficient dietary intake of iron often adds to the problem. Infants who are exclusively breastfed beyond 6 months of age may develop anaemia owing to the low content of iron in breast milk.

Table 11-3 Common types of anaemia

Type of anaemia	Cause	Features
Iron deficiency	Decreased iron intake, especially in exclusively breastfed infants; chronic blood loss from hookworm or whipworm	Hypochromic, microcytic RBCs
Malaria	Increased RBC destruction, especially in *P. falciparum* malaria	Haemolysis of parasitized RBCs. Haemoglobin level may drop rapidly (in hours).
Folate deficiency	Destruction of folate by excessive cooking of vegetables; persistent or frequent diarrhoea; protein-energy malnutrition	Large RBCs (macrocytes) with immature forms in blood smear.
Sickle-cell disease; thalassaemias	Genetic defect in synthesis of haemoglobin.	Haemolytic anaemia; distorted RBCs (sickle cells); painful crises (sickle-cell disease); hypochromic, microcytic RBCs (thalassaemias)

Management of non-severe and severe anaemia

About 50% of infants and young children in developing countries are anaemic and 1–2% are severely anaemic.

Non-severe anaemia (haematocrit 12–27%; haemoglobin 4.1–9.3 g/dl)) is usually asymptomatic. It is important that it be detected and that both the anaemia and its underlying cause be treated so that development of severe anaemia can be prevented. Where iron deficiency is common, treatment of anaemia is with oral iron and folate for one to three months. Mebendazole should be given for possible hookworm or whipworm infestation. In malaria-endemic areas, or if malaria is diagnosed, antimalarial treatment should also be given. Supplemental folate should **not** be given during treatment of malaria with a folate antagonist, such as sulfadoxine-pyrimethamine.

Severe anaemia (haematocrit ≤12%; haemoglobin ≤4 g/dl) can cause high-output heart failure or lactic acidosis, either of which can be rapidly fatal. Heart failure may occur when the cardiac output increases in an attempt to maintain adequate delivery of oxygen to essential organs. Lactic acidosis occurs when there is a significant switch to anaerobic metabolism owing to the lack of sufficient oxygen reaching vital organs; it is often precipitated by

physical stress or conditions that reduce blood volume, such as dehydration from fever or diarrhoea. In either condition, *prompt blood transfusion* is life-saving. When transfusion is delayed more than 24–48 hours, it is much less effective and mortality rates may reach 30–40%. Children in greatest need of urgent transfusion are those with a haemoglobin ≤4 g/dl ***and*** rapid deep breathing and altered consciousness (suggestive of lactic acidosis) or distended jugular veins (suggestive of heart failure). Concurrent conditions, such as dehydration or serious infection, must also be treated. Children with a haemoglobin >4 g/dl who do not have these clinical signs are at lower risk of dying and may be treated with anti-malarials and iron, especially if it is not possible to provide blood that is known to be HIV-negative. Such children should, however, be observed carefully to ensure their condition does not deteriorate.

Oxygen therapy

Relation of hypoxaemia to risk of mortality

The oxygen saturation of arterial blood (SaO_2) measures the availability of oxygen to support vital functions of the nervous, cardiovascular and other essential systems. At sea level the SaO_2 of healthy infants and children is about 97–100%; this falls to about 89% at 1600 metres elevation, and to about 80% at 4000 metres. When desaturated blood passes through the lung, from the pulmonary artery to the pulmonary vein, without becoming fully saturated, SaO_2 falls and hypoxaemia develops. This occurs in children with pneumonia, bronchiolitis and asthma because the blood continues to flow through poorly ventilated portions of the lung. In general, the reduction in SaO_2 parallels the severity of lung disease; reductions in SaO_2 are also somewhat greater in sick children living at high altitude.

The importance of hypoxaemia is that it correlates with, and may directly contribute to, an increased risk of death from ALRI, bronchiolitis or severe asthma. Studies in Kenya, Zambia and Gambia show that children with a diagnosis of ALRI and SaO_2 values <90–92% have an estimated 1.4 to 4.6-fold increased risk of dying compared to children with ALRI and higher SaO_2 levels. This is the basis for recommending that supplemental oxygen be given when oxygen desaturation is recognized. It should be made clear, however, that there are no controlled clinical trials that assess the therapeutic benefit of supplemental oxygen for children with ALRI.

Detection of hypoxaemia and priority for treatment with oxygen

Oxygen desaturation can be diagnosed by clinical signs or by measurement of SaO_2 using a pulse oximeter. The clinical signs of severe oxygen desaturation include:

- *central cyanosis*, or
- *inability to drink or feed*, when this is due to respiratory distress.

Children with either of these signs should have priority for receiving supplemental oxygen. The signs of less severe desaturation include:

- *severe lower chest wall indrawing*,
- *respiratory rate* ≥*70/minute*,
- *grunting* with every breath (in infants <2 months of age), and
- *head nodding* with each breath.

When supplies permit, children with any of these signs, but who lack signs of severe hypoxaemia, should also receive supplemental oxygen.

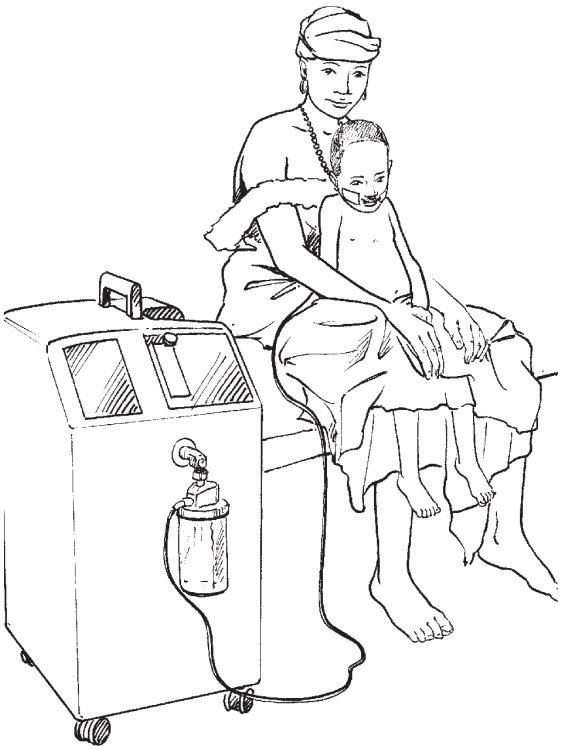
The proportion of children shown to have oxygen desaturation is increased when SaO_2 is measured using a pulse oximeter. This is a simple, non-invasive device that accurately measures SaO_2 in blood flowing through a child's finger. If a pulse oximeter is available, it should be used routinely to assess SaO_2 in all children who are hospitalized with a diagnosis of ALRI or other causes of respiratory distress. Because of the relationship between oxygen desaturation and risk of death from ALRI, it is recommended that, *if possible*, all hospitalized children with measured oxygen desaturation should receive supplemental oxygen.

Sources of oxygen to treat hypoxaemia

There are two possible sources of oxygen: oxygen-filled cylinders and oxygen concentrators:

- **Oxygen concentrators** work by pumping room air through a zeolite canister to remove nitrogen, thus concentrating the oxygen. The device is of moderate cost, requires little maintenance, and, once purchased, produces oxygen continuously at low cost. A continuous electrical supply is required, however, to operate the pump.
- **Oxygen cylinders** are easy to use, requiring only a flow meter and appropriate tubing, and can operate even when there is no electrical supply. The oxygen in cylinders is, however, relatively expensive and maintaining a constant supply is often difficult, especially at peripheral hospitals and health centres.

Fig. 11-4 Delivery of oxygen to a child from an oxygen concentrator



Giving oxygen

Oxygen may be delivered through nasal prongs, a nasal catheter or a nasopharyngeal catheter. All are effective. Nasal prongs are the safest because they avoid the risk of airway obstruction; they are preferred for use in small hospitals in developing countries. Nasal and nasopharyngeal catheters are cheaper and more likely to be available, but need close observation to detect and correct possible obstruction of the airway. Only nasal prongs should be used for children with croup. Face masks or head boxes do not deliver oxygen effectively and should not be used. With all methods it is important to check regularly that the prongs or catheter are positioned properly, and that oxygen is being delivered at the correct rate of flow and without leaks. The prongs or catheter should be cleaned at least twice a day.

Further reading

(1) Feeding

American Academy of Paediatrics. Breastfeeding and the use of human milk: American Academy of Paediatrics Working Group on Breastfeeding. *Paediatrics* 1997; **100**:1035–9.

Anonymous. HIV and infant feeding: a policy statement developed collaboratively by UNAIDS, WHO and UNICEF, 1997. *Breastfeeding Rev* 1999; **7**:14.

Brown KH, Gastanaduy AS, Saavedra JM, Lembcke J, Rivas D, Robertson AD, Yolken R, Sack RB. Effect of continued oral feeding on clinical and nutritional outcomes of acute diarrhoea in children. *J. Paediatr* 1988; **112**:191–200.

Mahmood DA, Feachem RG, Huttly SR. Infant feeding and risk of severe diarrhoea in Basrah city, Iraq: a case-control study. *Bull WHO* 1989; **67**:701–6.

Mbori-Ngacha D, Nduati R, John G, Reilly M, Richardson B, Mwatha A, Ndinya-Achola J, Bwayo J, Kreiss J. Morbidity and mortality in breastfed and formula-fed infants of HIV-1-infected women: A randomized clinical trial. *JAMA* 2001; **286**:2413–20.

WHO Collaborative Study Team on the Role of Breastfeeding on the Prevention of Infant Mortality. Effect of breastfeeding on infant and child mortality due to infectious diseases in less developed countries: a pooled analysis. *Lancet* 2000; **355**:451–5.

World Health Organization. *Complementary feeding of young children in developing countries: A review of current scientific knowledge*. Geneva, 1998 (WHO/NUT/98.1)

World Health Organization, Joint United Nations Programme on HIV/AIDS (UNAIDS). HIV and infant feeding: an interim statement. *Paediatr AIDS & HIV Infec* 1996; **7**:476–7.

World Health Organization. *Readings on diarrhoea. Student manual*. Geneva, 1991.

(2) Fluid management

Duke T. Fluid management of bacterial meningitis in developing countries. *Arch Dis Child* 1998; **79**(2):181-185.

Watkins SL. The basics of fluid and electrolyte therapy. *Paediatr Annals* 1995; **24**:16–22.

World Health Organization. *The treatment of diarrhoea. A manual for physicians and other senior health workers*. Geneva, 1995 (WHO/CDR/95.3)

(3) Fever

Kluger MJ. Fever: role of pyrogens and cryogens. *Physiol Rev* 1991; **71**:93–127.

Lesko SM, Mitchell AA. The safety of acetaminophen and ibuprofen among children younger than two years old. *Paediatrics*. 1999; **104**:e39.

Mahar AF, Allen SJ, Milligan P. Tepid sponging to reduce temperature in febrile children in a tropical climate. *Clin Paediatr* 1994; **33**:227–31

Russell FM, Shann F, Curtis N, Mulholland K. Evidence on the use of paracetamol in febrile children. *Bull World Health Organ* 2003; **81**(5):367-372.

Togawa T. Body temperature measurement. *Clin Physic Physiol Measurements* 1985; **6**:83–108.

Waldman RJ, Hall WN, McGee H, Van Amburg G. Aspirin as a risk factor for Reye's syndrome. *JAMA* 1982; **247**:3089–94.

World Health Organization. *The management of fever in young children with acute respiratory infections in developing countries*. Geneva, 1993 (WHO/ARI/93.30)

(4) Anaemia

Allen SJ, O'Donnell A, Alexander ND, Clegg JB. Severe malaria in children in Papua New Guinea. *Quart J Med* 1996; **89**:779–88.

Domellof M, Cohen RJ, Dewey KG, Hernell O, Rivera LL, Lonnerdal B. Iron supplementation of breast-fed Honduran and Swedish infants from 4 to 9 months of age. *J Paediatr* 2001; **138**:679–87.

English M, Muambi B, Mithwani S, Marsh K. Lactic acidosis and oxygen debt in African children with severe anaemia. *Quart J Med* 1997; **90**:563–9.

English M. Life-threatening severe malarial anaemia. *Trans Roy Soc Tropl Med Hyg* 2000; **94**:585–8.

Lackritz EM, Campbell CC, Ruebush TK, Hightower AW, Wakube W, Steketee RW, Were RB. Effect of blood transfusion on survival of children in a Kenyan hospital. *Lancet* 1992; **340**:524–8.

Marsh K, Forster D, Waruiru C, Mwangi I, Winstanley M, Marsh V, Newton C, Winstanley P, Warn P, Peshu N, Pasvol G, Snow R. Life-threatening malaria in African children: clinical spectrum and simplified prognostic criteria. *New Engl J Med* 1995; **332**:1399–1404.

van Hensbroek MB, Morris-Jones S, Meisner S, Jaffar S, Bayo L, Dackour R, Phillips C, Greenwood BM. Iron, but not folic acid, combined with effective antimalarial therapy promotes haematological recovery in African children after acute falciparum malaria. *Trans Roy Soc Tropl Med Hyg* 1995; **89**:672–6.

(5) Oxygen

Dobson MB. Oxygen concentrators and cylinders. *Int J Tub Lung Dis* 2001; **5**:520–3.

Duke T, Mgone J, Frank D. Hypoxaemia in children with severe pneumonia in Papua New Guinea. *Int J Tub Lung Dis* 2001, **5**:511–9.

Lozano JM. Epidemiology of hypoxaemia in children with acute lower respiratory infection. *Int J Tub Lung Dis* 2001, **5**:496–504.

Muhe L, Weber M. Oxygen delivery to children with hypoxaemia in small hospitals in developing countries. *Int J Tub Lung Dis* 2001; **5**:527–32.

Onynago FE, Steinhoff MC, Wafula EM, Wariua S, Musoia J, Kitonyi J. Hypoxaemia in young Kenyan children with acute lower respiratory infection. *Brit Med J* 1993; **306**:612–5.

Schnapp LM, Cohen NH. Pulse oximetry: uses and abuses. *Chest* 1990; **98**:1244–50

Smyth A, Carty H, Hart CA. Clinical predictors of hypoxaemia in children with pneumonia. *Ann Tropl Paediatr* 1988; **18**:31–40.

Usen S, Weber M. Clinical signs of hypoxaemia in children with acute lower respiratory infection: indicators for oxygen therapy. *Int J Tub Lung Dis* 2001, **5**:505–10.

Weber MW, Palmer A, Oparaugo A, Mulholland EK. Comparison of nasal prongs and nasopharyngeal catheter for the delivery of oxygen in children with hypoxemia because of a lower respiratory tract infection. *J Pediatr* 1995; **127**(3):378-383.

WHO. *Oxygen therapy for acute respiratory infections in young children in in developing countries*. Geneva: WHO/ARI/93.28, 1993. http://www.who.int/child-adolescent-health/New_Publications/CHILD_HEALTH/WHO_ARI_93.28.htm

World Health Organization. *Acute respiratory infections in children: Case management in small hospitals in developing countries. A manual for doctors and other senior health workers*. Geneva, 1990. (WHO/ARI/90.5)

Questions

11.1 Feeding should ALWAYS be continued during illness in children: list the benefits of continued feeding and list the only three exceptions to this rule.

11.2 List six ways of achieving nutritional objectives for the sick child.

11.3 List some advantages of exclusive breastfeeding in the first 6 months of life.

11.4 Describe the concept of internal fluid losses and explain when these can occur.

11.5 Give the most appropriate replacement fluids for the following: a febrile child, a child with diarrhoea, a child with persistent vomiting, and a child with shock or burns.

11.6 Explain how the normal core temperature is controlled and how it is affected by infection and antipyretic drugs.

11.7 Describe the possible physiological and pathological effects of high fever.

11.8 Describe the four main types of anaemia and their causes.

11.9 Describe the main pathological effects of severe anaemia.

11.10 Describe how commonly hypoxaemia is found in acute respiratory infections and explain how it occurs.

11.11 Describe the clinical signs of hypoxaemia in young children.

12. Monitoring progress of the sick child

Monitoring the clinical course of a sick child is an essential element in the treatment process. All hospitals should have a system for the regular monitoring of patients, which includes recording essential clinical information and ensuring that action to change treatment is taken promptly when this is required.

See also: ***Management of the Child with a Serious Infection or Severe Malnutrition*, Chapter 10, Page 113**

Introduction

All illnesses change over time, and acute illnesses often change rapidly. Change includes the appearance of new or more severe signs or symptoms as the illness worsens, or as complications develop. It also includes the lessening or disappearance of signs or symptoms as the illness subsides and the child recovers. To provide effective care it is *essential* that change in the pattern of illness be rapidly recognized and appropriate action taken. Such change may lead to a revised or more precise diagnosis, a need for new diagnostic tests, a different treatment, or a higher level of supportive care. It may also provide reassurance that the child is recovering satisfactorily, that the diagnosis is correct and that the treatment being given is appropriate; it will also assist in planning for discharge.

Detecting change requires careful *monitoring* throughout the child's stay in hospital. This involves frequent bedside observation and assessment of the sick child to determine the course of the illness over time. When monitoring is done correctly, and appropriate actions are taken, complications – including the risk of death – are reduced, improvement is hastened, and hospital stays are shortened. Unfortunately, monitoring the progress of sick children in developing countries is often inadequate. A recent review of hospital care suggests that this inadequacy contributes substantially to the high mortality rates seen in some hospitals.

All hospitals should have a system for regular patient monitoring, for more comprehensive monitoring of children who are most seriously ill, for recording essential clinical information, and for ***ensuring that appropriate action to change the treatment or to perform additional tests is taken promptly***.

Establishing regular monitoring – where it is not already in place – requires that the staff should be personally convinced that it leads to improved care and an improved outcome for sick children. It may be helpful to introduce changes during a "test period", after which they are assessed and the staff can decide on the elements which are to be kept, modified or discarded. Learning from the positive experience of staff in other hospitals may also be helpful.

Effective monitoring requires that the reported information is reliable. Efforts to promote regular monitoring may tempt busy health workers to fabricate data rather than appear unable to complete their task. It is important for health workers to understand that it is better to record no observations, with an explanation why they could not be made, than to fabricate them.

Approach to monitoring a sick child

Effective monitoring is only possible when the responsible health workers fully understand what is the expected progress of the child – based on the diagnosis, complications that may arise and how to recognize them, possible adverse effects of the treatment, and how the recommended treatment should be given.

The *process of monitoring* involves regularly checking each child to ensure that progress is satisfactory, and to detect:

- Any new signs or symptoms, including signs or symptoms of complications
- Lack of improvement or a deterioration in the child's condition
- Any adverse effects of treatment
- Errors in the treatment (e.g. wrong IV fluid or an incorrect amount).

Monitoring visits should also be used as an opportunity for hospital staff to discuss the child's condition with the family and to gain additional information about the history of the illness.

All acutely ill children in hospital require monitoring at least once daily. Those who are very sick should be monitored more frequently and at a higher level (see below). Keeping such children in beds close to the nursing station makes frequent monitoring easier.

Monitoring consists of two elements: *basic monitoring*, which includes routine assessments and measurements made regularly on all children, and *disease-specific monitoring*, which is based on the individual child's diagnosis or problems.

Examples of *basic monitoring* are:

- Measurement of temperature, respiratory rate, pulse rate and body weight.
- Observation of, for example, the level of consciousness and activity, willingness to eat or breastfeed, the occurrence of seizures, vomiting or diarrhoea.
- Check on medications or IV fluids being given, and intake of food and fluids.

Examples of *disease-specific monitoring* are:

- For a child with watery diarrhoea: check on signs for dehydration.
- For a child with pneumonia: count the respirations, check on difficult breathing and central cyanosis, and perform pulse oximetry (if available).
- For a child with unexplained fever: check on signs of focal inflammation or tenderness, signs of pneumonia, neck stiffness, and skin rash.

These are not complete examples of disease-specific monitoring, but are intended to show how monitoring should focus on the problems the child may present.

A higher and more comprehensive *level of monitoring* should be carried out on children who are more seriously ill. For example, the respiratory rate should be monitored in all children with cough or difficult breathing; if, however, a child has obvious respiratory distress, the child should be assessed for central cyanosis and pulse oximetry should be performed, if possible.

Monitoring should involve *all staff* who take part in caring for the sick child. The role of nurses and assistants is especially important as they are the ones who see the child most frequently – during routine assessments, or while feeding the child, giving medications, and providing routine care. If deterioration in the child's condition is observed by any staff, it should be reported immediately to the doctor or nurse in charge so that necessary action can be taken.

Mothers and other caretakers can also play an important role in monitoring, especially in busy hospital wards. Health workers should explain to them what signs to watch for and encourage them to bring the problem promptly to their attention. This is especially helpful for monitoring IV infusions, nasogastric tubes or oxygen catheters. Involving the mother or caretaker enhances their understanding of the child's illness and treatment, and also helps to improve communication between them and the health workers.

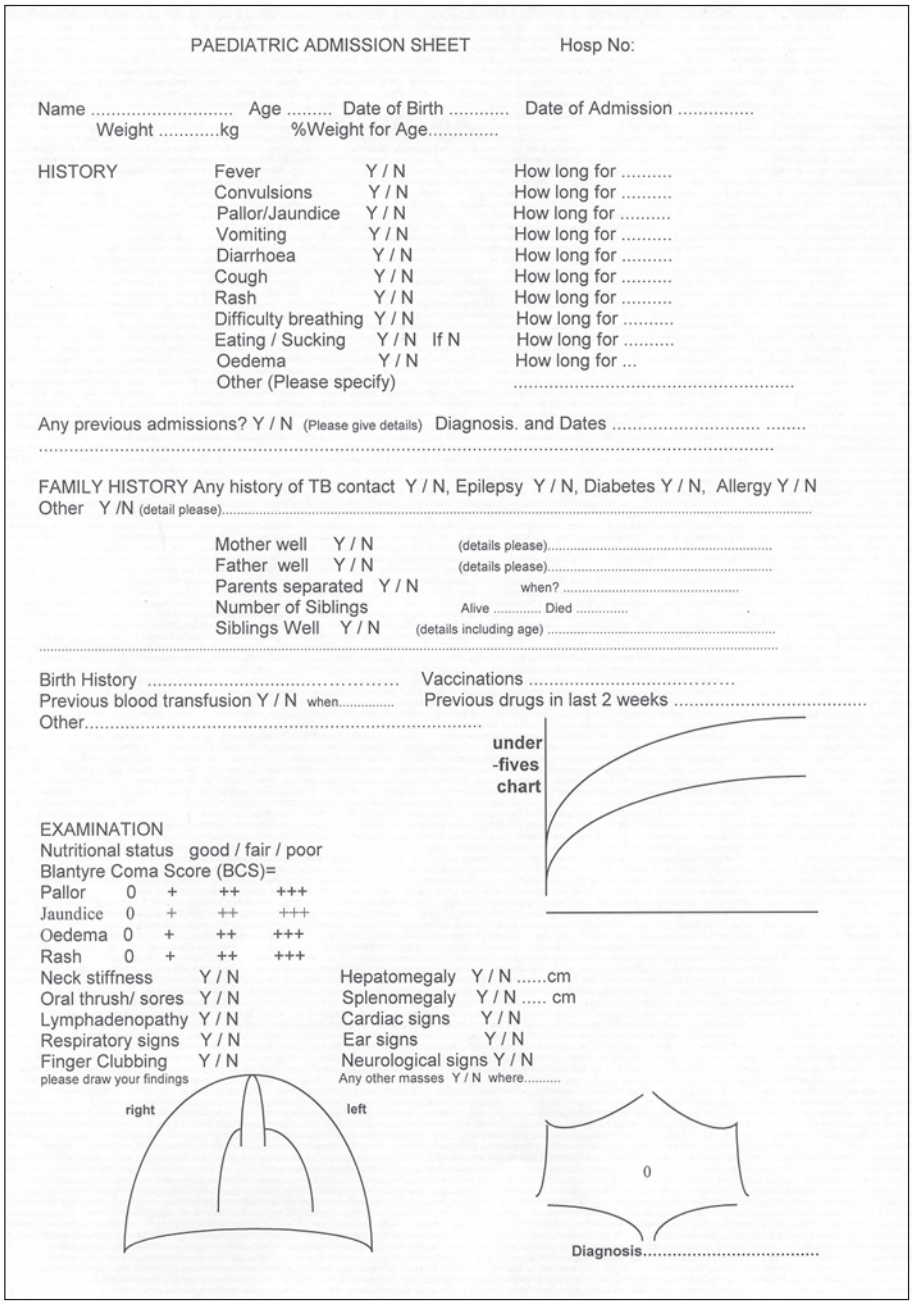
The *frequency of monitoring* depends on the severity and nature of the child's illness. All children should be assessed by the doctor or responsible caregiver at least once a day. Children who are seriously ill should have a monitoring visit soon after admission and several times daily thereafter, until the diagnosis is confirmed and their condition is stable or improving.

Recording of monitoring information

Information gained by monitoring should be recorded in the child's chart for use by caregivers when assessing the child's progress and to guide decision-making concerning further diagnostic tests or changes in treatment. One approach is to use an *admission form*, a *monitoring chart* and a *record of key findings and decisions* made during treatment in the hospital. Whatever system is used, it is important for it to be as simple as possible and require the minimum amount of time for adequate recording of findings so that staff are not diverted from other important clinical activities. Maintaining a *single set of records* that combines both medical and nursing notes is highly recommended.

The *admission form* should be used to record the history of the illness, the findings on physical examination, and any relevant laboratory studies. An example of an admission form is shown in **Fig. 12-1**. This form is designed to capture only the most essential information and to be easy to use. Diagrams are used, where possible, to record physical findings. The staff should be required to complete this form fully so that essential information is recorded on every admitted child. After the staff have become familiar with the form they will find they can easily and quickly access the information it contains.

A simple *monitoring chart* should be used to record the *basic monitoring information* for each child (**Fig. 12-2**). The chart shown as an example is easy to use; experience has shown that the time taken to complete this form can be less than required for traditional medical and nursing records. The



PAEDIATRIC ADMISSION SHEET Hosp No:

Name ..... Age ..... Date of Birth ..... Date of Admission .....
Weight .....kg %Weight for Age.....

HISTORY
Fever Y / N How long for .....
Convulsions Y / N How long for .....
Pallor/Jaundice Y / N How long for .....
Vomiting Y / N How long for .....
Diarrhoea Y / N How long for .....
Cough Y / N How long for .....
Rash Y / N How long for .....
Difficulty breathing Y / N How long for .....
Eating / Sucking Y / N If N How long for .....
Oedema Y / N How long for ...
Other (Please specify) .....

Any previous admissions? Y / N (Please give details) Diagnosis. and Dates .....
.....

FAMILY HISTORY Any history of TB contact Y / N, Epilepsy Y / N, Diabetes Y / N, Allergy Y / N
Other Y /N (detail please).....

Mother well Y / N (details please).....
Father well Y / N (details please).....
Parents separated Y / N when? .....
Number of Siblings Alive ..... Died .....
Siblings Well Y / N (details including age) .....
.....

Birth History ..... Vaccinations .....
Previous blood transfusion Y / N when..... Previous drugs in last 2 weeks .....
Other.....

under -fives chart

EXAMINATION
Nutritional status good / fair / poor
Blantyre Coma Score (BCS)=
Pallor 0 + ++ +++
Jaundice 0 + ++ +++
Oedema 0 + ++ +++
Rash 0 + ++ +++
Neck stiffness Y / N
Oral thrush/ sores Y / N
Lymphadenopathy Y / N
Respiratory signs Y / N
Finger Clubbing Y / N
Hepatomegaly Y / N .....cm
Splenomegaly Y / N ..... cm
Cardiac signs Y / N
Ear signs Y / N
Neurological signs Y / N
Any other masses Y / N where.....
please draw your findings
right
left
0
Diagnosis.....

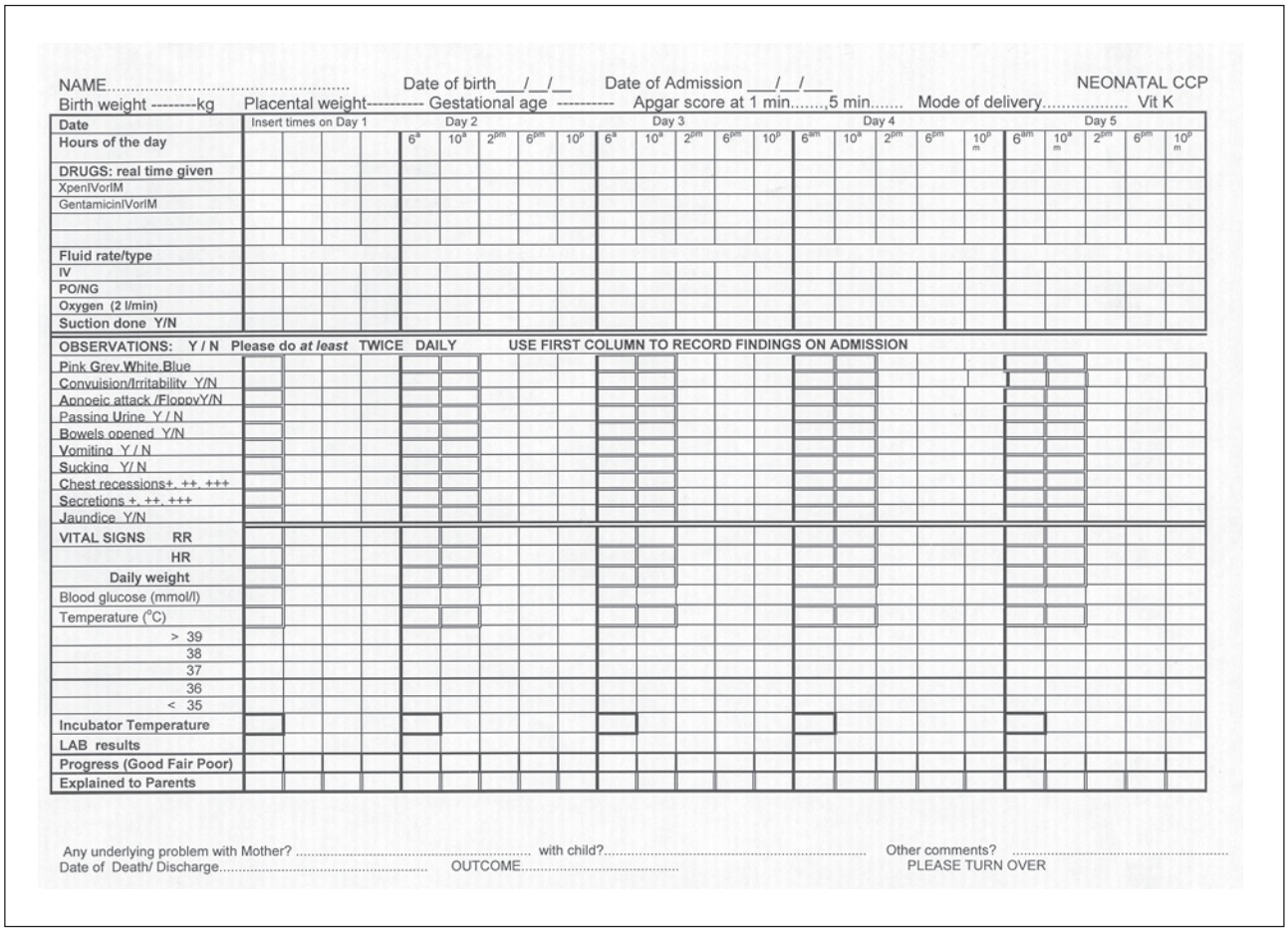
Fig. 12-1 Example of an admission form

chart is also efficient; by compressing essential information onto a single page, many parameters of clinical progress can be reviewed together quickly and the costs of paper are kept to a minimum.

The *record of key findings and decisions* is a blank, lined page on which the findings from disease-specific monitoring and laboratory or X-ray examinations are recorded by the responsible caregiver; decisions or actions regarding the diagnosis, treatment, and progress are described, and any complications are noted. Entries on this page should be brief and the dates given.

Regular review of the child using this 3-page chart helps to understand better the course of the child's illness and to identify problems that may require a change in treatment.

It is also helpful to print *standard guidelines* for the assessment or treatment of common conditions on the backs of the admission and monitoring charts. These might include assessment scales, such as a coma scale or criteria for classifying dehydration, and standard treatment regimens, including choice of medication and dose, for problems such as pneumonia, malaria, sepsis or dehydration. Ideally, medical and nursing staff should decide what information they would find most useful to be printed here.



NAME..... Date of birth___/__/__ Date of Admission ___/__/___ NEONATAL CCP

Birth weight --------kg Placental weight---------- Gestational age --------- Apgar score at 1 min.....,5 min..... Mode of delivery..... Vit K

Date	Insert times on Day 1				Day 2					Day 3					Day 4					Day 5				
Hours of the day					6^{a}	10^{a}	2^{pm}	6^{pm}	10^{p}	6^{a}	10^{a}	2^{pm}	6^{pm}	10^{p}	6^{am}	10^{a}	2^{pm}	6^{pm}	10^{p}_{m}	6^{am}	10^{a}_{m}	2^{pm}	6^{pm}	10^{p}_{m}
DRUGS: real time given																							
XpenIVorIM																							
GentamicinIVorIM																							
Fluid rate/type																							
IV																							
PO/NG																							
Oxygen (2 l/min)																							
Suction done Y/N																							
OBSERVATIONS: Y / N Please do *at least* TWICE DAILY	USE FIRST COLUMN TO RECORD FINDINGS ON ADMISSION																						
Pink Grey White Blue																							
Convulsion/Irritability Y/N																							
Apnoeic attack /FloppyY/N																							
Passing Urine Y / N																							
Bowels opened Y/N																							
Vomiting Y / N																							
Sucking Y/ N																							
Chest recessions+, ++, +++																							
Secretions +, ++, +++																							
Jaundice Y/N																							
VITAL SIGNS RR																							
HR																							
Daily weight																							
Blood glucose (mmol/l)																							
Temperature (°C)																							
> 39																							
38																							
37																							
36																							
< 35																							
Incubator Temperature																							
LAB results																							
Progress (Good Fair Poor)																							
Explained to Parents																							

Any underlying problem with Mother? ..... with child?..... Other comments? .....

Date of Death/ Discharge..... OUTCOME..... PLEASE TURN OVER

Fig. 12-2 Monitoring chart for paediatric admissions and monitoring the inpatient course of disease

Using information from monitoring to improve management of illness

Information gained during monitoring can be used to improve the management of a child's illness, for example:

- by helping to ensure that the prescribed treatments have been correctly given; the monitoring chart shows each treatment given to the child and should reveal whether a treatment was not given or was given incorrectly;
- by helping to recognize symptoms or signs as they appear, which will lead to a more precise diagnosis and, thus, to more effective treatment; for example, a child admitted with unexplained fever develops neck stiffness, which leads to a diagnosis of meningitis and treatment for this;
- by helping to recognize complications that require additional or revised treatment; for example, a child with pneumonia remains febrile longer than expected; this leads to further examinations that reveal an empyema.

It must be emphasized that the benefits of monitoring will only be realized when it is done regularly and thoroughly, the results are carefully recorded, the information is reviewed regularly by the responsible caregivers, and appropriate action is taken promptly, based on the findings.

Improving the quality of care by auditing

Regular, systematic review (auditing) of the outcomes of care in the hospital can substantially improve the quality of care for children who will be admitted in the future. Such a review could include:

- Examining case-fatality rates by diagnosis to identify trends over time; negative trends may point to specific problems that can be corrected;
- Reviewing with all staff the treatment of children who died in hospital; the goal should be to identify problems in the care of these children and suggest solutions;

- Comparing selected key aspects of care against a recognized standard, such as the WHO IMCI recommendations – for example, which antibiotic was used and what dose was prescribed for children with pneumonia. Use of the forms described above enables staff to record key clinical information on each child, which can be compared against the recommended standard practice during the audit.

All staff involved in giving care should take part in the audit. It should be emphasized that audits are not intended to assign blame for mistakes made, but to identify ways in which they can be avoided, and care improved in the future. They should be done in a positive atmosphere that encourages all staff to work together constructively.

Further reading

DeNicola LK, Kissoon N, Abram HS Jr, Sullivan KJ, Delgado-Corcoran C, Taylor C. Noninvasive monitoring in the paediatric intensive care unit. *Paediatric Clinics of North America* 2001; **48**:573–88.

Duke T, Tamburlini G, and the Paediatric Quality of Care Group. Improving the quality of paediatric care in peripheral hospitals in developing countries. *Arch Dis Child* 2003; **88**(7):563-565.

Mancey-Jones M, Brugha RF. Using perinatal audit to promote change: a review. *Health Policy and Planning* 1997; **12**:183–92.

Nolan T, Angos P, Cunha AJ, Muhe L, Qazi S, Simoes EA, Tamburlini G, Weber M, Pierce NF. Quality of hospital care for seriously ill children in less developed countries. *Lancet* 2001; **357**:106–10.

Scott D. Monitoring service development through audit. *Journal of Child Health Care* 2001; **5**:138–42.

World Health Organization. *Improving quality of paediatric care in small hospitals in developing countries. Report of a meeting. Geneva, 19–21 June 2000.* Geneva, 2001 (WHO/FCH/CAH/01.25)

Questions

12.1 List some reasons why monitoring a child's progress is an essential part of the management of a sick child.

12.2 List four things that monitoring should be able to detect.

12.3 Give some examples of how audit can help improve the quality of care.

13. Counselling and discharge from hospital

Studies of childhood deaths from acute illnesses in developing countries have shown that many children died after being in contact with the health services, and some shortly after discharge from hospital. Some of these deaths are preventable by giving attention to planning the discharge of the child. This section presents recommendations on when and how to discharge the child from hospital. Careful attention to monitoring the child's response to treatment in hospital and to correct discharge of the child are just as important as correct diagnosis and initial treatment of the child.

See also: ***Management of the Child with a Serious Infection or Severe Malnutrition*****, Chapter 11, Page 116**

Planning the discharge

The discharge process in all children should cover:

- Correct timing of the discharge from hospital
- Counselling the mother on the child's treatment and feeding at home
- Checking that the child's immunization status and record card are up-to-date
- Communication with the health worker who referred the child or who will be responsible for follow-up care of the child
- Clear guidance on when to return for follow-up care
- Assisting the family in obtaining special community support that may be required.

Carrying out the above procedures should:

- Ensure that the discharge is not too early, thereby avoiding the increased risk of preventable death or disability at home.
- Ensure that the discharge is not delayed too long. This will reduce the child's exposure to the risk of hospital-acquired infections. Long admissions block a hospital bed for another very sick child.
- Strengthen the links between the hospital and first-level health service that referred the sick child to the hospital.
- Help the families to provide proper follow-up care for their children at home.

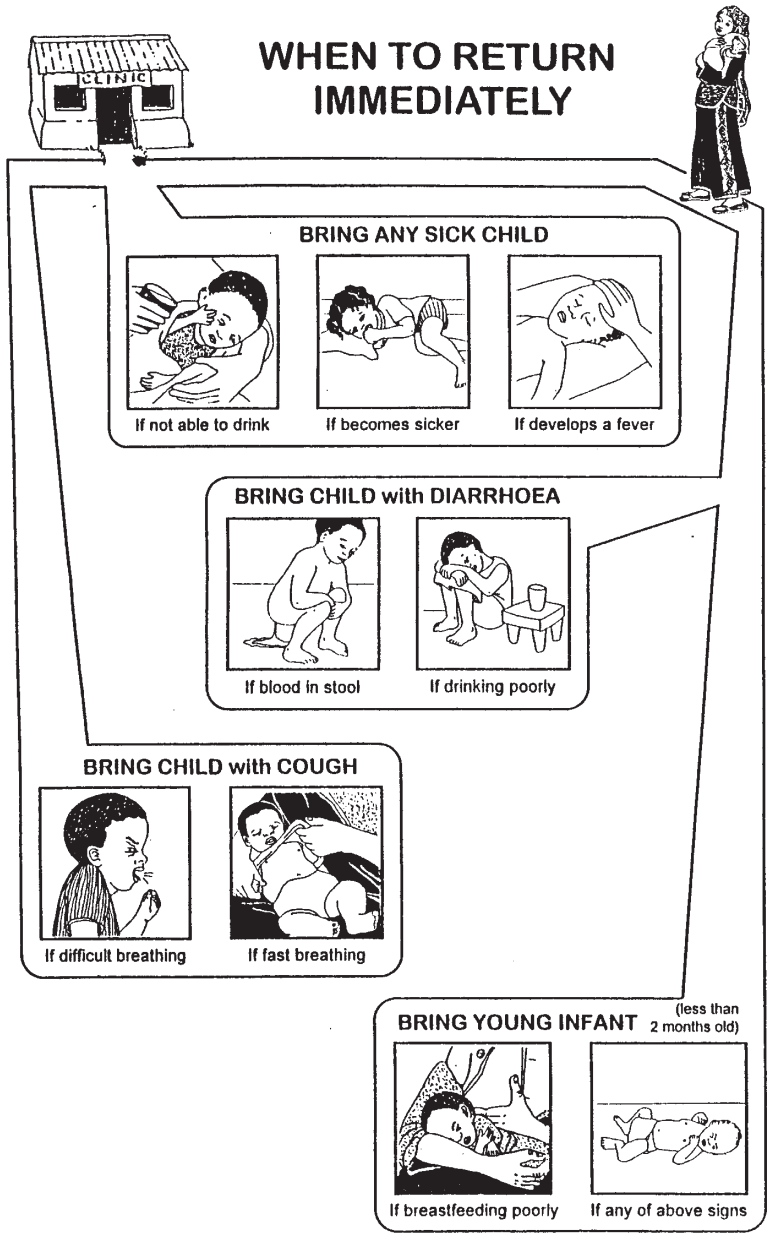
Timing of discharge from hospital

It is essential for the child to remain in hospital for treatments that are only available in the hospital (e.g. oxygen therapy, parenteral antibiotics or second-line antibiotics) or when careful observation and monitoring are needed. Admission to hospital also allows treatment that may be available in a health centre to be directly administered by better trained staff. Premature discharge of a very sick child could interrupt these treatments and greatly increase the risk of death. This needs to be balanced against the risk that a stay in hospital for longer than necessary may expose the child to serious infection acquired from other children, and will take up bed space and staff time for another acutely ill child. In general, once the child's clinical condition has improved markedly and oral treatment which can be given outside the hospital has been started, the child can be considered for discharge.

Decisions on the timing of discharge should be made on an individual basis and should take into consideration a number of factors including:

- The home circumstances of the family and level of support to care for the child.
- The staff's judgement of the likelihood that the remaining course of treatment will be completed at home.
- The staff's judgement of the likelihood that the family will return to the hospital if the child deteriorates.
- The nutritional status of the child, e.g. where there was severe malnutrition.

Fig. 13-1 Mothers Card giving counselling to mothers on the home management of illnesses and when to return immediately



Name: _____ M F Date of Birth: _____

Address: _____

Always bring this card with you to the clinic.

FLUIDS

FOR ANY SICK CHILD

- Breastfeed frequently.
- Increase fluid. Give soup, rice water, yoghurt drinks, or clean water.

FOR CHILD WITH DIARRHOEA

Giving more fluid can be lifesaving!

- Give these extra fluids, as much as the child will take:
  - ☐ ORS Solution
  - ☐ Food-based fluids, such as soup, rice water, yoghurt drinks
  - ☐ Clean water
- Breastfeed more frequently and longer at each feeding.
- Continue giving extra fluids until diarrhoea stops.

IMMUNIZATIONS (Record Date Given)

☐ BCG	☐ DPT 1	☐ DPT 2	☐ DPT 3	
☐ OPV 0	☐ OPV 1	☐ OPV 2	☐ OPV 3	☐ MEASLES

Return for next immunization on:

_____ _____

_____ _____

If the family removes the child prematurely against the advice of the hospital staff, every effort should be made to counsel the mother fully concerning treatment at home, to encourage her to attend for follow-up care, and to see that she contacts the local health worker for help in the follow-up of the child.

Counselling the mother

Key elements of good counselling include:

- Listening carefully to the mother's answers to your questions.
- Praising the mother for something helpful she has done.
- Giving advice, using simple language, that is relevant to the needs of the mother.
- Checking to see that the mother has understood what she has to do.

A *Mother's Card* (**Fig. 13-1**) can be given to each mother to help her remember the appropriate foods and fluids, and indicating when to return to the health worker. It is useful for several reasons:

- It will remind the staff of important points to cover when counselling mothers about foods and fluids and when to return for follow-up.
- It will remind the mother what to do when she gets home.
- The mother may show the card to other family members or neighbours, so that more people will learn the messages it contains.
- The mother will appreciate being given something during the visit.
- Multi-visit cards can be used as a record of treatments and immunizations given.

Nutrition counselling

The first step is to identify any *feeding problems* that have not fully resolved. The child's feeding should be compared with the recommendations for feeding a child of that age (see Manual, page 106) and any differences should be noted as feeding problems. Check for common problems such as:

- Difficulty in breastfeeding
- Use of a feeding bottle
- Lack of active feeding
- Not feeding well during illness.

Even when specific feeding problems are not found, all mothers should be advised on promoting breastfeeding, improving complementry feeding practices using locally available energy- and nutrient-rich foods, and giving nutritious snacks to children aged 2 years or older.

Local feeding recommendations should include details of locally appropriate energy- and nutrient-rich complementary foods. It is important that the kcal and protein content per 100 ml of these recommended foods have been calculated, that they are rich in micronutrients, and that they have been checked for acceptability.

Treating the child at home

If treatment has to be continued by the mother at home, it is very important to make sure that the mother has understood what she has to do and is able to give the child the recommended treatment. Mothers should be shown examples and be allowed to practise the instructions and ask questions, and they should be checked to confirm that they have understood.

Checking that the child's immunization status is up-to-date

Before discharge from hospital, the immunization status of all children should be checked against national recommendations to identify any immunizations that are required.

It is important to immunize all children, including those who are sick and malnourished, unless contraindicated. There are *only three contraindications* to immunization:

- Do not give BCG or yellow fever vaccine to a child with symptomatic HIV infection or AIDS.
- Do not give DPT-2 or -3 to a child who has had convulsions or shock within 3 days of the most recent dose.
- Do not give DPT to a child with recurrent convulsions or another active neurological disease of the central nervous system.

A child with asymptomatic HIV infection can be given BCG and yellow fever vaccinations.

Communicating with the first-level health worker

A hospital must inform the first-level health staff about the outcome of the hospital referral, so that they will know

Table 13-1 Immunization schedule for infants recommended by the Expanded Programme on Immunization

Vaccine		Age				
		Birth	6 weeks	10 weeks	14 weeks	9 months
BCG		x				
Oral polio		x[a]	x	x	x	
DPT			x	x	x	
Hepatitis B	Scheme A[b]	x	x		x	
	Scheme B[b]		x	x	x	
Haemophilus influenzae type b			x	x	x	
Yellow fever						x[c]
Measles						x[d]

[a] In polio-endemic countries

[b] Scheme A is recommended in countries where perinatal transmission of hepatitis B virus is frequent (e.g. in South-East Asia). Scheme B may be used in countries where perinatal transmission is less frequent (e.g. in sub-Saharan Africa).

[c] In countries where yellow fever poses a risk

[d] A second opportunity to receive a dose of measles vaccine should be provided for all children. This may be done either as part of the routine schedule or in a campaign.

whether their initial treatment prior to referral was correct. More importantly, follow-up supervision at home is not likely to take place and thus children who do not respond fully to treatment or who develop new complications will not be identified and treated appropriately. The first-level health worker who referred the child to hospital should therefore receive a summary about the care in hospital, which should include:

- The diagnosis/diagnoses
- Treatment(s) given (including length of stay in the hospital)
- The child's response to this treatment
- Instructions given to the mother on treatment and other care as well as follow-up
- Outstanding problems requiring follow-up (including immunizations).

Improving the hospital's feedback to first-level staff is likely to result, over a period of time, in more appropriate referrals to hospital and a better relationship with the community and first-level health staff.

Guidance on when the mother should return for follow-up care

Children who are discharged home after inpatient hospital treatment may need to have follow-up care for a number of reasons:

- To check that the treatment at home was actually given
- To check that the child's problem has resolved completely (if this had not already happened at the time of discharge)
- To check for delayed complications after the child has recovered (e.g. hearing loss or disability after meningitis)
- To check the child's nutritional status (e.g. follow-up of children who had been admitted with severe malnutrition).

The mother should be advised to return immediately if the child is not able to drink or breastfeed, or becomes more sick, or develops a fever, or shows signs of the illness returning after the treatment in hospital.

The mother should be reminded of the child's next visit for immunization and this date should be recorded on the Mother's Card or immunization record card.

Special community support

The family should be assisted in obtaining special support that may be required (e.g. aid for a disabled child; nutritional support for a severely malnourished child; and in the case of an HIV-positive child, links with AIDS community support organizations).

Questions

13.1 List six key elements of a correct discharge process.

13.2 List four advantages of a proper discharge procedure.

13.3 Discuss the correct timing of discharge of the child from hospital and list the factors influencing the timing of discharge.

13.4 List four key elements in counselling the mother.

13.5 List five important pieces of information to include in the discharge letter.

Key to find the answers to the questions

Chapter 1

1.1 What are the five main causes of death in young children in developing countries and what percentage of deaths do they account for? **page 1, column 1**

1.2 List the main risk factors which increase the incidence and severity of these diseases in childhood. **page 2, col. 1, para 2**

Chapter 2

2.1 List some of the common problems in the emergency care of sick children in developing countries. **page 5, col. 1**

2.2 Explain the meaning of A,B,C and D in the Advanced Paediatric Life Support (APLS) system. Describe the equivalent system in the WHO Emergency Triage and Assessment and Treatment (ETAT) approach. **page 5, col. 2**

Chapter 3

3.1 Give definitions for the following conditions: chronic cough, pneumonia, severe pneumonia, very severe pneumonia. **page 9, col. 1**

3.2 Describe the burden of disease due to respiratory infections in children in developing countries. **page 9, col. 2**

3.3 List the commonest causes of pneumonia and of wheeze in young children. **page 9, col. 2 bottom and page 10**

3.4 Describe an effective way to give oxygen therapy to children with hypoxia. **page 11, col. 2 middle**

3.5 Describe the pathophysiology of pulmonary tuberculosis in children. **page 11, col. 2 bottom**

Chapter 4

4.1 Give definitions for the following conditions: acute diarrhoea, persistent diarrhoea, and dysentery. **page 14, col. 1**

4.2 List the most important diarrhoeal pathogens in children. **page 14, Table 4-1**

4.3 Describe the clinical signs associated with increasing levels of fluid deficit. **page 14, col. 2 bottom and page 15**

4.4 Explain the mechanism by which oral rehydration works. **page 15, col. 2. para (ii)**

4.5 Describe the underlying causes and pathological findings in persistent diarrhoea. **page 16, col. 2 bottom**

Chapter 5

5.1 Describe the burden of disease due to malaria in young children. **page 18, col. 2**

5.2 Explain the mechanism by which malaria can cause severe pathology in vital organs. **page 19, col. 1**

5.3 Describe the signs of severe malaria. **page 19, col. 2 top**

5.4 Discuss the role of chloroquine, quinine and artesunate in the management of malaria in children. **page 19, col. 2 bottom and page 20**

5.5 Describe the management of anaemia in severe malaria. **page 20, col. 2 bottom**

Chapter 6

6.1 Describe the three main types of meningitis and the major microbiological causes in each category. **pages 22 and 23**

6.2 Describe the main CSF findings (appearance, cell count, protein content and glucose level) in each type of meningitis. **page 24, Table 6-1**

6.3 Explain why correct fluid management is important in children with meningitis and describe the main elements of fluid management. **page 24, col. 1 bottom**

6.4 List three long-term sequelae of meningitis and describe how they may present. **page 24, col. 2 bottom**

6.5 List the main causes of meningitis in children with HIV infection and describe how the treatment differs in these children. **page 25, col. 1**

Chapter 7

7.1 Describe the pathological effects of measles virus infection. **page 26, col. 1 bottom**

7.2 Describe the time sequence of the course of infection – from infection with the virus through to the disappearance of the rash. **page 26, col. 2**

7.3 List the main complications of measles. **page 27, col. 1**

7.4 Describe the key elements of nutritional support in the management of children with measles. **page 27, col. 2**

7.5 Describe the role of antibiotics in the management of the child with measles and explain the rationale for this approach. **page 27, col. 2 bottom**

Chapter 8

8.1 List the most important neonatal infections, in order of their importance, as causes of death in developing countries. **page 29, Table 8-1**

8.2 List the most important bacterial causes of sepsis in the young infant. **page 30, Table 8-2**

8.3 Describe the three main aspects of supportive care in the sick young infant. **page 30, col. 2 bottom**

8.4 Describe the management of skin infections in the young infant. **page 31, col. 2**

8.5 List the main microbiological causes of and risk factors for ophthalmia neonatorum. **page 31, col. 2**

Chapter 9

9.1 Define severe malnutrition and describe the three main types of presentation. **page 33, col. 1 bottom**

9.2 Describe ways in which poverty and social disadvantage can lead to severe malnutrition. **page 34, Fig 9-3**

9.3 Describe the effects of severe reduction in food intake on four tissues or organs (reductive adaptation). **page 34, col. 2**

9.4 List the main elements of each of the two phases in the management of severe malnutrition and describe their timing. **page 35, Fig 9-4**

9.5 Explain why hypoglycaemia and hypothermia are common in severely malnourished children, and describe hospital practices which place the child at risk of these complications. **page 35, col. 2 bottom and page 36, col. 1**

Chapter 10

10.1 Describe when mother-to-child transmission (MTCT) of HIV occurs and the risk factors for MTCT. **page 38, col. 2 bottom**

10.2 Explain the problems with the interpretation of HIV test results in young children. **page 38, col. 2 bottom and page 39**

10.3 Discuss the role of PCP (pneumocystis pneumonia) prophylaxis in infants and in young children. **page 39, col. 2 bottom**

10.4 Describe the three main approaches to reducing MTCT, which are promoted by WHO. **page 40, col. 1**

10.5 Discuss how you could decide the most appropriate feeding method for infants born in areas with a high prevalence of HIV infection. **page 40, col. 1 bottom**

Chapter 11

11.1 Feeding should ALWAYS be continued during illness in children: list the benefits of continued feeding and list the only three exceptions to this rule. **page 42, col. 2**

11.2 List six ways of achieving nutritional objectives for the sick child. **page 43, col. 1**

11.3 List some advantages of exclusive breastfeeding in the first 6 months. **page 43, col. 2 top**

11.4 Describe the concept of internal fluid losses and explain when these can occur. **page 45, col. 1 bottom**

11.5 Give the most appropriate replacement fluids for the following: a febrile child, a child with diarrhoea, a child with persistent vomiting, and a child with shock or burns. **page 45, Table 11-2**

11.6 Explain how the normal core temperature is controlled and how it is affected by infection and antipyretic drugs. **page 46, col. 1 top**

11.7 Describe the possible physiological and pathological effects of high fever. **page 46, col. 1 middle**

11.8 Describe the four main types of anaemia and their causes. **page 47, Table 11-3**

11.9 Describe the main pathological effects of severe anaemia. **page 47, col. 2 bottom**

11.10 Describe how commonly hypoxaemia is found in acute respiratory infections and explain how it occurs. **page 48, col. 1 bottom**

11.11 Describe the clinical signs of hypoxaemia in young children. **page 48, col. 2**

Chapter 12

12.1 List some reasons why monitoring a child's progress is an essential part of the management of a sick child.
page 51, col. 1

12.2 List four things that monitoring should be able to detect.
page 51, col. 2 bottom

12.3 Give some examples of how audit can help improve the quality of care. **page 54, col. 2**

Chapter 13

13.1 List six key elements of a correct discharge process.
page 56, col. 1

13.2 List four advantages of a proper discharge procedure.
page 56, col. 1 bottom

13.3 Discuss the correct timing of discharge of the child from hospital and list the factors influencing the timing of discharge. **page 56, col. 2**

13.4 List four key elements in counselling the mother.
page 58, col. 1

13.5 List five important pieces of information to include in the discharge letter. **page 59, col. 1 bottom**

Index