

# **A PAEDIATRIC HANDBOOK for Malawi**

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# PREFACE TO THIRD EDITION

In the ten years since the revised (second) edition of the Paediatric Handbook for Malawi many major changes have taken place in disease patterns and management of diseases in the country. It was therefore essential that the Handbook be thoroughly revised in every section, and not just reprinted to satisfy the demand for what is now the standard paediatric text for paramedics and nurses, and hopefully also very useful to doctors in Malawi. In preparing this edition we have maintained the general didactic approach of the first edition, although well aware there is room for differences in management of paediatric diseases even within Malawi, given the very varied situations and resources prevailing in different parts of the country. We have also tried to bring our text into line with current IMCI recommendations and to achieve compatibility with the protocols used in Queen Elizabeth Central Hospital paediatric department in Blantyre. In this we have been greatly helped by Professor Elizabeth Molyneux. Our thanks are also due to contributions from Dr Charles Mwansambo, head of the paediatric department in Kamuzu Central Hospital in Lilongwe.

We wanted to update the paediatric morbidity and mortality statistics for this edition, but unfortunately more up to date statistics were not available to us in the form we wanted. The weight and height curves for boys and girls from 2 to 18 years old, could have been updated, using more appropriate developing world figures available from WHO, but the changes in the graphs would have been relatively minor, and not worth the work involved in changing the diagrams, so we have stayed with those from earlier editions. We have also kept the original "Under 5s" weight chart, now used in the health passport, and all our original illustrations,

## PREFACE TO FIRST EDITION

This book aims to be a practical guide to paediatrics for hospital based Malawian clinicians, both clinical officers and junior doctors. Background information is included where it helps in understanding disease management. The book is, as far as possible, a consensus view of paediatric practice in Malawi.

**Section 1** is introductory, outlining the importance of paediatrics, paediatric morbidity and mortality, and existing paediatric health programmes in Malawi. Details of history taking, examination and normal growth and development are included as these form the basis of the care of sick children. **Section 2** deals with the presentation of neonatal problems and gives an approach to their diagnosis and management.

**Sections 3 and 4** cover children aged over 1 month, and should be used together. Section 3 gives a differential diagnosis of common disease presentations, and, where no precise diagnosis is possible, how they should be managed. Section 4 covers specific diseases, providing background information and management.

**Section 5** is an appendix for information not in earlier sections.

**Section 6** is a drug list with paediatric doses.

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# 1 INTRODUCTION

## 1.1 Introduction

### 1.1.1 Demographics

A large proportion of Malawi's population, 48 percent of nearly 13 million, were children under 15 in 2004 (Malawi Demographic and Health Survey, 2004; World Bank for population estimate),

Children, especially the very young, are more susceptible to illness and have a higher mortality than older age groups. Hospitals and other health facilities throughout Malawi are crowded with sick children. Illness and mistakes in treatment can potentially cause lifetime suffering or disability, and can compromise the nation's future.

According to UNICEF's State of the World's Children Report 2008 Malawi has made dramatic progress in child survival over the past few years. Child mortality declined 29% between 2000 and 2004. In addition the 2006 Multiple Indicator Cluster Survey (MICS) showed a fall in under 5 mortality from 154 to 118 per 1000 live births. The reasons for these improved statistics are multiple and multisectorial, including improving immunisation coverage, vitamin A supplementation, elimination of neonatal tetanus, malaria control activities (including insecticide treated bed nets) and access to safe drinking water (according to the 2006 MICS 75% of the population has access now to safe water) among other interventions.

Caution should be observed, however, in interpreting these statistics. The figures depend on questionnaires given to parents or carers about deaths of children, and clearly the families of parents who have died (importantly at present from AIDS related diseases) are likely to have worse statistics and cannot be fully assessed in such surveys. More accurate figures will only be available when there is registration of births (about to start in a pilot form) and deaths.

Previously the National Health Information System collected data on routine reporting of health facility in-patient and out-patient workloads. The tables on the following page give an indication of paediatric morbidity and mortality patterns (though the data are dated). A limited sample survey in 2006 showed the 3 leading U5 problems unchanged.

## 1 Introduction - Demographics

### Main outpatient diseases in children under 5 (1988)

<u>Disease</u>	<u>% of all new cases</u>
1. Malaria	35.8
2. Respiratory illnesses	18.2
3. Diarrhoeal diseases	6.9
4. Skin diseases	6.9
5. Inflammatory eye diseases	6.8
6. GI/abdominal disorders	4.8
7. Traumatic conditions	2.9
8. Malnutrition	2.6
9. Worm infestations	2.1
10. Disorders of nervous system	1.5
11. Others	11.5

### Main inpatient diseases in children under 5 (1988)

<u>Disease</u>	<u>% admissions</u>	<u>case-fatality rate</u>
----------------	---------------------	---------------------------

#### *Under 1*

1. Malaria	29.4	5.7
2. Anaemia	16.1	9.0
3. Pneumonia	12.8	11.1
4. Diarrhoeal diseases	8.3	10.2
5. Measles	6.3	12.0
6. Malnutrition	5.8	16.9
7. Perinatal diseases	3.8	29.4
8. Other causes	22.3	

#### *1-4 Years*

1. Malaria	30.5	6.5
2. Anaemia	10.4	8.8
3. Pneumonia	10.1	8.7
4. Measles	6.8	8.0
5. Diarrhoeal diseases	6.8	10.0
6. Malnutrition	3.2	17.8
7. Other causes	22.3	

Outreach clinics and community based health care programmes are essential if all children are to receive better medical attention when sick, and the burden of preventable disease is to be reduced. Increasing

emphasis is being placed by the Government of Malawi on primary health care (PHC), and child survival programmes.

In addition to endemic diseases, the nutritional status of many of the children in Malawi is poor. Household food security is a serious problem, and it has been estimated that 61% of rural children suffer from chronic malnutrition. The 2004 Demographic and Health Survey found no change in childhood malnutrition rates with 48% stunted, 22% underweight and 5% wasted in families with an adult head. Nutrition programmes (including vitamin A supplementation, which has been maintained at over 90% coverage for children under 1 year old), food-for-work schemes, multi-sectoral collaboration, and child spacing programmes are key strategies for tackling the problem.

Immunisation against measles has been a success story but is in danger of failing. An immunisation rate of 85% of children aged 1 to 2 against measles (2006 MICS study) is only borderline to protect against epidemics, and there is a significant risk of a return to the situation in the 1970s when measles outranked malaria as a cause of childhood death. Every opportunity of immunising children over 9 months old at underfive clinics must be used. In any measles outbreak, any susceptible children over 6 months old admitted to a health unit for any reason should be immunised.

### **1.1.2 IMCI**

Child and infant mortality have long been far too high in Malawi, but recently it has been realised that proven effective and inexpensive measures are available to change this. Malawi is responding through the Integrated Management of Childhood Illness (IMCI) approach. The first aim of IMCI is to provide health care workers with the necessary knowledge and skills to treat common childhood illnesses effectively and teach their carers how to prevent recurrences. The second is to ensure that essential drugs and supplies for preventing and treating childhood illnesses are consistently available. The third is to educate carers and their communities – increasingly important with the rise in number of orphans from the AIDS epidemic – in what they can do to keep children healthy, how they should handle illnesses themselves and when they should refer sick children to the health care system.

The main weaknesses of the programme are shortages of trained staff, and of essential drugs and materials, difficulties in travel and

communication, and insufficient emphasis in training staff on the need not to waste available resources and to put time and effort into teaching carers how to prevent illnesses recurring. An over elaboration of documents must not distract from these crucial practical issues.

In this edition of the Handbook we have tried to highlight for health workers those proven measures to reduce disease and death, the so called "high impact child survival interventions," that should contribute substantially to improving child health. The vitally important task of involving and educating the community in their role cannot be covered in this Handbook, but must not be ignored. This is why there is a major emphasis by the Ministry of Health on the use of health extension workers at grass roots level - Health Surveillance Assistants (HSAs).

## 1.2 History & Examination

### 1.2.1 The Paediatric History

A well taken history is worth much more than laboratory tests in the diagnosis and management of illness in children.

#### **Some Differences from Adult History Taking**

- \* Record from whom the history is taken, with an estimate of reliability.
- \* Details of the mother's pregnancy, delivery and the neonatal period are important for babies and toddlers.
- \* A developmental history - ages at which milestones like sitting, walking, first words are achieved - is very important to alert one to delayed development, an early indication of chronic illness.
- \* Immunisation history is very important, especially for children being admitted, to prevent cross infection with diseases like measles in the ward and to arrange immunisation where necessary.

#### **Hints on History Taking**

It is important not to have an aggressive approach, but to be gentle, kind and sympathetic, so as not to upset the child or guardian.

Do not be critical during history taking. Saying, for instance, "You should not have delayed so long before coming to hospital," during the history may antagonise or frighten the guardian, and prevent you from finding out important information.

Initially do not interrupt the guardian's complaint with questions. Save them for when the complaint has been completed.

Do not suggest complaints to the guardian or interpret the guardian's

words, but if necessary record actual words. On the other hand do not just literally translate what is said, but check by questioning exactly what is meant. For instance "mwana anakomoka" may mean the child fainted, or had a fit with twitching or just was very weak. Exactly what was meant must be found out by more questions.

The way you ask questions is very important. Do not ask leading questions that give the guardian a clue of the answer you expect. So the question "Was the urine coca-cola colour?" should be avoided, and "What colour was the urine?" asked instead. Another way of asking for information is to phrase the question so that the answer expected seems to be the opposite of what you want. For instance if you want to know whether chest pain is worse on coughing, ask "Does coughing make the pain better?" Or if you want to know whether the cough is worse at night, ask "When is the cough worse, in the day, or at night?" and not "Is the cough worse at night?" Finally, in sensitive areas do not ask questions that sound critical. For instance "What kind of village medicine did you give the child?" is better than "Did you give the child village medicine?" even though that is the question you really want an answer to: the first wording makes it sound as if you are interested in the kind of medicine, whereas the second sounds as if you will criticise the guardian for giving village medicine.

When recording the history do not omit facts and answers that seem to contradict the diagnosis you favour. When taking the history you are gathering information. The stage of evaluating it and deciding what is accurate or relevant comes later.

When there are physical findings that do not fit with the history, be prepared to go back and check over the history carefully. For example, worried relatives sometimes forget the exact timing of events, or previous episodes of illness, or history of contact with TB patients. They may withhold vital information because of fear. The guardian giving the history may not have first hand knowledge of an event, and the correct story will emerge only when another relative, who was an eye witness, comes.

### **History Taking for the Beginner**

When you are starting to take histories, follow a standard pattern to ensure you do not miss out important points. Only when you have mastered the technique of history taking and have a wide knowledge of



disease patterns in children should you take short cuts and ask selected questions. Of course in due time, with the numbers of children that come for medical care you will have to take quick histories, especially for minor complaints, but at the beginning do not assume you have enough knowledge to take short cuts, and practise taking full histories.

In certain emergency situations (e.g. children presenting with severe trauma, convulsions or severe dehydration or dyspnoea) you need to take a brief history initially, enough to allow you to start correct emergency treatment, leaving a detailed history until the situation is under control. Usually, however, you should follow a standard pattern as shown below.

### **Presenting complaint (PC)**

This is a brief summary of the main problem and its duration. Ask "Why have you brought the child to hospital?" or "What seems to be the matter with your child?" Record it as a short headline, with some assessment of the informant, e.g. Abdominal pain for 24 hours. (Informant grandmother; may be unreliable).

### **History of present illness (HPI)**

This is a detailed account of the presenting complaint - when it started, how (suddenly or gradually), location, severity, aggravating and relieving factors, associated symptoms, with the chronological order of their development. Record time by dates, or by numbers of days or hours before admission, NOT as "Monday" which is confusing a few weeks later when the history may be read again. A good way to check on the duration of illness is to find out when the child was last completely well (playing happily, going to school) and then lead the guardian through the development of the illness.

Record significant negative information, e.g. absence of constipation or vomiting in suspected intestinal obstruction.

Find out the details and timing of any treatment already given. This is especially important for malaria treatment when resistance to anti-malarials or antibiotics is a concern.

### **Review of systems**

This is a systematic review of the functioning of other organs or systems. You may find unexpected complications or unrelated disease that may affect management. Record only the positive findings.

#### **Ask about:**

- |      |                              |
|------|------------------------------|
| Eyes | - vision, discharge, redness |
| Nose | - discharge, bleeding        |

- Ears - hearing, discharge
- Mouth - sores, dental problems
- Throat - pain on swallowing
- Respiratory - cough, rapid breathing, dyspnoea, chest pain, wheeze
- Cardiovascular - similar symptoms to respiratory, oedema
- Gastrointestinal- appetite, nausea, vomiting, type and frequency of stool, abdominal pain
- Genito-urinary - urine colour, frequency of micturition, dysuria
- Neuromuscular - convulsions, tremor, weakness, limp, unsteadiness, speech
- Bones & joints - pain, swelling, limitation of movement
- Blood - pallor, bleeding, bruising
- Allergy - rashes, wheeze, oedema

### **Past History**

- Pregnancy - complications, drugs, HIV status
- Delivery - where (hospital or home),
  - maturity (term or preterm)
  - how delivered (vertex, breech, vacuum, caesarean)
  - birth weight and condition (resuscitation, cry, ability to suck)
- Neonatal history - cry, feeding, convulsions, cyanosis, pallor and jaundice
- Development & Growth - age when smiled at mother, sat without support, crawled, stood alone, walked, first spoke, weight curve in health passport
- Past illnesses - age at illness, symptoms, severity, complications, hospital admission, treatment, surgery, transfusions, drug allergy
- Immunisations - preferably look at health passport

### **Family and contact history**

- age, health, or cause and date of death of parents
- ask about consanguinity (are parents related e.g. cousins)
- list siblings (brothers and sisters) by age
- give health, or dates and causes of death
- include mother's abortions and stillbirths with gestations

## *1 Introduction - The Paediatric History and Examination*

- note especially contact with infections among other members of family and neighbours, visitors, lodgers.

### **Social history**

Home environment, marriage stability, parents' educational level, jobs and economic status (e.g. farming).

### **Summary of History**

Then record a brief summary of the main points in the history.

### **1.2.2 The Paediatric Examination**

Examination in the adult and older child can be by systems (i.e. cardiovascular, respiratory systems etc) or regional (i.e. hands, arms, head, neck, thorax, back, etc) and the methods are a combination of inspection, palpation, percussion and auscultation. The paediatric examination differs largely because of variable cooperation and emotional reaction of the child to the examination and examiner at different stages of development. Usually the adult comes seeking help (except in patients with nervous disorders or emotional disturbance), whereas the child often comes by a parent's decision and sometimes against his will. The child does not understand the procedure of the examination as an adult would, so is easily frightened.

It is important first to try to gain the trust of the child. In the very young baby this may not be necessary, but can be obtained by smiling and making friendly noises. From 6 months to a few years it is important not to rush at the child, but to give him or her some time to get used to you, while you are taking a history from the mother. Older children can be approached more directly, by using their first names in greetings or offering toys to attract attention.

At all ages it is very important to observe the child carefully first before approaching or touching him or her, as this may cause fear which can obscure all physical findings. Observe the child's apparent emotional state, posture and activity, relationship to mother or guardian, alertness and interest, colour, breathing, including respiratory rate, cough, any abnormalities of shape, such as abdominal distension, goitre, limb deformities, facial abnormalities, signs of dehydration etc.

The order of further parts of the examination, such as palpation of the pulse and abdomen, auscultation of the heart and lungs, inspection of the ears and throat and rectal examination should be partly dictated by

consideration of which findings are likely to be most important for diagnosis in that patient. These should be done before the child becomes upset, cries and is therefore hard to examine.

It is generally wise not to separate a younger child from the mother, but to examine the child on the mother's lap, rather than on an examination couch. Useful ways of getting round crying are to get the mother to breast feed, to distract the child with toys or lights, and to get the mother to hold a baby over her shoulder looking away from the examiner. In older children, demonstrate that apparently frightening examinations are not painful for the mother, and get her to chat to the child. Usually procedures like inspection of the ear drums, optic fundi, throat, and rectal examination, should not be performed until the end of the examination, and should be preceded by a warning that something uncomfortable is to be done.

Examining the nervous system is usually done by observing the child's behaviour, and special tricks may have to be used to decide on items such as neck stiffness, muscle tone, hearing, and reflexes.

### **Notes on specific signs by system**

#### ***Cardiovascular system***

Feel the radial, femoral and other pulses. Heart rate is often easier to count by auscultation. In small babies the jugular venous pressure (JVP) can be difficult to determine, and most important signs of heart failure are liver size, heart rate, dyspnoea, and triple rhythm. For auscultation of the heart, a baby may be quietened by getting the baby to breast feed, or in the absence of the mother, to suck the examiner's clean finger.

#### ***Respiratory system***

The respiratory rate is very important, provided the child is AT REST. Look closely for dyspnoea (nasal flaring, subcostal and intercostal indrawing and grunting breathing), and listen for audible wheeze and stridor. Percussion is specially useful in detecting effusion or empyema, though it requires practice and careful technique. Auscultation of the chest is often difficult to interpret, because of "transmitted noises" from the upper respiratory tract that may be heard widely across the chest. Reduced air entry is more important than various moist noises, and it is important to check whether or not crepitations persist after coughing or crying. The examination of ears and throat must not be forgotten, but should usually be done last, because it is frightening.

### ***Gastrointestinal system***

Examining the mouth, teeth and rectum should be left to the end. Inspection of the abdomen for distension, movement and peristalsis, should often be followed by palpation with the child sitting on the mother's lap. Older children should be examined lying on a bed or couch. Palpation for liver and spleen and masses needs to be gentle. Talking to the child to distract him or her while palpating is often very helpful in getting relaxation. The command "Relax!" is seldom useful.

### ***Nervous system***

Here careful observation is much more important than examination. Look for symmetry, freedom of movement or restriction in use of limbs during play. Look at posture, alertness and activity, and attention to visual and auditory stimuli.

Distracting the child may allow assessment of muscle tone, reflexes and neck stiffness. A useful way of testing whether neck flexion is free is to push the child down sharply from the sitting to the supine position, when usually the head will be flexed forward.

Whether the child can do certain things e.g. sit, stand and speak, is very useful in assessing developmental status. In infants palpation of the anterior fontanelle is important for assessment of intracranial pressure, or dehydration.

### ***Measurements***

- Weight should always be recorded.
- Head circumference is important in assessing neurological problems.
- Arm circumference can be helpful in malnutrition in children under 5 years old.
- Length or height is of value in nutritional and endocrine problems.

### ***Summary and assessment***

Finally give a brief account of the main significant points in the history and findings on examination. List your provisional and differential diagnoses, and end up with a plan of investigation and treatment.

## **1.3 Normal Growth and Development**

### **Definitions**

- Growth is increase in size.
- Development is maturation in form and function.

Understanding normal patterns is very important in paediatrics because this forms the basis for assessing all paediatric diseases.

### Growth

Patterns of growth (e.g. weight, height, head circumference) vary considerably [5.1]. The best known graph is the weight chart in the health passport [5.1.1] which you should refer to frequently when assessing normal growth in children.

The following table gives approximate values of AVERAGE normal growth which can be memorised.

AGE (Months)	0	3	6	9	12	18	24	36
Weight (kg)	BW	BW+4	BW+6	BW+8.5	BW+10.5			
Length (cm)	50				75		85	
Head circ. (cm)	35	40		45				50
Arm circ. (cm)			14					

### Development

**Maturation of Form** - this is less important. Examples are:

- age assessment from counting teeth (number of 1st teeth + 6 = age in months, and second set of teeth start at about six years of age)
- changing body proportions affect surface area calculations in burns

**Maturation of Function** - this is more important. Examples are:

- in the neonate maturation of kidney and liver function is a major consideration in managing fluid balance, problems of jaundice, and drug dosages.
- at puberty maturation of endocrine function and its effects are very significant.
- maturation of function of the nervous system determines the attainment of normal developmental milestones. Remember that African children may be more advanced in their motor milestones than the Caucasian children described in many paediatric textbooks.

## 1 Introduction - Development

### Developmental Milestones<sup>1</sup>

Developmental milestones can be divided into 4 categories:

Gross Motor - posture and body movement.

Fine motor - hand or finger manipulation.

Language and communication - vision and hearing.

Social adaptability - emotional reactions.

The following table lists milestones and indications for referral<sup>2</sup>

<u>Age</u>	<u>Social/language</u>	<u>Gross motor</u>	<u>Referral</u>
6 weeks	Responds to mother's voice Fixes eye contact Smiles	Head up	Extension of limbs Floppiness Doesn't smile Nystagmus Asymmetry of tone
6 months	Recognises faces Turns head to sound	Sits with support (from 4 months)	Floppiness Can't turn to sound Can't use both hands Squint Poor response to people
9 months	Shouts for attention	Crawls, sits unaided from 7 months	Can't sit Doesn't "babble"
18 months	Word sentences	Walks swinging arms Climbs chairs etc	Can't walk
24 months	Phrase sentences	Runs Climbs stairs	Can't understand simple commands

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<sup>1</sup> Reference book, "Development of the Infant and Young Child" by R.S. Illingworth, gives details of gestation assessment, growth graphs and developmental milestones.

<sup>2</sup> See section 3.16 for further details of developmental delay.

# 2 THE NEONATE

## 2.1 Introduction

### The Fetus

The fetus in utero is completely dependent on the mother for its bodily functions - mainly through the function of the placenta. At birth the neonate needs to develop independent function of its essential organs over a short space of time. The ease with which this change takes place can determine whether the baby dies, is abnormal, or normal.

Events that occur during and before delivery, including drugs given in labour and procedures done by the obstetrician or midwife, may damage or impair the function of immature organs. The first few minutes after birth are the most fragile period of life, and this contributes to the very high neonatal morbidity and mortality registered in Malawi.

### Some Changes That Occur at Birth

#### **Respiratory system**

Immediately after birth the brain initiates an inspiratory effort, and air enters the lungs which then expand - unless there is lack of surfactant, airway obstruction, hypoplastic lungs, diaphragmatic hernia, or severe chest wall abnormalities.

#### **Cardiovascular system**

The fetal heart is a two pump system. The right and left ventricles pump blood in parallel with blood from the two systems mixing at two levels; between the atria (through an atrial septal defect called the foramen ovale), and from the pulmonary trunk into the aorta (through the patent ductus arteriosus). After birth the circulation changes into two systems, with the left heart pumping blood to the rest of the body, while the right heart pumps blood to the lungs.

#### **Renal / urinary system**

Though the fetal kidneys function, at birth they have to take over from the placenta the excretion of waste products such as urea, as well as salt and water balance, and excretion of certain drugs. The ability of the kidneys to excrete substances and to concentrate urine correlates with the post-conceptual age, and not the postnatal age, and maturation to adult levels takes longer than a few days.



### Liver / hepatic system

After birth a number of chemical processes start occurring in the liver, particularly conjugation of bilirubin. The metabolism of most drugs will also take place in the liver, whose capacity to detoxify various drugs (e.g. chloramphenicol) is still limited in the neonatal period.

**Chloramphenicol is not the antibiotic of choice in the neonatal period: if you use it, be very careful with the dosage [section 6] as the neonatal liver metabolises it poorly and it can accumulate and give a state of shock known as “grey baby syndrome”.**

### Blood / haemopoietic system

There is a gradual change in the bone marrow production of fetal haemoglobin (which binds oxygen more strongly) to adult haemoglobin (which binds oxygen less strongly). The switch over appears to be based on post-conceptual age and is not related to the physical act of birth.

### Immune system

The neonate acquires some antibodies passively from its mother, but is quite limited in terms of producing its own antibodies, and has to depend for its defence mostly on white cell production and the mechanical barriers of its skin and mucosae. For these reasons there is special susceptibility to infections until about 3-6 months of age.

## 2.2 Examination of the Newborn

### 2.2.1 History Taking

Always take a careful history, which will differ from a routine paediatric history because of a greater need to know about antenatal and perinatal events. The history must explore three periods.

- Antenatal**      Include mother's age, gravidity, parity and previous pregnancy experience, last menstrual period, health during pregnancy, febrile illnesses, rashes, vaginal bleeding and drug intake. Ask about her HIV status.
- Perinatal**      Mode of delivery, complications, drugs during delivery, baby's condition at birth, especially time to cry and Apgar score if known, ability to suck.
- Postnatal**      Questions should include asking about maternal postnatal fever and breast feeding.

### 2.2.2 Examination

Like all paediatric examinations, this has to be "opportunistic" (so count breathing, listen for murmurs and palpate the abdomen while the baby is asleep, or breast feeding quietly). But examine from head to toe, so no gross abnormalities are missed.

**Head** Look particularly for hydrocephalus microcephaly, fontanelle size and fullness, facial features, shape of the eyes (upward slanting eyes and prominent epicanthic folds in Down's syndrome) and ears. Look in the mouth for cleft lip and palate and thrush. Check lips for cyanosis.

**Neck** Look for neck webbing (in Turner's syndrome), for swellings and goitre.

**Chest** Look at chest shape and for intercostal and subcostal retraction. Listen to lungs, especially for air entry, and heart for murmurs, when baby is quiet.

**Abdomen** Look for distension, scaphoid shape, umbilical hernia, and always carefully for umbilical sepsis or bleeding. Palpate the abdomen for masses especially kidneys, liver, spleen and bladder. Remember palpable kidneys and a liver palpable at 2-3 cm are not always abnormal at this age. Check for patency of the anus.

**Genitalia** In the male look for hypo- and epispadias. Examine the scrotum for testes; remember undescended testes are common in premature babies. Small hydrocoeles are fairly common and disappear spontaneously. In girls white vaginal discharge or even blood is not unusual in the first week of life, as a result of maternal or placental oestrogens. A skin tag or prolapse of vaginal mucosa is also common.

**CNS** The neurological exam can be limited at this stage to looking at the baby's alertness, posture at rest, and reaction to stimulation. Ensure that all limbs move well and check the Moro reflex (abduction and extension of the arms followed by flexion when the head is allowed to drop back a little way suddenly).

**Musculo-skeletal** Palpate the clavicles to rule out fractures and count all the digits. Look at the palms of the hands for a single transverse crease which may indicate Down's syndrome. In the foot look for club feet and extra digits. If club foot is present,

## 2 The Neonate - Examining the Newborn, Low Birth Weight Babies

try to decide if it is positional or structural. The positional type is secondary to muscle imbalance, often as a result of the position of the feet in utero, and is correctable by temporary splinting.

**Skin** Look for skin markings such as mongolian blue spots (buttocks and lower back), capillary naevi (mainly face), erythema toxicum (yellow or white papules on an erythematous background, that resemble septic spots - these usually clear within 48 hours and are harmless). Milia are small white spots, most often on the face, and are quite normal in the neonate.

### Note:

There are a lot of misconceptions about what normal neonates can do: they are not blind, respond to sound and can feel pain, and therefore it is important that when examining them attempts are made to make eye contact and even to talk to them. This also encourages the mother to do the same.

In neonates yawning, hiccoughing and sneezing (if there is no purulent nasal discharge) do not indicate disease, whereas coughing, especially if associated with a respiratory rate over 60/minute, is abnormal and almost always a sign of pneumonia.

### 2.2.3 Low Birth Weight Babies

By definition low birth weight (LBW) babies weigh less than 2.5 kg at birth. LBW can be due to:

- Prematurity i.e. babies born at less than 37 weeks gestation (premature/ preterm),
- Dysmaturity i.e. babies who are born at term but are small for their gestational age (SGA),
- Combination, i.e. some babies are both premature and SGA.

SGA babies are defined as those whose weight falls below the 10th centile<sup>1</sup> for their gestation [5.1.2]. It is important to differentiate LBW as a result of prematurity from that due to retarded growth (SGA), because the two groups face different neonatal problems:

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<sup>1</sup> The 10th centile is the weight below which 10% of normal babies at that gestation are.

**Premature**

Respiratory distress syndrome  
Hypothermia  
Feeding Problems  
Jaundice  
Acquired infections

**SGA**

Hypoglycaemia  
Hypocalcaemia  
Meconium Aspiration  
Congenital Infections  
Chromosome abnormalities

In Malawi LBW is one of the most important causes of neonatal mortality. The underlying causes for LBW are probably multiple, including poor maternal nutrition, placental malaria infection, and intrauterine infection. The table below gives approximate 10th centile weights at various gestations - babies are SGA below these weights.

<b><u>Gestation (weeks)</u></b>	<b><u>10th Centile Weight (kg)</u></b>
38	2.2
36	1.9
34	1.5
32	1.25
30	1.0
28	0.8

**2.2.4 Practical Maturity Assessment**

In our nurseries, we rarely have accurate information about the mother's last menstrual period (LMP) to be able to calculate the length of gestation. Therefore, in order to be able to estimate the baby's gestational age various scoring systems are available using both neurological development and external features. The best known is the Dubowitz system. Unfortunately this is time consuming, but with experience a rapid assessment can be made.

Set out on the next page is a simplified guide to the range of appearances seen from the very immature premature baby to the term baby using appearances of genitalia, breasts, ears, skin, creases on the soles, and baby posture and tone (especially wrist and ankles).

<b>Feature</b>	<b>Very preterm</b>	<b>Preterm</b>	<b>Term</b>
<b>Creases on soles</b>	None	Few near toes	All over soles
<b>Genitalia</b>	Smooth empty scrotum Protruding labia minora	Scrotum has few rugae Labia minora equal to majora	Scrotum has many rugae Majora cover minora
<b>Breasts</b>	Faint, flat areolae	Nipple but no breast tissue	Breast tissue > 10 mm diameter
<b>Ears</b>	Flat soft pinna	Springy flat pinna	Edge curved all round
<b>Skin over abdomen</b>	Thin, red skin, visible veins	Pale skin, veins less visible	Thick, pale opaque skin
<b>Posture (on back)</b>	Limbs straight	Frog posture	Full flexion
<b>Flexion at wrists</b>	90° window	45° window	Full wrist flexion

## 2.3 Routine Neonatal Care

In Malawi careful attention to basic routine care in the following areas will achieve more valuable results than expensive sophisticated interventions. Priorities are resuscitation at birth, the establishment of breast feeding, keeping babies at the correct (warm) temperature, prevention and early treatment of infection, detection and early treatment of hypoglycaemia and good management of jaundice.

### 2.3.1 Resuscitation of The Newborn

#### General Principles

Most babies will have no problem at birth and all they need is a little cleaning of the mouth and nose, and thorough drying. The first essential in neonatal resuscitation is to anticipate problems and have the right equipment ready. In our circumstances available equipment will vary from centre to centre, but the basic essentials are a suctioning device and some kind of bagging system for the apnoeic baby. The exact procedure will obviously depend on the equipment available, but the sequence of priorities should not vary from the standard A, B, C, D.

**A AIRWAY** - gently clear mucus from nostrils and oropharynx with mucus extractor or suction machine set to low (taking care not to suction

near the larynx, which can cause reflex vagal slowing of the heart).

**B BREATHING** - establish breathing by using an Ambu bag if needed. Give oxygen if necessary.

**C CIRCULATION** - If the heart beat after A and B is below 60/minute, start cardiac massage.

**D DRUGS** - If still no response after ventilation and cardiac massage, give IV adrenaline (0.1 ml/kg of 1:1000), IM naloxone (0.1 mg/kg) (when indicated by use of pethidine in the mother in the previous 4 hours), and IV 5% dextrose (see below for detailed steps in resuscitation and drug doses).

### Steps in Resuscitating a Neonate

1 Place baby on a warm towel to keep warm and dry. Ensure a clear airway. Count the APGAR score at 1 and 5 minutes.

2 If after 1 minute the baby is not breathing well, stimulate by flicking abdomen or feet (take about 15 seconds to do this).

3 If still not breathing, give oxygen by mask (or catheter) and stimulate again.

4 If still not breathing or heart rate < 100/minute, give artificial respiration with bag and mask. (Or intubate, if skilled in this technique).

5 If heart rate is absent or still < 60/minute give cardiac massage.

6 If still not breathing well at 5 minutes, or heart rate remains < 100/minute, give drugs:

### Adequate ventilation in resuscitation is more important than drugs.

- NALOXONE (Narcan) 0.1 mg/kg IM, if mother had pethidine or the lytic cocktail.

- ADRENALINE 1:1000 (0.1 ml/kg) is given IV or as a few drops down the endotracheal tube.

- give 10%<sup>1</sup> dextrose 2-4 ml/kg slowly IV, or (if no venous access) by NGT. Avoid 50% dextrose boluses which can cause rebound hypoglycaemia. After the bolus, continue dextrose 5% IV or by NGT. Check blood glucose after 30 minutes and as required after treatment.

7 Discontinue resuscitation if no response after 20 minutes, and maternal sedation is not responsible for failure to respond to

<sup>1</sup> 10% dextrose is made by adding 10 ml of 50% dextrose to 90 ml of 5% dextrose, or half strength Darrow's dextrose solution, most conveniently in an infusion burette.

resuscitation, and pneumothorax has been excluded [2.4.3].

**CAUTION**

Even though the newborn is known to have a remarkable ability to survive birth asphyxia, it is important that any intervention does not make the problem worse by:

- Use of dangerous, unnecessary or ineffective drugs or treatments.
- Letting the baby become cold.
- Poor timing of, and bad decisions on resuscitative measures.
- Unskilful interventions.

**The Apgar Score**

The APGAR score is used to assess the need for resuscitation. This is usually scored at 1, 5 and 10 minutes. The maximum total score is 10. All babies have peripheral cyanosis at 1 min, therefore a score of 10 is not possible at 1 min. Most babies still have peripheral cyanosis even at 5 min. For the score to be reliable, an accurate timing of the baby's response to resuscitation is necessary. Dr Apgar devised this system to make sure "someone is looking at the baby".

	0	1	2
Appearance (Colour)	Blue, pale	Body pink Extremities blue	Completely pink
Pulse (Heart rate)	Absent	Below 100	Over 100
Grimace (response to stimulation)	No response	Grimace	Cry
Activity (Muscle tone)	Limp	Some flexion in extremities	Active movements
Respirations (Resp. effort)	Absent	Slow, irregular	Strong cry

The Apgar score will give an indication of which babies need special attention. In general terms:

- Babies with 5 min Apgar scores of 6 and above need only cord care and wrapping to keep them warm.
- Those babies with Apgar scores less than 6 at 5 minutes need intervention or observation, depending on their respiratory and cardiac status.

- Babies with a low Apgar score at 5 minutes, but who are vigorous and well at 10 minutes do not need to be admitted to the nursery.

### **Resuscitation of Premature Babies**

The rationale and procedures are the same as for the term baby. However, keep in mind that these babies are more likely to get cold quickly, and are more susceptible to hypoglycaemia and respiratory illnesses. These babies therefore should be transferred to the special care nursery as soon as possible for warmth, early feeds and oxygen as necessary.

### **Sequelae of Birth Asphyxia**

Asphyxiated babies may show evidence of neurological damage within the first few minutes of birth, and may have convulsions requiring anticonvulsants. Other signs may include irritability, high pitched cry, stiffness, floppiness, irregular respirations, and apnoeic spells. Commonly a moderately asphyxiated baby progresses from a floppy stage to an irritable, stiff stage, before recovering normal tone.

Some of the more asphyxiated neonates may be unable to suck and may require nasogastric feeding, while some may need oxygen. Other organs which can be affected by asphyxia apart from the brain are:

**Heart** - transient myocardial ischaemia

**Kidneys** - acute tubular necrosis may lead to haematuria, oliguria, or even anuria.

**Gut** - necrotising enterocolitis presents as bloody stools and radiologically as gas shadows in the intestinal wall.

The prognosis for infants with birth asphyxia will depend on the extent of the hypoxic-ischaemic injury to the various organs as already mentioned. Residual brain damage is not always possible to assess in the immediate neonatal period. Some disabilities only become obvious months later, such as cerebral palsy [4.12.7], mental retardation, epilepsy [4.12.4] and more subtle findings like specific learning disabilities, which become apparent at school age.



### 2.3.2 Feeding

#### **Advantages of Breast Milk**

All milk is species specific, and cows' milk is not made for human babies. Even "modified" cows' milk does not confer all the advantages of human milk, because:

**NUTRIENTS** - The fat and protein of human breast milk are more easily digested, and there is a lower sodium content than that in cows' milk.

**INFECTION** - Human milk contains antibodies and iron binding protein (lactoferrin), which protect the baby against infection. Also breast milk encourages the growth of certain bacteria (*Lactobacillus bifidus*) in the baby's colon, which then protects the baby from colonisation with enteropathogenic bacteria, e.g. *E. coli*. There is also much less chance of contamination of breast milk by pathogenic bacteria.

**ALLERGY** - Human milk protects against allergy as shown by the fact that infants from atopic families seem to suffer fewer, less severe and

#### **Disadvantage of Beast Feeding**

If the mother is HIV infected, there is a risk of transmitting the infection to her baby through breast milk, though this is less than the risks of transmission during pregnancy and delivery. This risk has to be balanced against the risks of inadequate food intake and exposure to infection from formula feeds for mothers with very limited financial resources. There is evidence that the best strategy for HIV infected mothers, unless well off financially, is to give exclusive breast feeding till 6 months old and then wean quickly.

later atopic symptoms (e.g. infantile eczema and asthma) if wholly breast fed in the first 4-6 months of life.

**BONDING** - Breast feeding encourages mother and infant to bond.

**ECONOMIC** - Breast milk is cheap and readily available.

For all these reasons it is very important to establish not only breast feeding, but exclusive breast feeding for the first 6 months of life. The belief that babies who are breast fed need extra water because they get thirsty should be dispelled. It is this supplemental water and other fluids that can lead to diarrhoea. If the baby is unable to suck well (because he is ill or preterm), the mother must be taught and encouraged to express her breasts to maximise breast milk production. Infected breasts require urgent and vigorous treatment. As the infecting

organisms are almost always staphylococci, flucloxacillin, erythromycin or chloramphenicol are likely to be effective in the mother, whereas penicillin is usually ineffective.

### **Sucking and breast engorgement**

Babies with good Apgar score and neurologically intact will suck from birth, and therefore can be put to the breast as soon as possible. Most mothers are able to breast feed with no problems, as nature prepares the breast during pregnancy for this purpose. To avoid problems with breast engorgement, ensure that the baby feeds from both breasts. The frequency of breast feeding will vary, but most babies will feed up to 8 times a day (this is mainly because the emptying time of human milk from the stomach is less than for cows' milk).

### **Regurgitation**

Regurgitation of some feed is common due to immaturity of the gastro-oesophageal sphincter. This need not cause alarm so long as the baby looks well and the weight gain is satisfactory.

### **Expected weight gain**

Most term babies regain their birth weight by the 10th day of life. Most babies gain about 20-30 gm/day after the first week. Most infants will double their birth weight during the first 4 months and triple it during the first year.

### **Fluid and Energy Requirements**

The average energy requirements for infants up to the age of 6 months is about 120 kcal/kg/day, with a fluid requirement of 150-200 ml/kg/day. Since human milk and most cows' milk formulae contain 66-70 kcal/100 ml, to meet its energy requirement the infant needs to take about 180 ml/kg/day of milk.

For the premature baby the total volume of feed should be gradually increased during the first week of life. This is even more important where the baby is being fed by nasogastric tube. Volumes should not be increased further if the baby has not tolerated the last increase. Hopefully by the end of the first week the baby should be tolerating about 150 ml/kg/day.

**Do not exceed feed volumes of 250 ml/kg/day.**

## 2 The Neonate - Low Birth Weight Babies, Temperature Control

<u>Day</u>	<u>ml/kg in 24 hours</u>
1	60
2	90
3	120
4	150 (Maximum in full term infants)
5	180 (In LBW infants)

### **Low Birth Weight (LBW) Babies**

LBW babies should ideally be fed 3 hourly, and the very small (under 1200 gm) may need 2 hourly feeds to stop them getting hypoglycaemia, and also, because their smaller stomach capacity may not allow larger less frequent volumes of feed. Overfeeding may be followed by abdominal distension and vomiting, leading in turn to aspiration of milk into the lungs. The answer to this problem is usually simply to reduce the volume of feed given, or to increase the interval between feeds and allow the stomach to empty more completely. However, be aware that feed intolerance (vomiting, regurgitation, abdominal distension) may be an early sign of sepsis.

If a baby is not gaining weight after the first week (full term) or 2 weeks (preterm), consider:

- inadequate feeding from poor lactation, cleft palate preventing sucking, inadequate volumes of expressed breast milk (EBM) and careless feeding by mother.
- abnormal fluid losses (diarrhoea or polyuria).
- inadequate calories (wrong preparation of full strength milk).
- too low environmental temperature which diverts energy from growth to heat production.

### 2.3.3 Temperature Control

#### **Mechanisms**

For good temperature control heat loss must balance production.

- Heat is produced by movement, shivering (not in neonates) and brown fat. The neonate can vary this little.

- Heat may be lost by convection, evaporation and radiation. The neonate is vulnerable because of a relatively large surface area and no ability to control these.

Regulation is by nervous system control of circulation and sweating. In the neonate, especially the preterm neonate, this control is very limited.

### Problems

- Babies can be overheated, especially if sophisticated incubators are used and not maintained well, and are operated by staff who are not knowledgeable about them.

- A more common problem is cold. Those especially at risk are low birth weight babies, babies needing resuscitation (therefore exposed to heat loss) and babies being transported (in a cool environment). The risk is greater at night and in the cold season.

### Keeping Babies Warm

Babies temperature should be 36.5°C (axillary) or 37°C (rectal). Warmth can be ensured by different methods including incubators, hot rooms (28°C), hot water bottles, direct contact with the mother skin to skin), and insulation if the baby is warm before being wrapped. Direct skin to skin contact with the mother is the best method to use in transferring newborn babies between hospitals, and is very useful in keeping healthy babies warm safely at no cost. Hot water bottles are a low cost technology, but they require regular refilling with hot water. Careful checks that they are not leaking or in direct contact with the skin of the baby are necessary to avoid scalds and burns. Incubators vary in sophistication, but all require maintenance, understanding of how they operate, and regular checks of their operating temperatures. Temperature checks of the baby are important, whatever system is used.

For a baby is nursed naked in an incubator in a warm room, guideline temperatures for incubator setting are as follows:

**Covering the head with a hat reduces heat loss considerably in the newborn.**

### Incubator Temperature

Birth Wt	Week 1	Week 2	Week 3
< 1.0 kg	35°C	34°C	33°C
1.0-1.5 kg	33°C	32°C	31°C
1.5-2.0 kg	32°C	31°C	30°C
2.0-3.0 kg	31°C	30°C	29°C
> 3.0 kg	30°C	29°C	28°C

Lower temperatures than these are effective if the baby is wrapped for insulation.

### 2.3.4 Kangaroo Care

Babies who are premature but otherwise well can best be kept warm, encouraged to breast feed often and therefore grow well, and bond with their mothers by "kangaroo care." The baby is kept in skin to skin contact with the mother on the front of her chest, between her breasts. This is suitable for babies under 2.5 kg who are otherwise well. They feed from the breasts as required, but, if sucking is not strong, the mother must also express both breasts and feed by small cup. For kangaroo care to succeed the mother must want to use it, after a full explanation of its advantages and precautions. The mother will leave the top button of her dress undone to make room for the baby and wrap an extra layer of cloth round herself and the baby for extra support, and she needs to sleep mainly on her back. When she has to put the baby down to wash, the baby must be carefully wrapped to conserve heat. During the hospital stay check the baby's temperature and weight, and ask the mother about any problems she notices daily. The staff should make efforts to keep up mother's morale by entertainment (e.g. radio, television, hand crafts). Discharge can be allowed when the baby has regained birth weight or reached 1.8 kg – whichever is greater. Ask the mother to return for weighing weekly till the baby is 2 kg, then fortnightly till 2.5 kg.

## 2.4 Neonatal Diseases

### 2.4.1 Cold Injury

#### **Definition**

Cold injury can occur if a baby is cold (35.5°C or below) for some hours. Short term cold is less serious, but babies when cold are often grey in colour and grunting until rewarmed. A day or two later those that have cold injury may have a red face and hands and firm cheeks. Babies with cold injury are liable to further complications of sepsis and hypoglycaemia.

#### **Management**

- \* rewarm (fast if the cold is of short duration, slowly with checks of blood glucose if the baby has been cold for some hours).
- \* give antibiotics for very probable sepsis, gentamicin and benzyl penicillin being the usual first choice.
- \* do dextrostix (or glucostix, BM stix as available) test to exclude hypoglycaemia and treat if necessary (see next page).

## 2.4.2 Hypoglycaemia

### Definitions

Newborn babies tend to have lower blood sugars than the older child. In addition, premature babies tend to have lower normal blood sugars than do term babies, so that the exact definition of hypoglycaemia remains controversial. Evidence suggests that brain dysfunction starts when the neonate's blood sugar falls below 45 mg/dl (2.5 mmol/l). But most experts are satisfied with maintaining blood sugar above 40 mg/dl (2.2 mmol/l).

### Causes

The following babies are at risk of hypoglycaemia.

- SGA babies - due to low glycogen stores.
- Premature babies - especially when feeding is delayed.
- Babies of diabetic mothers - hyperinsulinaemia is present.
- Babies with cold injury - secondary to disordered carbohydrate metabolism.
- Various uncommon congenital metabolic abnormalities, e.g. the Beckwith-Wiedemann syndrome [2.5.4].
- Babies with brain damage.

### Diagnosis

Dextrostix tests should be done on all babies listed above. In the absence of dextrostix, blood should be taken for blood glucose measurement, ideally within the first 2 hours after birth. Presenting features may include: jitteriness, irritability, convulsions, apnoeic spells or floppiness. Many babies have asymptomatic hypoglycaemia.

### Treatment

- \* Dextrose should be given to all hypoglycaemic babies.
- \* If the baby is asymptomatic and if the dextrostix can easily be checked again about 1 hour after the feed, then oral 5% dextrose may be sufficient.
- \* The symptomatic baby with very low blood glucose (25 mg/dl or 1.3 mmol/l) should be treated with intravenous dextrose.
- \* Initially give 5% dextrose at the standard maintenance rate for the number of days old [2.3.2]. Recheck dextrostix 1 hour later.
- \* If dextrostix is still low, make up a 10% dextrose solution, by adding 25 ml of 50% dextrose to a quarter bag (250 ml) of 5% dextrose. This approximate 10% dextrose solution should be infused at maintenance rate as discussed in section 2.3.2.

- \* If the baby is convulsing and has hypoglycaemia, add a bolus of 5 ml/kg 14% dextrose (1.0 ml 50% with 4 ml 5% dextrose) into the drip.
- \* If venous access defeats you, give the 10% dextrose by continuous naso-gastric drip.

Remember (while treating the hypoglycaemic neonate) that you are performing a difficult balancing act between giving the baby enough of the right concentration of dextrose whilst avoiding fluid overload.

### 2.4.3 Respiratory Distress

#### **Clinical Features**

In neonates this is manifested as tachypnoea (> 60/minute), rib or sternal recession, grunting and cyanosis in room air.

#### **Management**

First exclude causes that require urgent and specific treatment:

- \* Cardiac failure
- \* Pneumothorax
- \* Diaphragmatic hernia

The specific treatment for the above three diagnoses is outlined below. However beyond that our options are limited. All babies with respiratory distress should be on antibiotics (gentamicin and benzyl penicillin) to cover for pneumonia or sepsis. Oxygen should be given at a minimum flow rate that keeps the child pink. When dyspnoea persists, attention to nutrition is essential and a nasogastric tube should be used if the child is very tachypnoeic.

#### **Differential Diagnosis**

Many conditions in the neonate can give respiratory distress including:

#### **CARDIAC FAILURE**

There is usually a murmur (but in the neonate some large VSDs, PDAs and coarctations [4.9.5] have none), and hepatomegaly and oedema may be present. Treatment consists of frusemide, digoxin, oxygen, fluid restriction and propping the baby up.

#### **PNEUMOTHORAX**

There is often sudden onset of respiratory distress, usually as a result of too vigorous resuscitation. There is reduced air entry on the affected side, and a displaced apex beat away from the affected side. If life-threatening, aspirate the pneumothorax with a 10 ml syringe and 21 gauge scalp vein (butterfly) needle. If less urgent, insert a scalp vein needle between the upper anterior ribs on the affected side, with the hub

of the tube dipped in a small bowl of sterile water, held below the level of the baby, till bubbling stops. This arrangement should then be replaced with a plastic IV cannula attached to an under-water-seal drain.

### **DIAPHRAGMATIC HERNIA**

The problem is of compromised lung volume as from abdominal contents in the chest cavity. There is reduced air entry (usually on the left) and bowel sounds can be heard in the chest. If the diagnosis is suspected, refer urgently for surgery, but anaesthesia and surgery are difficult.

### **RESPIRATORY DISTRESS SYNDROME**

Respiratory distress syndrome (RDS) is one of the most common causes of respiratory distress in newborn babies. A disease of premature babies, it is due to immaturity of the lungs from inadequate production of surfactant. This surfactant normally increases the compliance of the lungs. In the absence of surfactant the lungs are stiff, do not inflate easily and are poorly aerated. Where the disease is moderate to severe, these babies die because in Malawi they cannot be given assisted ventilation. Fortunately RDS is uncommon among African babies.

#### **Presentation**

These babies may be well in the first few minutes after birth but become progressively more dyspnoeic with time, being always dyspnoeic by 4 hours after birth and worst between 24-48 hours. After this they start to improve if they survive. The main features, which may vary in severity, are increasing dyspnoea, nasal flaring, grunting respiration, intercostal retractions and cyanosis. A chest Xray will show the so-called "ground-glass appearance", or may show a "white out" or solid lung in severe disease. If available give nasal oxygen 2 l/min.

### **PNEUMONIA**

Often there may be a preceding history of maternal fever, prolonged rupture of the membranes or smelly, offensive liquor. Tachypnoea, dyspnoea, subcostal and intercostal retraction and cyanosis are the usual presenting features.

### **TRANSIENT TACHYPNOEA OF THE NEWBORN**

This condition is self limiting as the name suggests and is caused by temporarily excessive fluid in the lungs. Usually the baby is term, born by caesarean section and is tachypnoeic right from birth and progressively improves.

### **MECONIUM ASPIRATION**

Occurs usually in post-mature and SGA babies who showed fetal distress



before birth. Meconium staining of the skin is often obvious. There is usually hyperinflation of the chest and these children are at increased risk of pneumothorax.

### **AIRWAY OBSTRUCTION**

There are various causes e.g. choanal atresia, tracheal obstruction by goitre.

### **PERSISTENT DUCTUS ARTERIOSUS**

Persistent ductus arteriosus (PDA) may occur in association with RDS, and treatment of the PDA may ameliorate the baby's symptoms. It is the preterm baby that has problems with a ductus that does not close quickly and may lead to cardiac failure and respiratory distress.

Clinical diagnosis is not always easy and the continuous (or machinery) murmur is not always present. More commonly there is an ejection systolic murmur, and the presence of bounding pulses will support the diagnosis.

Closure of the ductus can be induced by indomethacin, a drug that inhibits the production of prostaglandins, which are chemicals that keep the ductus open. The dose to be used is 0.2 mg/kg/dose orally 8 hrly for 3 doses. This can be repeated. Check for side effects which include oliguria or anuria, and gastrointestinal or other bleeding due to interference with platelet function.

#### **2.4.4 Apnoea**

Apnoea is generally defined as the cessation of respiration for 15-20 seconds, and is often accompanied by a bradycardia (heart rate less than 100/min). Apnoeic spells are common in premature babies. The cause of primary neonatal apnoea is not known, but may be related to immaturity of the respiratory centre in the brain. Apnoea of prematurity is uncommon on the first day of life, but commonly occurs after the first 48 hours of life.

#### **Diagnosis**

The diagnosis of apnoea of prematurity is one of exclusion, therefore it is necessary to rule out other causes which include:

- Intrinsic lung diseases e.g. pneumonia, RDS, pneumothorax.
- Sepsis and/or meningitis.
- Metabolic causes e.g. hypoglycaemia, hypocalcaemia
- CNS problems e.g. convulsions and intracranial bleeding.
- Cardiac diseases e.g. PDA.

- Obstructed airway e.g. congenital malformations and aspiration of feeds.

### **Treatment**

Most apnoeic spells can be terminated with just tactile stimulation. Occasionally babies may require ventilation with an Ambu bag and mask. Cardiac compression may be necessary when there is severe bradycardia. Oxygen should also be given either with face mask or with a nasal catheter. Exclude the causes listed above and treat specifically where indicated, otherwise start the baby on aminophylline treatment (give a loading dose of 6 mg/kg stat, followed by 1.3 mg/kg/dose 6 hrly). The drug can be given orally, but if the child is vomiting, should be given IM or slowly IV, well diluted.

Where the cause of apnoea is unclear, it is wise to give antibiotics for presumed sepsis. Remember to treat for hypoglycaemia if this cannot be excluded [2.4.2].

It is rare to see apnoea of prematurity after 34 weeks of gestational age, so do not give aminophylline in more mature babies.

## **2.4.5 Convulsions**

### **Classification**

The neonatal period is when the baby is most liable to have convulsions. Their diagnosis in neonates is not always easy. Many, especially preterm neonates, have benign jittery movements. The most common feature of convulsions are jerky repetitive movements which cannot be overcome by a firm grip on the involved limb as opposed to jittery movements which stop when the limb is held.

Neonatal convulsions are usually divided into the following groups: tonic, clonic (usually hemi-clonic), myoclonic and subtle. Generalised convulsions are unusual in neonates. Subtle convulsions may present as repetitive sucking, swallowing, blinking of the eyes, conjugate deviation of the eyes, etc.

### **Causes**

Convulsions during this period are rarely idiopathic. There are many causes, but the main ones include:

- asphyxia
- metabolic abnormalities especially hypoglycaemia, hypocalcaemia and hyperbilirubinaemia
- meningitis

- birth trauma
- congenital cerebral abnormalities

The time of onset of convulsions may give a clue to its cause. Hypoglycaemia, asphyxia and trauma account for most convulsions in the first 48 hours of life. After the first week of life, meningitis and congenital brain abnormalities are more common.

### **Diagnosis**

The history and examination may provide the initial clues to the aetiology e.g. traumatic delivery, low Apgar score, and the presence of early jaundice. Some laboratory results may confirm the aetiology e.g. blood glucose, calcium and bilirubin, as well as CSF results.

### **Prognosis**

The prognosis depends on the aetiology. Generally, neonates with convulsions secondary to perinatal asphyxia have the worst prognosis in terms of mortality and long term neurological sequelae. Term babies generally do better than preterm babies, especially if the physical examination is normal. Close to 50% of babies with convulsions secondary to neonatal hypoglycaemia will be normal assuming the hypoglycaemia is properly treated.

### **Management of neonatal convulsions**

The aim of treatment is to stop the convulsion, and to prevent further convulsions from occurring. Phenobarbitone in adequate doses (see below) is good for both aspects. Paraldehyde and diazepam are only good for temporary convulsion control.

The approach to managing neonatal convulsions is essentially the same as that in older children with a few minor differences [3.4]

1 Control the convulsion with:

- PARALDEHYDE (0.2 ml/kg) IM (paraldehyde dissolves plastic so use a glass syringe if possible or use a plastic syringe quickly).
- PHENOBARBITONE (10-20 mg/kg) IM stat, then 5 mg/kg daily.

2 Do a dextrostix and treat hypoglycaemia if necessary or if it cannot be excluded [2.4.2].

3 If after 5-10 minutes the convulsions continue, then do the following (sequentially):

- Repeat PARALDEHYDE and PHENOBARBITONE injections.
- Give DIAZEPAM (0.25 mg/kg) either slowly IV or rectally (which is easier and safer).

4 Do a lumbar puncture.

5 Give antibiotics.

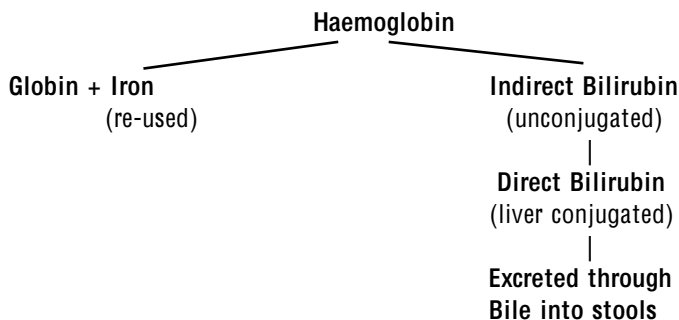
**Summary of drugs used**

Step	Drug	Loading dose	Maintenance dose
1	Paraldehyde	0.2 ml/kg IM PRN	
2	Phenobarbitone	10-20 mg/kg IM	5 mg/kg daily
3	Diazepam	0.25 mg/kg IV/rectal PRN	
4	Phenytoin <sup>1</sup>	20mg/kg IM or IV	5-8 mg/kg daily (or 12 hrly)

2.4.6 Jaundice

**Physiology**

Jaundice is the yellow discoloration of skin and sclerae that results from elevated levels of bilirubin, which comes from the break down products of proteins that contain haem. The most important source of bilirubin is the breakdown of haemoglobin from old red blood cells. The bilirubin in its initial (unconjugated) form is insoluble in water and is transported in the blood attached to albumin. It has to be conjugated (joined) to glucuronic acid in the liver to make it water soluble and therefore excretable in the bile via the bile duct into the intestine. (See diagram of bilirubin metabolism below).



**Physiological Jaundice**

In the fetus the bilirubin is transported to the placenta in unconjugated form and excreted by the mother. After birth the neonate has to develop the necessary enzymes in the liver for conjugation. This process takes a variable period of time and consequently the neonate has a limited ability

<sup>1</sup> Phenytoin is rarely available in injectable form. Oral formulations are of doubtful use in neonates. Phenytoin can come as step 3 in uncontrolled convulsions.

to conjugate bilirubin initially. As a result, jaundice is quite common during the immediate neonatal period and this is commonly referred to as "physiological." It usually starts between the 3rd and 5th day of life and not in the first 24 hours of life. The bilirubin is unconjugated (water insoluble or indirect) but rarely rises to dangerous levels.

### **Patterns of Neonatal Bilirubin Blood Levels**

#### **Jaundice of Prematurity**

This is an exaggerated form of physiological jaundice, resulting from immaturity of the hepatic mechanism for conjugating as well as excreting bilirubin. The bilirubin is indirect and can rise high enough to cause kernicterus (brain damage). Remember that a lower level of bilirubin can cause kernicterus in a premature baby than in a term baby. The level for phototherapy and exchange transfusions are therefore lower (see below).

#### **Haemolytic Jaundice**

Increased red blood cell breakdown will lead to a more rapid rise in bilirubin and earlier onset of jaundice - even within a few hours of birth.

The bilirubin is water insoluble but lipid soluble, therefore can cross the blood-brain barrier, to give kernicterus. ABO blood group incompatibility is probably the most common cause of this jaundice in Malawi.

Haemolysis can arise from Rhesus blood group incompatibility, glucose-6-phosphate dehydrogenase (G6PD) deficiency, congenital syphilis, and neonatal sepsis.

Some clues for haemolysis

- positive Coombs test.
- high reticulocyte count.
- low haemoglobin.

### **Obstructive Jaundice**

In this type of jaundice the bilirubin is conjugated (i.e. there is a high direct, or water soluble hyperbilirubinaemia), and can occur either from liver damage resulting from neonatal hepatitis and other congenital infections or from physical obstruction of the bile duct e.g. biliary atresia. Obstructive jaundice tends to present after the first week of life, and there is no danger of kernicterus, though the underlying cause for the jaundice may be dangerous. Biliary atresia is usually inoperable in our circumstances, and these babies die of cirrhosis during the first 2 years of life.

### **Kernicterus**

The main reason for emphasising early recognition and treatment of neonatal jaundice, is to prevent the bilirubin rising high enough to cause kernicterus (bilirubin encephalopathy).

#### **PATHOPHYSIOLOGY**

- Initial, unconjugated bilirubin may become "free" when all the binding sites on albumen on which it is carried in the blood are filled. This free fraction is lipid soluble and can cross the blood-brain barrier, and be deposited in the brain cells. This leads to toxic encephalopathy, brain damage and possibly death.

#### **CLINICAL SIGNS**

- These are varied. Early signs may be increasing lethargy, irritability or poor feeding. This may be followed by hyperextension of the head leading to opisthotonus position. Vomiting, apnoeic spells and convulsions may all occur.

#### **LONG TERM SEQUELAE**

- Include mental retardation, deafness, and choreoathetotic cerebral palsy.

### **Management of Neonatal Jaundice**

Early diagnosis is essential in order to avoid kernicterus. Measuring the total bilirubin is adequate initially. If the total bilirubin is very high (20 mg/dl in full term and 15 mg/dl in preterm), the direct bilirubin should be checked. A high direct bilirubin as already mentioned has a different aetiology from a high indirect bilirubin, and the treatment may be with antibiotics rather than phototherapy or exchange transfusion.

#### **Feeding**

Ensure that the jaundiced baby is well hydrated. Oral feeds should be pushed to reduce the entero-hepatic circulation of bilirubin.

#### **Phototherapy**

Start phototherapy as soon as possible (see guidelines below). To prevent damage to the eye, use eye patches during phototherapy. Phototherapy works by blue light energy changing the bilirubin into an excretable water soluble form (a photoisomer). This is then excreted partly in bile and partly in urine. The process is slow, involving change of bilirubin in the skin and diffusion of more to the skin from the blood. Check the bilirubin again 6-8 hours after stopping phototherapy, because sometimes bilirubin rebounds.

#### **Exchange transfusion**

Where phototherapy has failed to keep the indirect bilirubin at safe levels (see guide), an exchange transfusion should be done [5.4.17]. This can also correct severe anaemia associated with haemolytic jaundice.

Jaundice on the first day is likely to need exchange transfusion, and this possibility should be discussed with a paediatrician. It can only be of value if transfer to a central hospital can be achieved within a very short space of time, so that beneficial effects of phototherapy are not interrupted for too long.

The aim is to exchange twice the baby's blood volume (180 ml/kg). Dangers of the procedure include under and over-transfusion, hypoglycaemia, hypocalcaemia, sepsis, blood-borne infection, cold injury and hyperkalaemia. The table on the next page is a guide to the management of neonatal jaundice.

<u>Age</u>	<u>Birth weight</u>	<u>Repeat bilirubin after 24 hours</u>	<u>Start Phototherapy</u>	<u>Exchange transfusion</u>
<i>(days)</i>	<i>(kg)</i>	<i>Total bilirubin in mg/dl</i>	<i>Total bilirubin in mg/dl</i>	<i>Indirect bilirubin in mg/dl</i>
1 - 1.5	1.5	6	7.5	14
	1.5 - 2.5	7.5	9	17
	2.5	9	10.5	20
1.5 - 3	1.5	9	10	14
	1.5 - 2.5	10.5	12	17
	2.5	12.5	14	20
>3	1.5	10.5	13	15
	1.5 - 2.5	12.5	15	18
	2.5	16	18	21

**NOTE:** Exchange transfusion is quite dangerous where ideal equipment and expertise are not available. Bilirubin levels are liable to laboratory and other error. The above levels are therefore only a suggested guideline for deciding when to perform phototherapy or an exchange transfusion. The decision is often difficult, and in general we recommend that phototherapy (not a dangerous procedure) be started earlier rather than later, and the decision to perform an exchange transfusion be weighed carefully in the light of the whole practical situation. Where bilirubin levels cannot be measured, jaundice occurring in the first 48 hours is an indication for urgent phototherapy.

#### 2.4.7 Infections

Neonatal infections can be congenital (crossing the placenta during pregnancy), acquired during labour or acquired after birth.

##### **Congenital Infections**

Few organisms can cross the placenta. Most important are HIV, and syphilis, but others include rubella, cytomegalovirus, toxoplasmosis, chickenpox (varicella) and *Listeria monocytogenes*. Suspect congenital infection in any ill neonate with a combination of some of the following: hepatosplenomegaly, jaundice, anaemia, purpura.

##### **CONGENITAL RUBELLA**

- may present with some of the following: microcephaly, microphthalmia, cataract, deafness, persistent ductus arteriosus.



### **Treatment**

Of these congenital infections the only easily treatable one is congenital syphilis, so give benzathine penicillin 50,000 units/kg stat to neonates with splenomegaly and repeat after 1 week, and/or benzyl penicillin 50,000 units/Kg/dose 6 hrly for 10 days (preferably after a VDRL test on the mother). Treat the parents. Management of the HIV exposed baby is covered in section 2.4.7.1.

### **Infections Acquired During Delivery**

#### **GONORRHOEA**

Gonococci can cause a severe conjunctivitis with thick yellow pus and eyelid oedema (ophthalmia neonatorum), and may lead to corneal perforation if not treated.

- \* Wash the face.
- \* After careful cleaning, tetracycline eye ointment 1% 6 hourly for 3 days to both eyes.
- \* Gentamicin 5mg/kg as a single dose IM.
- \* Remember to treat the parents for gonorrhoea.<sup>1</sup>

#### **HIV, CANDIDA (OR THRUSH), HEPATITIS B AND HERPES SIMPLEX**

These infections may also be acquired during birth.

#### **GROUP B STREPTOCOCCI**

Group B streptococci are frequently found in the vagina and faeces of mothers and they (and other organisms) can cause acute septicaemia, pneumonia and meningitis, if they infect the liquor amnii. This may result from prolonged rupture of the membranes or after many vaginal examinations.

### **Infections Acquired after Birth**

These usually come from the mother or attendants, or from other babies if in a nursery, especially if sharing a cot or incubator (a bad, but often unavoidable, practice). Preterm babies are much more susceptible to infections. The most common organisms are staphylococci, coliforms and group B streptococci, but in nurseries outbreaks of infections with salmonellae, klebsiellae, pseudomonas and other gram negative organisms can occur. Neonatal tetanus is an infection requiring special management (see two pages further on).

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<sup>1</sup> See Malawi Standard Treatment Guidelines.

### **Clinical Patterns**

Infections may be minor (mild conjunctivitis, septic spots, sticky umbilicus, paronychia, breast abscess, oral thrush) but, in preterm neonates especially, treat all seriously because of the ease with which minor infections can lead to major infections such as septicaemia, meningitis, pneumonia, osteomyelitis, peritonitis and pyelonephritis.

Apart from possible preceding minor infections, major infections tend to present with non-specific signs of illness - inactivity, poor colour, poor feeding, vomiting, diarrhoea, jaundice, low or high temperature, apnoeic attacks, convulsions, dyspnoea, sclerema.

A few signs are more specific, such as cough which indicates pneumonia, abdominal distension which suggests peritonitis and bulging fontanelle which suggests meningitis.

### **Management**

#### **INVESTIGATIONS**

- If possible specimens should be taken for culture, but treatment should never be delayed as infection may progress very rapidly to death in neonates.

#### **DRUGS**

- Give benzyl penicillin (or alternatively ampicillin) and gentamicin but, during outbreaks of infection, experience or laboratory studies may indicate better antibiotics. When there is a strong clue that the infection is staphylococcal (e.g. the presence of septic skin blisters - to be differentiated from milia/sudamina, or umbilical sepsis) the best choice is flucloxacillin.

Meningitis is particularly difficult to treat, as causative organisms may be resistant to common antibiotics that penetrate the blood-brain barrier, and chloramphenicol and cotrimoxazole are both dangerous in neonates. Gram positive neonatal meningitis should be treated with ampicillin and gentamicin for 14 days, while gram negative meningitis should be treated for 21 days. If the baby is still febrile after 72 hours, change to ceftriaxone. An alternative is chloramphenicol, but in neonates give a lower dose than for older children. Give 6 mg/kg/dose 6 hrly during the first two weeks of life and 12.5 mg/kg/dose 6 hrly during the third and fourth weeks. Thereafter give the normal dose of 25 mg/kg/dose 6 hrly. The low dose is to avoid "grey baby" syndrome [2.1].

### **Prevention**

Especially in nurseries preventive measures are important. Wash hands, destroy insects like cockroaches, ants and flies, avoid overcrowding by restricting unnecessary admissions, isolate infected babies and keep infected adults out of the nursery. Sterilise, or at least carefully clean, feeding utensils for artificial feeds. Clean equipment such as incubators, cots, sheets, weighing machines and thermometers, using antiseptics.

### **NEONATAL TETANUS**

This is usually caused by tetanus spores contaminating the umbilicus from the use of unsterile instruments, dressings or the application of traditional treatments. This infection is now rare thanks to antenatal tetanus toxoid immunization, but beware mothers who may miss the vaccine antenatally by attending a TBA.

#### **Clinical**

The most common initial feature is cessation of breast feeding, followed by constipation, stiffness and spasms. Diagnosis is usually easy, but sepsis especially meningitis, kernicterus and severe birth asphyxia can cause confusion. All can cause increased tone and sometimes convulsions (not spasms as in tetanus).

#### **Management**

- \* First sedate the baby, usually with paraldehyde (0.2 ml/kg IM), followed by phenobarbitone (10 mg/kg/day) and diazepam (0.5 mg/kg 3-6 hrly).
- \* Then give penicillin and ATS 10,000 units.
- \* Nurse the baby in an intensive care area where observation can be close, with attention to airway, temperature and spasms. Give nasogastric feeding with expressed breast milk (EBM). In neonates, unlike older children, light and noise do not provoke spasms, but handling does, so keep it to a minimum with a 3 hourly schedule of nursing procedures, tube feeds, temperature checks and drug dosing.

#### **Prognosis**

Prognosis is poor if the onset of symptoms is before 7 days, if spasms occur within 12 hours of stopping sucking, if spasms are frequent and prolonged, if treatment is begun over 24 hours after symptoms start or if there is evidence of chest infection. Skilled nursing and adequate sedation are the most important factors for survival.

### **Prevention**

This can be achieved by immunisation of mothers antenatally with tetanus toxoid, by encouraging hospital delivery with the use of autoclaved equipment, or by training traditional birth attendants (TBAs) to use sterile or at least very clean instruments and dressings for the cord. On admission give neonates born outside hospital whose mothers did not get adequate tetanus toxoid antenatally, 750 units ATS IM or alternatively penicillin to prevent tetanus. Tetanus does not confer immunity so give recovered babies pentavalent vaccine at the appropriate age for immunisation.

Educate the mother of baby admitted with tetanus about the cause and start her on tetanus toxoid.

#### **2.4.7.1 Management of the HIV exposed neonate**

Clean blood and other fluids from the baby's surface at birth. At birth it is not known whether a baby is infected by HIV. If the mother has signs of HIV infection or AIDS or a positive HIV test, you must try to prevent the baby acquiring infection from her after birth as part of Prevention of Mother To Child Transmission (PMTCT). The mother should have had a single oral dose of 200 mg nevirapine in labour (or perhaps be on ART for AIDS or a low CD4 count). Give the baby a single dose of nevirapine 4 mg/kg soon after birth and not later than 72 hours if possible.

Consider whether the mother can afford and manage formula milk feeding, to avoid the risk of transmitting HIV through breast milk. In the great majority of cases the mother has to breast feed, because this is much safer than poorly or unhygienically prepared substitutes. It is then most important to teach her to feed her baby exclusively on the breast for the first 6 months, then wean rapidly. Give all HIV exposed babies cotrimoxazole to prevent *Pneumocystis carinii* pneumonia (PCP) in a dose of 30 mg/kg/dose of combined drug (5 mg/kg/dose of trimethoprim) daily from 6 weeks old until the baby has tested HIV negative at 18 months and at least 6 weeks after stopping breast feeding. Increasing availability of polymerase chain reaction (PCR) tests on dried blood spots makes early infant diagnosis (EID)<sup>1</sup> possible. HIV test the HIV exposed infant at 6 weeks, and if negative check the PCR. If that is positive start ART. If PCR is negative continue PCP prophylaxis till 18 months old and 6 weeks or more after weaning. If then HIV negative, discharge.

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<sup>1</sup> See National HIV/AIDS Guidelines

### 2.4.8 Bleeding

Bleeding can occur from many sites in neonates, especially the umbilicus, the gastrointestinal tract and operation sites.

Local causes may be important e.g. poor cord ligation, later umbilical sepsis. Bleeding tendencies are also seen, most often haemorrhagic disease of the newborn, though haemophilia may present as bleeding from a circumcision. Severe congenital or acquired infection may lead to disseminated intravascular coagulopathy (DIC) with lack of platelets and clotting factors, so that bleeding occurs, often haemoptysis.

#### **HAEMORRHAGIC DISEASE OF THE NEWBORN**

This is due to deficiency of vitamin K (platelets are normal). This occurs usually about the 2nd-5th day of life, and is related to lack of intestinal bacteria which produce vitamin K.

#### **MANAGEMENT**

The treatment is with vitamin K either IM or orally, but both routes take time to act, as vitamin K has to be converted into prothrombin in the body. Occasionally a transfusion of fresh blood is needed.

## 2.5 Congenital Abnormalities

### 2.5.1 Causes

There are a very large number of congenital abnormalities, many of them rare, but overall about 4% of children born have some form of congenital abnormality. In many cases the cause is not known. Some causes are:

#### ***Inherited gene defects***

- Dominant e.g. achondroplasia
- Recessive e.g. albinism
- Variable Expression e.g. cleft lip/palate.

#### ***Chromosomal abnormalities***

- Down's syndrome (extra chromosome 21), related to maternal age
- Turner's syndrome with only one X chromosome – and no Y - (webbed neck, short stature and abnormal ovaries)

#### ***Congenital Infection***

- Congenital Rubella

#### ***Folic Acid Deficiency***

Folic acid deficiency early in pregnancy may give spina bifida. Any mother who delivers a spina bifida baby should go on folic acid supplements well before later pregnancies.

**Drugs including alcohol**

- Fetal alcohol syndrome (causes unusual facial features, mental retardation and growth retardation)
- Thalidomide (caused severe limb reduction). Thalidomide may be used in leprosy patients or patients with Kaposi's sarcoma.

**Never give thalidomide to women who may be pregnant, and warn patients on thalidomide about this.**

**Radiation**

- Microcephaly occurred after atomic bombs in Japan and the Chernobyl nuclear accident.

**Intrauterine**

- Positional (genu recurvatum – leg bent forwards at the knee)
- Amputations from amniotic bands.

**2.5.2 Management**

Detailed knowledge of the abnormality is needed. Some parents want to know the cause, and in some cases it is possible to give advice on this, especially in hereditary conditions. When counselling, take care if at all possible to tell both parents together, and not to break up marriages by blaming either parent.

Parents may want to know of the prognosis both for their abnormal child (especially in relation to possible mental deficiency), as well as for further children (in hereditary conditions). In an autosomal recessive condition like albinism [4.15.3] or sickle cell disease [4.16.2], the risk of recurrence is 1 in 4.

Teach parents if simple management will make a difference, e.g. spoon feeding of EBM in cleft palate, protection of albinos from sunlight.

Follow up in sickle cell disease or in biochemical abnormalities such as congenital hypothyroidism.

There may be need of surgery - urgently, soon after birth or delayed.

**2.5.3 Practical Approach**

***Some need urgent ambulance transfer to a unit able to treat***

- imperforate anus and other intestinal obstruction
- exomphalos/gastroschisis
- micrognathia (small lower jaw causing obstructed breathing)

## 2 The Neonate - Congenital Abnormalities

- a few spina bifida babies with exposed meninges but no other defects
- a few patients with congenital cardiac defects and failure (tachycardia, hepatomegaly).

### ***Some need referral soon (by bus) to unit able to treat***

- severe club foot
- congenital dislocation of the hip
- inguinal hernia
- goitre
- cystic hygroma
- cataract
- cleft lip with or without palate (after EBM feeding is established and baby is gaining weight well)
- encephalocele
- hydrocephalus

### ***Some need referral after immunisation completed, because of risks of cross infection in hospital***

- undescended testis
- hypospadias
- fused fingers
- congenital heart disease without failure.

### ***Some need treatment at local health unit***

- genu recurvatum, i.e. a leg bent forwards at the knee (figure of eight bandage wound round the front of upper and lower leg alternately, passing behind the knee to keep the knee flexed for 1 to 2 weeks)
- mild to moderate club foot (serial POPs)
- sternomastoid tumour (repeated supervised manipulation by mother)
- albino (education on protection from sunlight) [4.15.3]

### ***Some are untreatable, so referral is useless***

- Down's syndrome (mongol) - though some have other treatable abnormalities [2.5.4]
- achondroplasia
- spina bifida, where the anal sphincter does not work, the legs are weak or the head is enlarged.

***In some treatment varies***

- umbilical hernia seldom needs surgery, but very rarely leads to intestinal obstruction, when operation becomes urgent.

- extra digits are commonly attached by a narrow base, and can be treated locally by tight ligation, but occasionally they contain cartilage. Then refer for surgery after immunisation is completed.

- preauricular sinus only needs treatment if it become infected and leads to abscess formation, when careful surgical excision after control of infection is required to prevent recurrence.

**2.5.4 Some abnormalities in more detail**

**EXOMPHALOS** (omphalocele, US omphalocele)

This is congenital protrusion of intestine or other abdominal contents, covered only by peritoneum, through the base of the umbilical cord. (A protrusion through the abdominal wall at a different site, e.g. through a defect in the abdominal muscles, is called gastroschisis). Immediate management is to cover the sac with sterile saline-soaked gauze. Urgent surgical repair is preferred treatment, but if access to surgery is delayed or the sac is too large for repair, even with lateral releasing incisions, an alternative is to apply an antiseptic like mercurochrome daily until the skin at the edge can grow to cover the sac. The constant danger is peritonitis and sepsis. Watch for this and treat with antibiotics if you suspect it.

Exomphalos may be associated with other abnormalities such as malrotation of the gut and intestinal obstruction.

**BECKWITH-WIEDEMANN SYNDROME**

Occasionally a baby with exomphalos may have other abnormalities such as large tongue, large size or hemihypertrophy, large liver and kidneys and severe hypoglycaemia. The risk of severe and prolonged hypoglycaemia makes this syndrome important to recognise. If the baby survives there is a greater risk of tumours like Wilm's tumour later.

**IMPERFORATE ANUS**

If a baby fails to pass meconium within 24-48 hours, check for congenital imperforate anus. This may be of the low or high type. In the low type meconium in the rectum may be seen bulging under skin cover at the anus, and surgery on the perineum can relieve the obstruction easily,



though follow up of anal function is needed. If meconium is not bulging at the anus, take a lateral Xray of the baby's abdomen and pelvis with the baby held upside down and an opaque marker at the anal site. A gap of more than 1 cm between gas in the rectum and the anus indicates a high obstruction and an urgent colostomy is needed, with later repair of the anus. Often by the time of diagnosis, the baby is dehydrated from vomiting, and preoperatively gastric emptying by nasogastric tube and IV electrolytes and glucose are needed to make the baby fit for surgery.

### **DOWN'S SYNDROME** (extra chromosome 21)

There may be some of characteristic features often found in this syndrome: eyes that slant upwards laterally, small nose, protruding tongue because the mouth is small, often small head flattened posteriorly, epicanthic folds at the inner angles of the eyes, little fingers short and incurved, a single transverse palmar crease and general hypotonia. There is a variable degree of mental deficiency, often severe, which leads to delayed motor and speech development. There may be other abnormalities like congenital heart disease, imperforate anus and duodenal atresia. These may be treatable, but the mental retardation cannot be cured, and it is important to explain to parents that it is a waste of time taking the child round different hospitals looking for a cure. The child will usually be pleasant and happy but slow to do things, and unable to learn at school. Down's syndrome is more often seen in babies of older mothers, but if a young mother has a baby with Down's she has a higher risk of having more.

### **CLEFT PALATE**

Cleft lip and palate often but not always go together and quite often occur with other abnormalities. Sometimes the abnormality runs in families. The first difficulty the baby faces with cleft palate is inability

**Always check for cleft palate in a baby not regaining birth weight by 2 weeks old, or failing to thrive in the first 2 months.**

to suck at the breast, requiring feeding by cup with expressed breast milk. If there is a cleft lip this will be noticed and the mother helped right away, but if there is no cleft lip, the cleft palate may be missed and the baby present as failure to thrive after a few weeks. Once the baby is

gaining weight well, refer for repair of the lip. This not only improves appearance, but helps to bring the cleft gums and palate together. Repairing the cleft palate is difficult and requires specialist surgery. It should be done by about a year, after full immunisation, in order to help speech develop. Even after repair of the palate there is a greater incidence of otitis media in cleft palate babies. If a baby with cleft palate has a fever, always check for otitis media.

**SPINA BIFIDA** (occulta, meningoceol, meningomyeloceol)

There are different degrees of spina bifida. Mild defects are hidden (occulta) the spinal defect covered but with an overlying dimple, hairy patch or lipoma. A meningoceol has a defect in the skin with meninges on the surface. A myelomeningocele has both meninges and spinal cord nervous tissue on the surface. Usually the defect is lumbar, but occasionally it is in the neck or skull (encephalocele). There may be associated hydrocephalus.

There may be different degrees of nerve damage below the spina bifida, often with leg deformities, leg muscle weakness and loss of sensation over the perineum and legs. These can lead to difficulties in walking later, but more immediate problems are the risk of meningitis if meninges are on the surface, the problem of increasing hydrocephalus and loss of control over passing urine. That in turn may give urinary retention and back pressure or reflux of urine up the ureters with frequent secondary urinary infections and kidney damage. There is urgent need to get skin closure over exposed meninges on the back to prevent meningitis but this has to be balanced against the long term problems of hydrocephalus, bladder dysfunction and kidney damage and problems over walking, and many surgeons will not want to operate on babies who have evidence of hydrocephalus, possible bladder dysfunction (shown by dribbling urine and loss of reaction to pin pricks on the perineum and flaccid anal sphincter). So in the baby with spina bifida, measure head circumference, look at the anus, watch for reaction to pricking the perineum, see if urine is constantly dribbling from the urethra, and check for leg deformities. If none of these problems is found, surgery on a meningocele is urgent. (Myelomeningoceols are always associated with damage). These considerations are especially important when deciding about referring patients some distance to a central hospital. If transferring, cover the meningocele with a sterile dressing and keep the

baby warm by skin to skin contact with mother, or, if she is ill, a supporting relative.

Spina bifida may be partly genetic, but there is a proven connection with folic acid deficiency in early pregnancy. So always advise a mother who has one spina bifida baby to start taking folic acid regularly before she has intercourse with her husband again.

### **HYDROCEPHALUS**

This may be associated with spina bifida, but can also occur alone before birth (when it often causes delayed or obstructed labour) or afterwards, and can occur as a complication of neonatal meningitis. Diagnosis may be obvious with huge head and "sunset sign" of eyes (white sclera seen above the iris), but in earlier cases depends on careful measurement of occipito-frontal head circumference, and demonstration that it is increasing faster than normal [1.3]. Be careful not to diagnose hydrocephalus in other conditions with large heads, especially achondroplasia, where there are abnormally short limbs and poor development of the base of the skull leading to expansion of the vault (upper part of the skull). Ultrasound examination through the usually wide anterior fontanelle can confirm enlarged lateral ventricles in cases of doubt. Insertion of a shunt with valve to take CSF from a lateral ventricle to the peritoneum is needed. This requires early (but not emergency) referral to a central hospital.

The main problems with shunts are blockage from clotting in the shunt

**If a child with a shunt for hydrocephalus is vomiting, do not perform a lumbar puncture, before you are sure the shunt is working well. Otherwise there is a risk of the child dying from coning of the brain from the raised intracranial pressure.**

(much more likely if CSF protein is high from congenital infection or previous meningitis) and infection. So check CSF for cells and protein before shunting, and be careful no infection occurs in the operation wound.

## **2.6 Birth Injuries**

### **2.6.1 Fractures**

Fractures heal very well in neonates, and need little or no treatment.

### **No treatment**

- Crack fractures of the skull.
- Fractures of the clavicle: reassure the mother that the lump that appears after some days will disappear fully after a few months.

### **Immobilisation**

- Fracture of the humerus is easily managed by bandaging the arm, with the elbow bent to 90° to the chest wall.

### **Traction**

- A fractured femoral shaft is treated by gallows traction i.e. skin traction applied to both legs, attached vertically above to a fixed support so that the baby's buttocks are just off the bed. Pad the ankles and dorsum of the foot carefully to protect against pressure sores. Continue traction for about 3 weeks, looking regularly for pressure sores.

### **Other treatment**

- Depressed fracture of the frontal bone is best treated within 24 hours by application of a suitably sized (obstetric) vacuum extractor cup and rapid generation of a strong vacuum by vigorous pumping. The skull will be restored to normal shape, but the area of the fracture will be obscured for some hours by a "chignon" caused by the vacuum extractor.

## 2.6.2 Nerve Injuries

### **ERB'S PALSY**

This is the most common type of injury and is due to damage to the upper brachial plexus, commonly occurring after shoulder dystocia or breech extraction. It presents as an arm that does not flex at the elbow or abduct at the shoulder (the so-called waiter's tip posture), so that the moro reflex on the affected side is abnormal. Apart from regular mild passive movement of the arm through a full range daily, no treatment is indicated, and recovery usually occurs spontaneously, though it may take months in severe cases.

### **KLUMPKE'S PARALYSIS**

This is less common and is due to damage to the lower brachial plexus. It presents as a wrist drop, and again no active treatment is indicated.

### **SPINAL CORD INJURY**

This is rare and no treatment is possible.

### 2.6.3 Soft Tissue Injuries

#### **FACE/VERTEX PRESENTATION**

Bruised and abraded areas should be kept clean (shaving of the scalp and application of spirit twice daily to the abraded scalp is helpful). Eye ointment should be used when there are abrasions around the very swollen eyelids in a face presentation.

#### **CEPHALHAEMATOMA**

This is common and is due to a blood clot that forms a few days after birth beneath the periosteum of one or more of the occipital or parietal bones. It usually resolves spontaneously over a few months. While healing, the swelling changes from a fluctuant mass clearly limited by the skull suture lines, to an area of apparently soft central depression surrounded by a circular edge of new bone formation (which can suggest a false diagnosis of depressed fracture). There is no indication to aspirate the blood, as this may introduce infection. With large cephalhaematomas there may be increased neonatal jaundice needing phototherapy.

#### **STERNOMASTOID TUMOUR**

Another occasional haematoma may occur in one of the sternomastoid muscles, a swelling appearing a few days after birth. To prevent this haematoma progressing to scarred tissue with contraction of the sternomastoid muscle and torticollis, make sure that the baby's head is regularly completely turned to the side affected several times a day. The mother should be taught to do this, but as the procedure will cause the baby to cry, it is important to arrange regular follow up to ensure stretching is being carried out well.

# 3 CLINICAL PRESENTATIONS

## 3.1 Introduction - IMCI

The Integrated Management of Childhood Illness approach (IMCI) sets out advice on measures to be taken by primary care workers with limited resources. All health care workers need to know what to do in such conditions, and those in health centres and hospitals should know what to expect frontline workers to have done already for patients referred to them. We have therefore summarised appropriate first line actions in an initial box like this for each clinical presentation. Remember even if you do not have resources for treatment, you are a trained observer, and record your observations such as weight, temperature, pulse and breathing rates, and capillary refill time as a valuable baseline for future treatment. Also remember to pass on your knowledge to guardians.

Part of the IMCI idea is that efficient triage of ill children will identify and rapidly manage emergency and priority diseases. Treat emergency conditions in the "ABCD" order, where A is for Airway, B Breathing, C Circulation and D Depressed consciousness. We have changed the sequence of this clinical presentation section to fit with this order. Sections 3.1 - 3.4 cover signs suggesting the need for emergency treatment. Other priority conditions include oedema [3.7], high fever [3.5], pallor [3.8] burns [4.20.1] surgical problems [4.20.2&3], poisoning [4.18] and sick babies under 2 months [covered mainly in section 2].

**In this sort of box we put important information and warnings.**

## 3.2 Breathlessness, Cyanosis & Cough

### 3.2.1 Breathlessness

Refer breathless children. Give first aid for any inhaled foreign body [4.8.6]. If there is pallor send a blood donor with the child. If there is dehydration supply ORS to be given on the way. If you suspect cholera start erythromycin. If you suspect pneumonia start antibiotic. If there has been recent measles and the child can swallow, give vitamin A.

### 3 Clinical Presentations - Breathlessness

First exclude or treat causes of airway obstruction.

#### History

Onset - an abrupt onset suggests inhaled foreign body. [4.8.6].

- a more gradual onset, or long history may also occur with obstruction, but the causes and treatment are different.

Obstructive Breathlessness (stridor)

- Rapid, often deep, breathing
- Barking cough or inspiratory stridor
- Difficulty breathing IN

Causes include inhaled foreign body [4.8.6], croup or laryngo-tracheo-bronchitis [4.8.2], retropharyngeal abscess [4.8.9], acute epiglottitis [4.8.3], measles laryngitis [4.6] and diphtheria [4.7.5]. Chronic stridor from soon after birth may be due to soft larynx or trachea (laryngomalacia or tracheomalacia), or (later in onset) to perhaps oral haemangioma [4.15.2] or laryngeal papilloma (warts).

If there is no respiratory obstruction from an inhaled foreign body, think of the other possible causes of dyspnoea, and look for

Associated features - fever, inability to drink and feed, drooling, sputum, or signs suggesting AIDS.

#### Examine

- \* Look for signs of dyspnoea (grunting, nasal flaring or subcostal recession).
- \* Listen for stridor (inspiratory noise), associated with upper airway obstruction, or wheeze (expiratory noise), associated with bronchial or bronchiolar obstruction.
- \* Look for severe pallor of palms, nail beds, tongue, conjunctivae (admit child with blood donor if possible).
- \* Count respiratory rate. Normal respiratory rates are < 60/min under 2 months of age, < 50/min from 2-11 months, and < 40/min from 1-4 years. Do this when the child is undisturbed (sleeping or breast feeding).
- \* Look at breathing pattern (deep, shallow, or prolonged expiration).
- \* Examine for tachycardia, hepatomegaly > 3 cm, gallop rhythm.

Breathlessness may be the result of rapid breathing (tachypnoea) which may occasionally be the result of fever or anxiety. Many children with tachypnoea also have dyspnoea (grunting, nasal flaring and subcostal recession). This may be the result of respiratory disease [see under cough

in section 3.2.3], cardiac or metabolic problems. Severe malaria can also be the cause.

#### **Cardiac Breathlessness (Cardiac Failure)**

- Rapid and shallow breathing
- Possible intercostal or subcostal recession
- Tachycardia – always present
- Hepatomegaly
- Gallop heart rhythm (most specific sign)
- Displaced apex, suggesting heart enlarged, and murmurs may be found.

#### **ANAEMIA<sup>1</sup>**

Examine the child for signs of pallor. If very pale, confirm by Hb check and cross-match blood urgently for transfusion [3.9.1]

\* Give frusemide 1 mg/kg.

Transfuse preferably 10 ml/kg packed cells or 15-20 ml/kg of blood.

**If you do not know the child's Hb, think carefully whether pallor could be due to poor heart output, when blood transfusion may overload the failing heart and cause pulmonary oedema.**

#### **OTHER CAUSES OF CARDIAC FAILURE [4.9.1]**

\* Look for evidence of fluid overload (IV drip or marked oedema).

**Fluid overload is especially likely to occur in malnourished children given IV fluids, partly because it is easy to over estimate the degree of dehydration in marasmus, and partly because the malnourished child has difficulty excreting sodium and water.**

\* Check BP to exclude hypertension, listen for murmurs [4.9.6]

\* Treat with frusemide and digoxin.

\* Chest Xray and perhaps ECG, cardiac ultrasound.

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<sup>1</sup> Sometimes very anaemic children may be breathless but not in cardiac failure - this is probably due to hypoxia although coexistent pneumonia must be considered. A transfusion is indicated in these circumstances. Cardiac failure from other causes (e.g. myocarditis) can cause pallor from vasoconstriction.



### 3 Clinical Presentations - Breathlessness, Cyanosis

#### Metabolic Breathlessness (usually without cough)

- Rapid and deep breathing
- There may be drowsiness
- There may be dehydration

#### DIABETES [4.14.1]

Ask about polyuria, polydipsia and weight loss. The breath may smell ketotic (sickly sweet)

- \* Check blood and urine sugar.
- \* Restore intravascular fluid with saline or Ringer's lactate urgently and control high blood glucose by insulin.

#### SEVERE DEHYDRATION [4.10.1]

History of severe diarrhoea and/or vomiting and clinical evidence of dehydration.

- \* Urgent fluid replacement [4.10.1]

#### ASPIRIN INGESTION [4.18.4]

History of ingesting excess aspirin.

- \* Induce vomiting or stomach wash-out [4.18.1]

#### RENAL FAILURE [4.11.5]

History of anuria, oliguria or previous renal disease.

- \* Check blood pressure.
- \* Urinalysis and U&Es, abdominal ultrasound.

### 3.2.2 Cyanosis

**If onset recent, refer urgently, but cyanosis for more than a week is often from congenital heart disease: refer to a specialist clinic.**

Cyanosis may be caused by airways obstruction, lung disease (including lung collapse and pneumothorax), or congenital heart disease [4.9.5] - most often Fallot's Tetralogy, (when cyanosis starts towards one year old), or transposition of the great vessels or various diseases with right to left shunting of blood when pulmonary artery pressure rises above systemic blood pressure. Cyanosis is not seen in anaemic patients, as cyanosis only occurs if there is > 5 gm/dl deoxygenated Hb circulating

So cyanosis rules out severe anaemia.

- \* Try giving oxygen for half an hour: in congenital heart disease there will be no improvement (best assessed by pulse oximeter).

### 3.2.3 Cough

Ask whether the child is feeding: inability to feed tells you the child is seriously ill and needs referral. Listen for stridor, look for indrawing, count breathing rate for one minute while the child is quiet. Stridor with suggestive history needs first aid to remove a foreign body, indrawing suggests severe or very severe pneumonia needing referral after first dose of benzyl penicillin IM, cough with rapid breathing but no dyspnoea calls for 5 days of cotrimoxazole with review after 2 days for mild pneumonia. Also educate guardian on what to notice that would need urgent review, and the need for review if the cough lasts for 3 weeks.

Check breathing rate, ears, throat, lymph nodes, chest.

#### Recent Onset Cough - Not Breathless

- \* If no abnormality, other than a runny nose (coryza), then a viral infection is likely and NO specific treatment is required.
- \* If pharyngitis or tonsillitis with lymphadenopathy [4.8.5] - give amoxicillin for 10 days (or benzathine penicillin IM stat).
- \* If fever present and in a child under 5 there is fast breathing but no dyspnoea - treat for mild pneumonia [4.18.4]

**Normal respiratory rates are < 60/minute under 2 months of age, < 50/minute from 2-11 months, and < 40/minute from 1-4 years.**

#### Pneumonia [4.8.14]

Count respiratory rate, look for dyspnoea and cyanosis and listen for localised fine crepitations and bronchial breath sounds

MILD PNEUMONIA - tachypnoea but no dyspnoea

- \* Give cotrimoxazole as an outpatient.

MODERATE PNEUMONIA - dyspnoea with or without tachypnoea

- \* Admit.

- \* Give benzyl penicillin IM (followed by oral amoxicillin) for 7 days.

### 3 Clinical Presentations - Cough - Pneumonia, Bronchiolitis, Asthma, HIV

**SEVERE PNEUMONIA** - severe dyspnoea, drowsiness or failure to feed, cyanosis and/or not responding to penicillin

- \* Admit.
- \* Give chloramphenicol with benzyl penicillin IV, followed as child improves by oral amoxicillin. Use gentamicin rather than chloramphenicol in children under 2 months.
- \* Give oxygen if cyanosed or low saturation on pulse oximeter.
- \* Do a chest Xray [5.3.1] if complications of pneumonia such as effusion or empyema are suspected (dyspnoea or cough for more than 1 week with asymmetrical chest expansion, dullness, poor air entry and reduced breath sounds).

#### **Bronchiolitis [4.8.11]**

Viral illness of infants, often in small outbreaks, with peak age of occurrence 3 months (uncommon after 9 months). Examination shows expiratory rhonchi and possible inspiratory crepitations. There is loss of cardiac dullness on percussion and hyperinflation of the chest.

- \* Give cotrimoxazole to cover possible infective pneumonia.
- \* Give oxygen as required.
- \* Ensure adequate fluid and energy intake, by IV fluids or NG tube feeding as necessary.

(The most important differential in this situation is PCP [4.8.15]).

#### **Chronic Cough - with or without breathlessness**

If cough has been present for more than four weeks investigate further to establish the diagnosis. Possible causes include:

##### **ASTHMA [4.8.10]**

Cough is often worse at night, perhaps with past severe episodes, and there may be family history of asthma or a past history of eczema. Examine for expiratory rhonchi (specially on forced expiration).

- \* If not breathless, trial of oral salbutamol or aminophylline for 2 weeks, or, if available, inhaled salbutamol by nebuliser or inhaler.
- \* If breathless, admission and more vigorous treatment is needed.

##### **HIV INFECTION [4.3]**

History of recurrent chest infections with partial improvement on antibiotic treatment and recurrent bouts of diarrhoea. Look for evidence

of failure to thrive (health passport), for oral thrush and for lymphadenopathy (cervical, axillary, inguinal) parotid enlargement, herpes zoster, Kaposi's sarcoma.

In infants consider possible *Pneumocystis carinii* pneumonia (PCP) and in older children Lymphoid Interstitial Pneumonitis (LIP). In all children over 3 months old keep the possibility of TB in mind.

#### TUBERCULOSIS [4.5]

Ask about TB contact history and look for evidence of weight loss or failure to thrive and cervical lymphadenopathy. Consider associated HIV infection.

In a child with chronic cough, always keep HIV/TB in mind.

- \* Chest Xray, though not diagnostic, may be helpful [5.3.1]

#### COLLAPSE OF LUNG

Ask for a history of recent respiratory tract infection with persistent symptoms or past inhaled foreign body.

Feel for displacement of trachea or apex beat towards the side of collapse, and listen for lung crepitations, bronchial breathing.

- \* Chest Xray [5.3.1]
- \* Consider bronchoscopy
- \* Start amoxicillin and postural drainage with percussion
- \* Confirm resolution with repeat chest Xray after 4 weeks.

#### WHOOPING COUGH (pertussis) (usually without dyspnoea) [4.7.9]

Cough for up to 3 months, worse at night, often in spasms that may be followed by vomiting (in children over 6 months) or apnoea and cyanosis (infants under 6 months). Ask if immunisation incomplete, and if other children have cough or there is a known outbreak of whooping cough.

Look for subconjunctival haemorrhage, rectal prolapse, torn tongue tie.

Listen for whoop at end of coughing bout (in older infants and children). Otoscope examination of an ear can set off coughing.

#### ' PARAFFIN ASPIRATION PNEUMONIA (4.18.2)

Ask for history of swallowing paraffin, often with choking or vomiting. Look for signs of pneumonia initially without fever.

- \* Treat with postural drainage and chest percussion, amoxicillin.

### 3.3 Shock

Shocked patients always need referral. If there has been external bleeding or possible major fracture, abdominal or chest injury, send blood donors with the patient. If there has been fluid loss from burns or diarrhoea and vomiting or severe polyuria, start an IV drip of Ringer's lactate or saline. Give a well nourished child shocked from blood or fluid loss 20 ml/kg rapidly, and repeat this dose once if necessary (see second box below for managing shock in malnutrition). If you suspect cholera, start on erythromycin. Record what you have done.

Shock may be defined as a state of vascular collapse, characterised by slow capillary refill, tachycardia and low blood pressure and often low

**Blanching caused by pressure for 3 seconds on a finger nail normally reverses within 3 seconds on release: longer is slow capillary refill.**

urine output, altered or depressed consciousness and acidosis. Note that circulatory collapse can lead to cool peripheries and peripheral cyanosis. In hospital this may give a low reading on a pulse oximeter.

Ask about external blood or fluid loss and history of trauma.

Shock can come from blood loss from trauma or medical causes and can be external or internal. (External examples are scalp wounds, severe epistaxis, bleeding from gastric erosions caused by aspirin or excessive iron tablets: internal bleeding may occur with fractures of femur or pelvis, rupture of spleen or liver, or bleeding into gut before vomiting).

Fluid loss can also cause shock, e.g. in burns [4.20.1], cholera

**Estimating severity of dehydration in the malnourished child is very hard, as marasmus gives loose skin and sunken eyes. Also malnourished children are easily overhydrated, so do not resuscitate malnourished children with IV fluids unless there is a clear history of severe fluid loss and the guardian spontaneously describes recent loss of skin turgor and sinking of the eyes. Pulmonary oedema easily occurs in malnourished children. If you are sure there is shock in a malnourished child give Ringer's lactate with 5% dextrose added 15 ml/kg over 4 hours. Carefully check heart and breathing rates hourly and if they increase, stop the IV infusion.**

[4.10.3], gastroenteritis [4.10.2], severe vomiting with intestinal obstruction [4.20.2] or in the urine in diabetes [4.14.1] or from excess diuretic. Note that fluid loss from the circulation can occur into the gut in ileus without diarrhoea, and into ascites in patients with low serum albumin from malnutrition [4.2], nephrotic syndrome [4.11.1] or cirrhosis [4.10.13], especially if ascites is tapped rapidly and excessively.

Nephrotics [4.11.1] can go into shock if given frusemide or if ascites is tapped too rapidly, and are liable to septic shock from pneumococcal infection.

Septic shock in severe infections is caused by an exaggerated inflammatory reaction leading to loss of vascular tone, pooling of blood in the veins and leaky capillaries. This tends to respond poorly to IV fluids, and may require an adrenaline drip as well as antibiotics.

Adrenal failure can also give shock, if adrenals are suppressed by prolonged high dose steroids that are then stopped abruptly. Very wasted children with TB may go into shock just after starting rifampicin which accelerates metabolism of their own reduced steroids. Treat this with physiological doses of steroids for a week or two.

Anaphylactic shock may occur due to sensitivity to drugs (e.g. penicillin), foodstuffs (e.g. nuts) or bee [4.19.3] or other stings. Histamine release is part of the cause. Give adrenaline urgently, then antihistamines and steroids.

Cardiogenic shock in children may occur with myocarditis (rheumatic [4.9.2] or viral) or cardiac tamponade from large pericardial effusion [5.3.1, 5.4.13] or in trauma from haemopericardium.

**Be careful not to think pallor due to poor peripheral perfusion in cardiogenic shock is anaemia, as blood transfusion can cause pulmonary oedema if the heart is not pumping well.**

Another possible cause of shock is giving an ACE inhibitor (e.g. captopril) for heart failure to a child on high dose diuretics. So only start ACE inhibitors under close hospital supervision in children on diuretics.

### **Giving Fluids for Shock**

Give IV or IO [5.4.3] fluids (Ringer's lactate or saline) 20 ml/kg in 10-20 minutes and then fluid with dextrose and potassium (e.g. half strength Darrow's dextrose) 100 ml/kg over 3 hours (6 hours under 1 year, but not in the malnourished - see previous page). Observe response carefully.

### 3.4 Coma

Coma always needs referral. First check the airway and keep child on the side during transfer. Control any convulsions. If there is fever, give IM quinine and benzyl penicillin. Check and record the coma score and when it was assessed in your referral note. If you have the resources, carry out full emergency measures below.

#### Emergency Measures

A child with coma requires urgent assessment and treatment.

- \* Check airway (place child on side and suction secretions).
- \* Check breathing (artificial respiration if absent, or if cyanosed).
- \* Check carotid pulse (cardiac compression if absent).
- \* Check dextrostix and if low give 1.0 ml/kg of 50% dextrose IV (preferably diluted with 4 ml/kg of 5% dextrose to give 14% dextrose<sup>1</sup>).
- \* Control convulsions if present [3.5]
- \* Commence an IV drip with 5% dextrose.
- \* Record a coma score<sup>2</sup> (sum 3 observations to total 0 to 5).

Blantyre Coma Score				
SCORE	MOTOR RESPONSE	VERBAL RESPONSE	EYE TO OBJECT	
2	Localises pain	Appropriate cry		
1	Withdraws limb	Inappropriate	Directed gaze	
0	None	None	does not follow	

#### Causes of Coma

Once the patient is stable and breathing well find the cause of the coma.

**History**                      Onset - rapid or slow.

Associated features - trauma, toxin ingestion, convulsions, preceding symptoms or illness, fever.

**Examine**                      Skull for trauma, breathing pattern for acidosis, hydration status, jaundice, purpura, pupil size and reactions, fundi for papilloedema, neck stiffness, blood pressure.

Intracerebral causes include tumour [4.12.6], abscess [4.12.5] (localising signs), epilepsy [4.12.4] as coma may follow a fit.

<sup>1</sup> 50% dextrose undiluted may cause vein thrombosis and blockage.

<sup>2</sup> Blantyre Coma Score is valid for children of 6 months or more.

### Extracerebral causes of coma

Suspect hypoglycaemia if neonate (especially preterm, with cold injury, diabetic mother or Beckwith syndrome) [2.4.2], or if patient is jaundiced or severely malnourished [4.2] or not feeding because of vomiting or on IV fluids not containing dextrose postoperatively. Toxins such as alcohol, herbal remedies, drugs including insulin and poisoning with aspirin and iron, or insecticides may also cause hypoglycaemia. Other causes of coma include diabetic coma [4.14.1] when hyperglycaemia gives polyuria, polydipsia and weight loss; hypernatraemia and hyponatraemia which are usually due to severe dehydration from gastroenteritis [4.10.2] or incorrect preparation of oral rehydration solution; hyperthermia if history of exposure to excessive heat (patient may or may not sweat); liver failure from fulminating hepatitis, which is often associated with severe jaundice; renal failure [4.11.5] with oliguria, anuria or haematuria and/or oedema; Reye syndrome [4.10.12] which presents with coma, vomiting and hypoglycaemia, especially after aspirin treatment of a child under 12 years old with chickenpox or influenza.

### Management of Coma

- \* Emergency measures as above.
- \* FBC and MPS.
- \* U&Es & blood glucose.
- \* Urinalysis (blood, protein, glucose, bilirubin).
- \* LP if coma score > 0, no cranial nerve lesion, no irregularity of breathing and no papilloedema.
- \* Perhaps skull and chest Xray.
- \* Start presumptive treatment for malaria and meningitis with IV quinine, penicillin and chloramphenicol while awaiting results.
- \* Consider referral to a higher level from peripheral unit, if the diagnosis remains unclear and the child fails to improve after 48 hours. Great care must be taken during any transfer to ensure that the child's airway is maintained.



### 3.5 Convulsions (or fits, or seizures)

**Ensure the child can breathe. Stop fits with paraldehyde 0.2 ml/kg deeply IM in the lateral thigh or laterally in the buttock (avoid the sciatic nerve) or diazepam 0.5 mg (*not ml*)/kg rectally (use a long narrow syringe without a needle). Then find and treat the cause, not forgetting hypoglycaemia.**

Priorities for the emergency management of convulsing patients are:

- 1 Ensure a clear airway
- 2 Stop the convulsion
- 3 Determine the cause of the convulsion

#### Control of convulsions

- \* Check dextrostix or give 1.0 ml/kg of 50% dextrose IV (if dextrostix not available) preferably diluted with 4 ml/kg of 5% dextrose
- \* Give paraldehyde 0.2 ml/kg by IM injection<sup>1</sup> or 0.4 ml/kg rectally.
- \* Give phenobarbitone 10mg/kg by IM injection as loading dose.
- \* If after 5 minutes the convulsion continues then:
  - (a) Repeat paraldehyde 0.2 ml/kg by IM injection
  - (b) Give diazepam 0.5 mg/kg rectally<sup>2</sup> or 0.2 - 0.3 mg/kg by slow IV injection.

OR in neonate repeat phenobarbitone 10 mg/kg IM

- \* If after a further 10-15 minutes the child is still convulsing give phenytoin 10 mg/kg (slow IV), if available, and if not, repeat phenobarbitone 10 mg/kg IM.
- \* Give oxygen if there is cyanosis.
- \* If child is pyrexial, lower the child's temperature with tepid sponging and give paracetamol.
- \* Give maintenance oral phenobarbitone 5 mg/kg per day.  
Prolonged convulsions (status epilepticus) may cause permanent brain and the prompt control of convulsions is imperative.

#### Causes of Convulsions

Once the convulsion has been controlled it is important to determine the

<sup>1</sup> Paraldehyde interacts with plastic so use a glass syringe, but if this is not available, you can use a plastic syringe as long as the drug is injected rapidly, and not left in the syringe for any length of time.

<sup>2</sup> Rectal diazepam is as reliable as IV and easier and safer to give.

cause. Abnormal electrical activity in the brain is the basis of convulsions, and this may be precipitated by various traumatic, metabolic or infective factors. A convulsion usually presents with tonic (stiffening) and clonic (regular muscle contraction and slower relaxation) movements. Such convulsions are termed grand mal convulsions but convulsions may be focal (one limb or muscle group), temporal lobe (unusual sensations and behaviours) or petit mal (short absences).

**History** Age of onset - different causes at different ages  
Type of convulsion - focal, generalised, or petit mal

**Examine** Fever, neck stiffness coma score, papilloedema, hypertension

### **Neonatal [2.4.5]**

In neonates convulsions may be grand mal, but are often subtle and present as twitching movements of the mouth or eyes. Jitteriness should not be confused with convulsions in neonates. Convulsions at this age have serious implications and causes include birth asphyxia [2.4.5] which is suggested from obstetric history, metabolic disorders such as hypoglycaemia [2.4.2] and hypocalcaemia, kernicterus [2.4.6] in severe jaundice, meningitis [2.4.7] and septicaemia. Tonic spasms suggest neonatal tetanus [2.4.7]

### **1-6 months**

Consider cerebral malaria [4.1], meningitis [4.12.1], hypoglycaemia and rare metabolic diseases may cause fits at this age. Febrile convulsions are rare under 6 months. If the cause of convulsions is unclear, and fits persist, refer to a paediatrician in case of rare treatable causes of the fits.

### **6 months - 6 years**

Febrile convulsions are the commonest cause at this age. Any cause of fever, including malaria, may precipitate a febrile convulsion in a vulnerable child. A history of similar fits associated with fever in the child, his siblings, parents or relatives supports the diagnosis. Febrile fits are usually single and not followed by long coma (< 20 minutes) or nerve deficit.

It is very important to differentiate meningitis and cerebral malaria from febrile convulsions. Consider a lumbar puncture [5.4.6] in ALL cases of convulsions to exclude meningitis. During LP avoid excessive neck flexion in small sick babies as this may cause deterioration in condition and death. If the child has a coma score of 0, irregular respirations or

### 3 Clinical Presentations - Convulsions

cranial nerve signs delay the LP, and immediately start treating for both cerebral malaria [4.1] and meningitis [4.12.1] with IV quinine, penicillin and chloramphenicol. The LP should then be done as soon as the coma score rises above 0.

Malaria with febrile convulsions and cerebral malaria may be difficult to tell apart. In febrile fits the child's general condition improves rapidly following control of convulsions and fever, whereas in cerebral malaria the level of consciousness remains depressed. In cerebral malaria there may be hypoglycaemia, which you must also treat.

Other important causes of convulsions to consider are epilepsy [4.12.4] and intracranial pathology such as head injury, brain abscess [4.12.5], and brain tumour [4.12.6]. The child will often be afebrile in these cases and intracranial lesions often produce focal convulsions. Hypoglycaemia may cause convulsions in malnourished children and severe dehydration may result in hypernatraemia and convulsions. Remember the possibility of toxic herbal remedies or drug overdose.

- \* Control convulsions as above.
- \* Do dextrostix (give 1.0 ml/kg of 50% dextrose, preferably diluted with 4 ml/kg of 5% dextrose, if dextrostix low or not available).
- \* Do an LP to exclude meningitis.
- \* make a blood film for malaria.
- \* Take family and past history of convulsions, history of contact with febrile illness and recent drug history.
- \* Depending on findings consider further tests such as:
  - Urinalysis
  - Skull Xray (if seizures persist or head injury suspected)
  - Chest Xray
  - U&Es
- \* If after 2-3 days convulsions persist, or if neurological deficits are noted and diagnosis is unclear, referral should be considered.
- \* Give malaria prophylaxis and possible maintenance phenobarbitone for children with recurrent (> 3) febrile convulsions.

**6 years and over** Febrile convulsions rarely occur for the first time after age six. Epilepsy [4.12.4] is the most likely cause of fits at this age, but consider other causes listed for the six month to six year age group. If fever is present, cerebral malaria and meningitis must be excluded.

### 3.6 Fever

Always ask how long fever has lasted, and check whether the child is seriously ill and for a rash.

Causes of fever for less than a week include malaria [4.1], virus infections including measles [4.6], bacterial and viral meningitis [4.12.1], pneumonia [4.8.14] and osteomyelitis [4.13.2]. Look if the child is seriously ill, e.g. has stopped feeding, has a reduced conscious level, difficulty breathing or convulsions, which suggest serious causes. If there are none of these and no rash, treat with paracetamol, and if no respiratory symptoms, for malaria, after asking whether the child has had antimalarials. Recent onset fever with rash may be measles, chickenpox [4.7.1], meningococcal meningitis or some viral infections. If the child is only mildly ill but has respiratory symptoms, check the ears for acute otitis media [4.8.7].

Causes of fever longer than a week include malaria, pneumonia complicated by pleural effusion or empyema [4.8.16], typhoid [4.7.8], TB [4.5], AIDS [4.3], osteomyelitis [4.13.2] and urinary infection [4.11.3]. Always ask carefully if any treatment, including antimalarials and antibiotics have been given and ask for fever in contacts and family features of AIDS. All children with fever over a week need hospital referral.

Ask yourself, "how ill is the child?" and "how long is the fever?" A mildly and briefly ill child may have a minor viral illness or malaria, so check for upper respiratory symptoms (give paracetamol) and if none, treat for simple malaria [4.1].

Check an ill child with a short illness for severe malaria [4.1], pneumonia [4.8.14], meningitis [4.12.1], measles [4.6], septicaemia, osteomyelitis [4.13.2], start appropriate treatment and refer.

With fever over a week consider HIV [4.3], TB [4.5], typhoid [4.7.8], hepatitis, osteomyelitis [4.13.2], urinary infection [4.11.3], complications of recent measles (chest infection, TB), trypanosomiasis [4.7.10]. Admit or refer, as appropriate, after malaria treatment.

**History**            Onset - acute (< 1 week)<sup>1</sup> or chronic (> 1 week),  
prior treatment

**Examine**            Coryza, cough, vomiting, loss of weight

<sup>1</sup> Bear in mind that the history may be very unreliable

### Acute Fever

#### **The "Well" Child**

If the child is not very sick, consider malaria [4.1] and viral infections [4.7.1]. A runny nose makes coryza likely. Check for otitis media.

- \* If nothing specific is found on examination, start presumptive malaria treatment, unless recently given [4.1].
- \* Prescribe paracetamol or, in child over 12, aspirin for 48 hours.
- \* Advise parents to return if fever persists, child becomes more unwell, lethargic or refuses feeds.

#### **The Sick Child**

If the child is sick, drowsy and not eating consider more severe malaria [4.1], pneumonia, [4.8.14] if there is dyspnoea, meningitis [4.12.1] if neck stiffness and irritability, measles [4.6] before the rash appears, TB [4.5] if lymphadenopathy and weight loss, hepatitis if there is vomiting and jaundice, osteomyelitis [4.13.2] and septic arthritis [4.13.1] if there are swollen tender bones or joints, or typhoid [4.7.8] if there is splenomegaly and toxicity (but this is now rare in children). If there are gastro-intestinal symptoms consider amoebiasis [4.10.4], shigella [4.10.5] or salmonella infections. Urinary tract infection [4.11.3] may be suggested by urinary symptoms but there may be no specific symptoms.

- \* Admit, and if no obvious diagnosis start malaria treatment.
- \* FBC and differential:
  - low to normal WBC is seen in malaria, viral infections and typhoid.
  - a high WBC suggests a pyogenic infection, but acute severe haemolytic anaemia in malaria may also give a high WBC.
- \* Urinalysis and culture - infants need bladder stab [5.4.9].
  - over 5 white blood cells / high power field, and a pure growth of bacteria suggest a urinary tract infection.
- \* Stool examination and culture.
- \* Chest Xray.
- \* Blood culture if available.

#### **The Critically Ill Child**

Assume septicaemia if the child is very ill.

- \* Do an LP, blood culture, and urine culture.
- \* FBC and chest Xray.
- \* Start penicillin and chloramphenicol IV (in the neonate penicillin and gentamicin). Alternatively give ampicillin IV instead of penicillin.
- \* Start Quinine IV.

### Chronic Fever

If prior malaria treatment has been given, the child may have resistant malaria. TB [4.5] is an important cause specially if the child is ill and malnourished. Consider HIV infection [4.3] if there is weight loss, recurrent chest infections, diarrhoea, bilateral parotid enlargement, or lymphadenopathy. Typhoid [4.7.8] often presents with prolonged fever, cough, abdominal pain, constipation or diarrhoea and headache. Abscesses (specially intra-abdominal) and chronic urinary infection cause persistent unexplained fever. Remember drugs may cause fever.

Rarer causes include infective endocarditis [4.9.3] - changing murmurs, splenomegaly, splinter haemorrhages and haematuria; trypanosomiasis [4.7.10] - check whether living in endemic area and for possible neurological features; borreliosis (relapsing fever) - headache, vomiting, myalgia and arthralgia; juvenile rheumatoid arthritis [4.13.4] prior to the development of joint problems, malignancies [4.17] especially lymphomas; and drugs, e.g. antibiotics.

#### Investigations<sup>1</sup>

- \* FBC and blood films for malaria, trypanosomes and borrelia.
- \* Stool and urine microscopy and culture.
- \* Blood cultures for typhoid, infective endocarditis.
- \* Chest Xray for TB.
- \* Serology for HIV.
- \* Lymph node and liver biopsies, bone marrow examination.

### 3.7 Failure to Thrive

Use evidence from health passport growth chart to diagnose this. Check the feeding, family and social and economic history to see if food shortage is likely or HIV infection or TB or other chronic underlying disease. Assess the severity [4.2] of the malnutrition to decide whether to send to an outpatient nutrition clinic or admit for investigation and feeding. Always arrange follow up.

A diagnosis of failure to thrive is readily made by examining a correctly completed health passport growth chart. A guardian may believe there is weight loss or failure to gain weight when the child is actually growing

<sup>1</sup> Consider a trial of TB treatment [4.5] if diagnosis remains uncertain despite these investigations and there is no response to antibiotics.

### 3 Clinical Presentations - Failure to Thrive

and changing proportions. Unless the child is underweight for age or height, obtain concrete evidence of weight loss or failure to gain adequate weight before diagnosing failure to thrive. See section 5.1 for growth charts.

#### Causes of Failure to Thrive

##### **Inadequate Intake**

This is the most common cause and in young infants may result from inadequate breast milk due to poor lactation. This in turn may be from maternal malnutrition or illness, such as TB or HIV. Milk supplements are usually necessary (although often unaffordable) while the mother tries to establish lactation. The infant must be fed from both breasts before getting any milk supplement. (If HIV is likely, stop breast feeding after 6 months).

Infants, not breast fed, who fail to thrive are usually receiving inadequate or inappropriate feeds. This may be because a working mother leaves someone else to care for the child, or following a maternal death when there is inability to provide sufficient milk for the infant's needs. The older child who fails to thrive for lack of adequate food intake usually presents with features of protein energy malnutrition [4.2].

##### **Inadequate Absorption**

An infant who appears to be receiving sufficient milk and other food but fails to thrive may not be absorbing the nutrients. This may be due to poor sucking with cleft palate or from persistent vomiting from pyloric stenosis [3.10], or from malabsorption.

**In breast feeding small infants of mothers with good lactation that are not thriving check carefully for cleft palate [2.5.4]**

##### **Underlying Infection**

Various chronic infections may present with failure to thrive. Consider TB [4.5] (check for TB contact, chest Xray), HIV infection [4.3] (age often under 3 years, chronic cough, fevers, diarrhoea and lymphadenopathy), UTI [4.11.3] (check urine microscopy and culture).

##### **Other Causes**

Infants with serious congenital heart disease [4.9.5], other congenital

abnormalities and congenital infections, [2.5, 2.4.7], brain damage, mental retardation, renal and liver disease. Hirschsprung's disease may prevent normal growth despite adequate intake of food. Psychological stress and emotional deprivation may result in failure to thrive. This may contribute to the failure to thrive seen in orphans who often also have inadequate intake.

#### **Management<sup>1</sup>**

- \* correct identification of the cause(s).
- \* treatment, where possible, of underlying infections and contributing factors.
- \* supply of adequate calories for growth - large volumes of feeds may be necessary to achieve growth in severely marasmic infants.

### 3.8 Oedema

Define whether oedema is localised or general. General oedema is often from kwashiorkor, but other causes of oedema need to be considered. All are serious conditions requiring hospital investigation and treatment. Kwashiorkor needs admission for treatment also. As part of management record the child's weight and height. Localised oedema may be inflammatory (e.g. cellulitis, treated with amoxicillin) or due to venous or lymphatic obstruction, which need paediatric outpatient assessment.

Oedema is an important presenting sign in paediatrics. Determine whether the oedema is generalised or localised.

#### **Nutritional Oedema**

The most common paediatric cause of oedema in Malawi is protein energy malnutrition [4.2]. The oedema usually involves the lower limbs, but may be more general when severe. Other signs of malnutrition will be found (poor hair texture, dermatitis and apathy). If these features are absent consider other causes of oedema.

Bilateral oedema of the legs in a malnourished child indicates severe malnutrition requiring admission for rehabilitation.

<sup>1</sup> See section 4.2 for details of protein energy malnutrition management.



### **Renal oedema**

This can be due to nephrotic syndrome, glomerulonephritis or renal failure. The oedema is characteristically facial but is often generalised. The oedema of nephrotic syndrome [4.11.1] like kwashiorkor and cirrhosis [4.10.13] is associated with hypoalbuminaemia. Diagnosis is based on demonstrating proteinuria without marked haematuria. Blood pressure is normal or low.

Glomerulonephritis [4.11.2] causes renal damage which impairs excretion of potassium, urea and water. Fluid overload may result and, with hypertension, may cause cardiac failure. Dark red urine may have been noticed and on testing there is haematuria (usually macroscopic). Reduced urine output (oliguria) may also occur.

The treatment of nephrotic syndrome (high protein diet and steroids), and glomerulonephritis (low protein, low potassium, high carbohydrate diet with fluid restriction) differs markedly. Thus the correct diagnosis, based on urine examination, is vital.

### **Cardiac oedema**

Oedema as the presenting feature of cardiac failure [4.9.1] is uncommon in young children. More often cardiac failure at this age gives tachycardia, dyspnoea, hepatomegaly and sometimes gallop rhythm (with or without murmurs). However cardiac failure must be considered in young children and always excluded in older children presenting with leg oedema.

### **Allergic Oedema**

Localised oedema resulting from insect bites, snake bites and other allergic and toxic causes is common, but allergy may cause generalised oedema.

### **Obstructive Oedema**

Obstruction to lymphatic drainage may produce oedema. This may occur as the result of lymph node infection from TB [4.5] or tumour. An important clue to diagnosis is that the oedema is often asymmetrical.

### **Miscellaneous**

Liver disease may cause oedema. In this situation there is usually abdominal distension from ascites and there may be hepatomegaly, splenomegaly, jaundice and a bleeding tendency. Sickle cell disease [4.16.2] may cause swelling of the small bones of the hands and feet, which can be confused with oedema. Other inflammatory conditions that cause swelling of a limb may be confused with oedema e.g. pyomyositis.

### 3.9 Pallor and Anaemia

Pallor is commonly due to anaemia, but can be caused by vasoconstriction, as in shock. Check the conjunctiva, tongue, fingernails and palms. Remember that conjunctivitis can make the eyes and stomatitis the mouth red even in an anaemic child. Palmar pallor is more reliable: compare with a healthy palm. Check the fingernail for slow capillary refill to diagnose shock (treat as in section 3.3). If refill is normal but pallor is marked, check for rapid respirations, dyspnoea, tachycardia, hepatomegaly and gallop heart rhythm. Finding any of these indicates anaemia may be severe. Give a first dose of quinine IM and refer to hospital with a blood donor. If pallor is less severe, treat malaria [4.1], give iron, and in a child over 1 year, albendazole, if none has been given in the previous 6 months. Review in 2 weeks, or at once if rapid or difficult breathing develops or the child stops feeding or convulses.

Pallor can be caused by vasoconstriction or anaemia<sup>1</sup>. The table below shows the lower limit of normal haemoglobin (Hb), the normal ranges for mean corpuscular volume (MCV), and white blood count for age.

AGE	Hb (g/dl)	MCV	WBC(10 <sup>9</sup> /l)	(%Polys)
Birth	13.0	110 - 125	9000-30000	(60%)
< 6 months	9.5	75 - 95	6000-18000	(30%)
6 months-6 years	10.5	70 - 90	6000-15000	(45%)
7-12 years	11.5	78 - 95	4500-13500	(55%)

#### Determining the cause of anaemia

##### History

Enquire about prematurity, diet, blood loss, jaundice, fever

##### Examine

Look for purpura, bruises, cardiac failure, jaundice, splenomegaly, lymphadenopathy

In Malawian children malaria [4.1] and iron deficiency are the most important causes of anaemia. Sickle cell disease [4.16.] is also fairly common. There are four main groups of causes of anaemia.

<sup>1</sup> Anaemia is defined as a blood Hb below the normal for age.

### 3 Clinical Presentations - Anaemia

#### 1 Loss of blood

Blood loss is an uncommon cause of anaemia in children, but may occur with severe repeated epistaxes which will be obvious on history, melaena (typhoid [4.7.8] and Meckel's diverticulum) and haematemesis (oesophageal varices). Rarely haemophilia [4.16.3] may cause anaemia. More commonly a chronic low-grade blood loss from hookworm [4.10.9] or schistosomiasis [4.10.11], may cause anaemia if iron intake is low. Acute blood loss will cause normocytic anaemia whereas chronic blood loss will usually cause microcytic anaemia (iron deficiency).

#### 2 Increased destruction of haemoglobin (Hb)

Haemolysis is suggested by a normocytic anaemia with reticulocytes  $> 1\%$ . There may be jaundice, a palpable spleen and urobilinogen in urine. Malaria [4.1] is the usual cause of haemolytic anaemia (between 6 months and 6 years). Sickle cell disease [4.16.2] and sepsis are other causes. In neonates think of blood group incompatibility [2.4.6].

#### 3 Reduced Hb production due to nutrient deficiencies

##### MICROCYTIC ANAEMIA (low MCV)

\* A major cause of anaemia is iron deficiency. Iron is transferred across the placenta in the 3rd trimester of pregnancy and thus preterm babies have low iron stores. Breast milk iron is low but it is well absorbed and breast feeding will help prevent iron deficiency. However after 6 months mixed feeding (with green vegetables and meat) is needed. Fruits containing vitamin C (guavas, mangos) promote iron absorption.

##### MACROCYTIC ANAEMIA (high MCV)

\* is usually caused by folic acid deficiency which can occur in very preterm babies, in malnutrition and sickle cell disease (chronic haemolysis).

##### NORMOCYTIC ANAEMIA (normal MCV)

\* can be caused by malnutrition [4.2], chronic infection or hypersplenism.

#### 4 Reduced Hb production due to marrow depression

Bone marrow depression is uncommon. Chloramphenicol and some other drugs (methotrexate, cyclophosphamide, rarely cotrimoxazole) can cause aplastic anaemia. Chronic infections (TB, chronic infected burns, chronic empyema, chronic osteomyelitis) may depress marrow function and in

leukaemia [4.17.5] the normal marrow is replaced by malignant cells. If there is purpura or bruising or white cell or platelet abnormalities on the blood film, bone marrow examination is needed for diagnosis.

### Management of Anaemia

\* If anaemia is severe, with cardiac failure, admit and transfuse. If safe blood is not immediately available give frusemide and oxygen at 1-2 l/min. Sometimes there is enough improvement to avoid transfusion.

**In children in heart failure there is always tachycardia, often dyspnoea and hepatomegaly and sometimes gallop heart rhythm (most specific sign). In older children raised jugular venous pressure and oedema may be found.**

- \* If mild to moderate anaemia, treat for both malaria and iron deficiency but complete malaria treatment first before starting iron therapy.
- \* Follow-up after 2 weeks with Hb and MPS to exclude resistant malaria.
- \* If anaemia persists or recurs assess the red blood cell size (MCV)<sup>1</sup> to help diagnose the cause of anaemia.

#### 3.9.1 Blood Transfusion

Where possible avoid blood transfusions because of the risk of HIV and hepatitis transmission. Even screened blood can transmit HIV during the "window period." However in severe anaemia, when there are hypoxic symptoms or cardiac failure, transfusion is needed urgently.

Severity of anaemia depends both on Hb level and speed of onset, but symptoms are likely when the Hb is below 5 gm/dl in acute or 4 gm/dl in chronic anaemia or malnutrition. Transfusion may be indicated below 7 gm/dl if there is serious intercurrent disease (severe infections, cardiac or respiratory disease) or cytotoxics are being given or below 10 gm/dl if surgery is urgent. Neonates with Hb < 9 gm/dl may need transfusion if requiring oxygen or if in shock or heart failure.

Try to find the cause of the anaemia. Take blood for FBC, thick and thin films, reticulocyte count and sickling test before giving blood.

Give 10 ml/kg of packed cells or 15-20 ml/kg of whole blood. Because there is danger of over-transfusion, give frusemide 1 mg/kg with the transfusion. Malnourished children should get no more than 10ml/kg.

<sup>1</sup> See table on page 71 for normal values.

### 3.10 Jaundice

Mild jaundice, only visible in daylight, not under electric light, may be due to haemolysis or hepatitis. Ask about past blood transfusion, drugs, especially nevirapine, triomune and TB treatment, fever, vomiting, stool and urine colour. If possible inspect stool and urine for colour and record what you find in the health passport. Check the coma score and for pallor, dehydration, fever, oedema and abdominal distension. Palpate for hepatomegaly, splenomegaly, other masses and ascites.

If jaundice is mild, without drug intake, high fever ( $> 38.5^{\circ}\text{C}$ ), marked pallor, very pale stools or very dark urine and with only mild hepatomegaly ( $< 5$  cm below ribs in right mid clavicular line) and mild tenderness, treat malaria and give paracetamol (ibuprofen is safer) for 2 days (not longer because of liver damage) with review in 3 days.

Refer children with severe jaundice, jaundice lasting over 2 weeks, high fever, pallor, dehydration, reduced coma score, marked hepatomegaly, liver tenderness, very pale stools or very dark urine.

<b>History</b>	Colour of stool and urine, fever, previous episode, contacts with jaundiced patients, toxins or drug ingestion, prodromal symptoms, preceding blood transfusions.
<b>Examine</b>	Depth of jaundice, fever, ill or well, anaemic, hepatomegaly, splenomegaly, purpura or petechiae, and level of consciousness.

Three types of jaundice may occur haemolytic, hepatocellular and obstructive.<sup>1</sup>

#### HAEMOLYTIC JAUNDICE

During haemolysis there is excess destruction of red blood cells resulting in release of large amounts of indirect (unconjugated) bilirubin. This overloads the liver's conjugating ability and excess indirect bilirubin accumulates in the blood. As this bilirubin is not water soluble, it cannot be excreted by the kidneys and urine colour remains normal. Furthermore, as the liver continues to excrete normal amounts of conjugated bilirubin in the bile, the stools remain normal in colour. Malaria [4.1], sickle cell

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<sup>1</sup> Neonatal jaundice is discussed in section 2.4.6.

disease [4.16.2], haemolytic-uraemic syndrome [4.11.6] and septicaemia are the main causes of haemolytic jaundice. Malaria and sickle cell disease are diagnosed on the basis of clinical features, a blood slide and sickle cell test. Septicaemic children are extremely ill and may have evidence of shock or disseminated intravascular coagulopathy (DIC).

### HEPATOCELLULAR JAUNDICE

Damage to the liver cells causes loss of conjugating ability and blockage to outflow of already conjugated bilirubin (damaged cells swell and close small bile ducts). This partial blockage to conjugated bilirubin excretion in the bile causes the urine to become dark (the water-soluble conjugated bilirubin leaks back into the blood and is excreted by the urine instead). The stools may be pale. Hepatitis A (infective hepatitis), the main cause of hepatocellular jaundice, spread by the faeco-oral route, has an incubation period of 2 weeks to 2 months. Another cause is hepatitis B (serum hepatitis), with an incubation of 6 weeks to 6 months, and mainly spread through the placenta or by transfusion, though now preventable by immunisation with pentavalent vaccine [5.2]. Treatment of infective hepatitis is conservative, and outlined as follows:

- \* Avoid drugs (e.g. paracetamol, stemetil) that may increase liver damage.
- \* Educate parents - the jaundice will take 10 days or more to resolve.
- \* Encourage high calorie fluids initially (e.g. sugar water).
- \* Refer if jaundice persists after 2 weeks or is associated with other abnormalities e.g. ascites, drowsiness.

Occasionally with infective hepatitis (and more commonly with serum hepatitis) liver failure develops. The jaundice deepens and the child becomes drowsy. Give IV dextrose 5%. The outlook is usually very poor.

Other causes of hepatocellular jaundice include infections such as malaria [4.1], typhoid [4.7.8], leptospirosis, drugs (notably nevirapine), toxins and metabolic defects like galactosaemia.

### OBSTRUCTIVE JAUNDICE

Obstruction to excretion of bilirubin through the biliary system results in pale stools and dark urine. Biliary atresia (congenital absence of bile ducts), gall stones (occasionally in children with sickle cell disease) and bile duct obstruction by ascaris, enlarged TB lymph nodes, or lymphoma in the porta hepatis are causes of obstructive jaundice in children.

### 3 Clinical Presentations - Jaundice, Vomiting

Biliary atresia usually presents some weeks after birth with deepening jaundice and a hard enlarged liver. The outcome is usually death in the second year.

Consider the following investigations in jaundiced children, if available:

- \* Hb or PCV
- \* MPs
- \* Sickling test
- \* Abdominal ultrasound
- \* Liver function tests
- \* Hepatitis B serology and VDRL

### 3.11 Vomiting

Ask and check for intake of drugs or chemicals, fever, diarrhoea or constipation, drowsiness, jaundice. Examine for dehydration, shock [3.3] and abdominal distension and tenderness. Weigh the child and record weight. If there is no shock, dehydration, drowsiness, or abdominal distension, give ORS, treat malaria and review in 3 days. Advise the mother to return early if vomiting becomes worse or child stops passing stools or urine, becomes lethargic, tachypnoeic or dyspnoeic. After giving appropriate initial care, refer children who are shocked, dehydrated, drowsy, or have abdominal distension urgently.

#### History

Onset and duration - sudden, progressive or persistent.

Type of vomit - projectile, small or large, mixed with blood or bile, or associated with cough.

Age and sex - pyloric stenosis usually presents in males between three and six weeks of age

Associated features - fever, abdominal pain, distension, constipation, or blood in stools

#### Examine

Pyrexial, drowsy, dehydrated, or with neck stiffness

#### NON-ORGANIC

Many normal infants will vomit small amounts after feeds. Provided the infant is gaining weight, passing stools, and otherwise well there is no need for concern.

### OBSTRUCTIVE

Significant vomiting occurring in the few days following birth may be caused by tracheo-oesophageal fistula (commonest variety has a blind oesophageal pouch) or a duodenal/intestinal stenosis or atresia. Hirschsprung's disease may present with bile stained vomiting and abdominal distension in the first week of life, although late presentation with massive abdominal distension can occur. From three to six weeks of age think of pyloric stenosis and always ask if the vomiting is projectile, look for peristaltic waves in the epigastrium (going from left to right) and try to feel the pyloric "tumour," a groundnut sized lump in the right epigastrium, while the baby is feeding. (Pyloric stenosis affects mostly boys, and often the first born in a family, unless there is a family history). Treatment is surgical splitting of the thickened pyloric sphincter muscle.

**Do not diagnose intestinal obstruction in a child whose abdominal distension was preceded by diarrhoea for a few days: ileus from potassium loss is more likely.**

Consider intussusception in the child aged 3 months to 3 years who has a sudden onset of acute distress with uncontrollable crying, followed by bile stained vomiting and blood stained stools. The spells of crying stop but recur at shorter and shorter intervals. Feel for a sausage-shaped abdominal mass when the child is quiet and abdomen soft. Do a rectal examination to detect a mass and blood. Early diagnosis and surgery has a low mortality, but late diagnosis has a high one. Erect (lateral decubitus if the child cannot stand) and supine Xrays of the abdomen can be helpful. Appendicitis is another important surgical cause of vomiting. If the cause of vomiting is thought to be obstructive do the following:

- \* Give the child nil by mouth.
- \* Insert a nasogastric tube to aspirate stomach contents.
- \* Commence glucose containing IV fluids to correct and maintain hydration. (preferably with half strength Darrow's dextrose).
- \* Refer for a surgical opinion.

### INFECTIVE

Many infections may be associated with vomiting. Vomiting may precede diarrhoea by several hours in children with gastroenteritis [4.10.2]. Urinary tract infections [4.11.3] commonly cause vomiting and need to



### 3 *Clinical Presentations - Vomiting*

be excluded when no other obvious cause is present. Vomiting is common with hepatitis [3.10]. Meningitis [4.12.1] and septicaemia must be considered in both the neonate and older infant and child. Malaria [4.1] is an important cause of vomiting to consider in Malawi. Whooping cough [4.7.9] may lead to vomiting. Vomiting is prominent in Reye syndrome [4.10.12]

#### OTHER

Birth asphyxia may cause vomiting in the neonate as may various rare metabolic disorders. The vomiting caused by a hiatus hernia is typically associated with flecks of blood and is worse when the baby lies flat. Diabetes [4.14.1] may present with vomiting and should be suspected if there is weight loss associated with polyuria and polydipsia. Progressively severe, effortless vomiting (specially occurring in the early morning) may be a sign of raised intracranial pressure [4.12.5 & 4.12.6]. Ask about associated headache, a change in personality and increasing drowsiness. Other causes to consider are renal failure [4.11.5], drugs like metronidazole, quinine, cyclophosphamide and poisons e.g. insecticides.

### 3.12 Diarrhoea

Ask how long? Is there blood in the stool, is there fever, vomiting? How many stools per day? Check for malnutrition (oedema, look at growth curve in health passport). Look for lethargy (coma score), eagerness or refusal to drink, sunken eyes, slow return of skin pinch, slow capillary refill. Weigh the child.

Blood in the stool suggests bacillary or amoebic dysentery [4.10.5 & 4.10.4] or perhaps schistosomiasis [4.10.11].

Treat any dehydration [4.10.1] and refer to hospital.

Severe malnutrition (marasmus or kwashiorkor) makes assessing and managing dehydration harder, so refer without giving ORS.

If stools have no blood, there is no or mild malnutrition and diarrhoea has lasted less than 2 weeks and is not recurrent, assess and manage this way.

<b>Signs</b>	<b>Severity of dehydration</b>	<b>Management</b>
2 or more of lethargy or coma sunken eyes unwilling/unable to drink skin pinch goes back v. slowly	severe	Child needs IV or NG tube fluids. Refer to hospital, giving ORS on way. If cholera around, give erythromycin.
2 or more of restless, irritable sunken eyes drinks eagerly, thirsty skin pinch goes back slowly	some	give 70 ml/kg ORS in 1st 4 hours, and reassess. If breast feeding, continue, if not, give 100-200ml water
insufficient of above signs to classify as dehydrated	none	teach mother to give extra fluids, to continue to feed (especially if breast fed), to look for dangers signs.

Refer babies under 2 months with any degree of dehydration to hospital. They are likely to need antibiotics for sepsis [2.4.7]

If diarrhoea has been for over 2 weeks with dehydration, correct dehydration and refer to hospital. If there is no dehydration, advise on feeding, give vitamin A, review and consider HIV infection.

### 3 Clinical Presentations - Diarrhoea

<b>History</b>	Onset - Recent or chronic Associated features - blood, mucus, fever, vomiting
<b>Examine</b>	Assess dehydration from features listed on previous page and depressed fontanelle, sunken eyes, loss of skin turgor, dry mouth, oliguria, tachycardia. Deep acidotic breathing is seen in severe dehydration.

#### Recent Onset Diarrhoea

<u>Cause</u>	<u>Fever</u>	<u>Blood</u>	<u>Mucus</u>	<u>Dehydration</u>
<b><i>Viral</i></b>				
Rotavirus	- to+	-	-	- to+++
<b><i>Bacterial</i></b>				
Cholera [4.10.3]	-	-	-	+to+++
E. Coli	- to+	- to+	- to+	- to+++
Shigella [4.10.5]	- to++	+	+	- to+
Campylobacter	- to+	- to+	- to+	- to++
Salmonella	- to++	- to+	- to+	- to++
<b><i>Parasitic</i></b>				
Giardia [4.10.6]	-	-	-	-
Amoebic Dysentery [4.10.4]	-to+	+	- to+	- to++
Strongyloides [4.10.10]	-	-	- to+	-
Schistosomiasis - [4.10.11]	-	+	-	-
<b><i>Dietary</i></b>				
Lactose intolerance	-	-	-	- to +
Malnutrition [4.2]	-	-	-	- to +
<b><i>Other Infections</i></b>				
Malaria [4.1]	- to++	-	-	- to +
Measles [4.6]	++	- to+	-	- to +
Otitis media [4.8.7]	+	-	-	- to +

### Treatment

- \* Treat shock if present [3.3].
- \* Replace fluid and electrolyte losses [4.10.1 & 5.5].
- \* Check for and treat hypoglycaemia if low coma score.
- \* Specific Treatment:

Fluid replacement is all that is required in viral diarrhoeas and mild bacterial diarrhoeas. Erythromycin 12.5 mg/kg/dose 6hrly for 3 days is given in cholera [4.10.3], antibacterials such as nalidixic acid or ciprofloxacin in severe bacillary dysentery, [4.10.5] and metronidazole for amoebic dysentery [4.10.4]. Lactose intolerance (rare) should be treated with lactose free feeds if possible [5.6]. (Lactose is the sugar in milk).

### Chronic Diarrhoea

If a child with chronic diarrhoea is not gaining weight adequately or is losing weight investigation is required. Important causes to consider are malnutrition [4.2], HIV infection [4.3], bacterial infections and parasitic infestations such as giardiasis [4.10.6] treated with metronidazole. TB and urinary infection can also cause chronic diarrhoea.

- \* Examine a stool specimen for parasites, urine for pus cells.
- \* Trial of treatment with metronidazole and/or cotrimoxazole.
- \* Look for evidence of HIV infection.

## 3.13 Abdominal Pain

Remember that when a baby has "abdominal pain" this is an interpretation by the guardian of observed symptoms. A small child under about 6 is not able to localise pain to any particular part of the abdomen so a history of pain is not as clear until later childhood. Your observations therefore become more important in reaching a diagnosis and deciding how to treat or whether to refer the child. The most important decision is whether the pain is from a condition needing surgery. Non-surgical illnesses to be considered include tonsillitis, sickle cell crisis, diabetic ketoacidosis and pneumonia.

- **History**                      Onset - sudden or gradual  
   Associated features - distension, vomiting, passage of stools, loss of weight, fever.

### 3 Clinical Presentations - Abdominal Pain

**Examine** Tenderness, guarding, rebound, hepatomegaly, splenomegaly, ascites or mass.

#### Acute Abdominal Pain with Distension

##### INTESTINAL OBSTRUCTION

Sudden onset of intermittent pain with abdominal distension and vomiting. The lower down the obstruction the greater the distension. Causes include strangulated hernia (check hernial sites), adhesions (scar of previous abdominal surgery), intussusception (severe distress in a young child, blood in the stools and a sausage shaped abdominal mass), and ascariasis.

\* Commence IV fluids and insert a nasogastric tube.

**Do not diagnose intestinal obstruction in a child whose abdominal distension was preceded by diarrhoea for a few days: ileus from potassium loss is more likely.**

\* Erect and supine abdominal Xray.

\* Refer for urgent surgical opinion.

##### PERITONITIS

Sudden continuous pain with variable distension, rebound and rigidity. Causes include perforated viscus (from trauma or appendicitis, typhoid [4.7.8] - toxic looking, splenomegaly and a preceding illness). There is an increased risk of pneumococcal peritonitis in patients with nephrotic syndrome [4.11.1] but in Malawi "primary" peritonitis (i.e. without an obvious cause like appendicitis) is not rare. At operation try to culture the peritoneal pus to find the causative organism.

\* Blood culture if possible.

\* IV penicillin, chloramphenicol and metronidazole.

\* Erect chest Xray to look for free air under diaphragm.

\* Refer for surgical opinion.

##### TRAUMA

Pain with variable abdominal distension following trauma and possible hypovolaemic shock, may be the result of damage to liver, spleen or other organs.

\* If shocked emergency resuscitation with IV fluids and blood.

\* Refer for urgent surgical opinion.

### **Acute Abdominal Pain Without Distension**

If pain is the main symptom with no evidence of distension consider uncomplicated appendicitis. There is typically a history of pain (colicky central abdominal becoming constant in the right iliac fossa), fever, anorexia and nausea. There is often vomiting and there may be diarrhoea and frequency of micturition. Look for tenderness, guarding and rebound in the right iliac fossa. Gentle rectal examination may show right lateral or anterior tenderness.

Other causes of abdominal pain include:

- Dysentery - diarrhoea with blood or pus, diffuse tenderness but no guarding.
- Pneumonia [4.8.14] - chest signs are usually obvious.
- Sickle cell crisis [4.16.2] - usually a known history of sickle cell disease, pallor, and splenomegaly (if under 6 years of age).
- Henoch-Schonlein purpura [4.16.5] - purpuric lesions seen over buttocks and legs, sometimes blood in the stool.
- Psychogenic pain - this typically presents with vague central abdominal pain, which is often recurrent. Unilateral pain usually has an organic basis.

### **3.14 Abdominal Distension (with or without pain)**

Where abdominal distension is the main complaint, the front line health worker needs to determine what is causing the distension, so as to refer the patient to the appropriate hospital department. Read the section below.

Establish whether abdominal distension is caused by bowel distension, hepatomegaly, splenomegaly, ascites or abdominal mass.

#### **3.14.1 Bowel distension**

In toddlers the abdominal wall muscles are lax and so protrusion may occur in health. In kwashiorkor [4.2] the abdomen is often distended as a result of weak abdominal muscles and hepatomegaly. Heavy worm infestation [4.10.8] may cause abdominal distension. In Hirschsprung's disease there is usually massive distension starting within the first few months of life. Typically the rectum is empty in Hirschsprung's disease, but is always full in chronic constipation, which is due to poor bowel

### 3 Clinical Presentations - Abdominal Distension - Intestinal, Hepatomegaly

habit and a low fibre diet – rare in Malawi - and presents with faecal masses and overflow incontinence.

- \* Do abdominal Xrays if obstruction suspected.
- \* Send stool for microscopy and treat with albendazole for suspected worms.
- \* If Hirschsprung's disease is suspected refer for surgical opinion and barium enema.
- \* Treat faecal loading with enemas and regular toilet training.

#### PARALYTIC ILEUS

Abdominal distension occurs with absent bowel sounds. Causes include septicaemia (specially in the neonate), hypokalaemia (common in kwashiorkor and after diarrhoea and vomiting) and herbal remedies.

- \* Treat suspected neonatal septicaemia with penicillin and gentamicin.
- \* Do U&Es, give IV potassium supplements if hypokalaemia suspected. If potassium chloride injection is unavailable, half strength Darrow's dextrose has a high potassium content [5.5].
- \* Abdominal Xray.

#### 3.14.2 Hepatomegaly

This may be caused by cardiac failure [4.9.1] so look for dyspnoea, tachycardia and gallop rhythm, hepatitis (acute illness with jaundice), schistosomiasis [4.10.11] with splenomegaly and possible ascites, malaria [4.1], amoebic abscess [4.10.4] with local tenderness and fever, kwashiorkor (fatty liver) and rare metabolic abnormalities which may also cause abnormal growth and development. Hepatomegaly may be due to tumour, most often Burkitt's lymphoma. [4.17.1]

- \* Treat cardiac failure [4.9.1], hepatitis or kwashiorkor [4.2].
- \* Do rectal snip [5.4.8], stool and urine microscopy.
- \* If acutely ill with tenderness and fever give metronidazole.
- \* If available check abdominal ultrasound.
- \* Give praziquantel if schistosomiasis strongly suspected.
- \* Fine needle aspiration (in central hospital), if tumour suspected.

#### 3.14.3 Splenomegaly

Malaria [4.1] is the most common cause of splenomegaly in children in Malawi. In younger children splenomegaly may be due to sickle cell disease [4.16.2]. Consider portal hypertension, schistosomiasis [4.10.11], malignancy [4.17.1], and TB [4.5].

### 3 Clinical Presentations - Abdominal Distension - Splenomegaly, Ascites

Massive splenomegaly may be caused by tropical splenomegaly syndrome (in children over 10), schistosomiasis with liver damage and portal hypertension, malignancy (only lymphomas, including Burkitt's and Hodgkin's involve the spleen) and leishmaniasis which is very rare in Malawi. Splenomegaly may be accompanied by hypersplenism with anaemia, low WBC, and low platelets

- \* FBC to exclude leukaemia and hypersplenism.
- \* Sickle cell test.
- \* If fever, do MPS and chest Xray.
- \* Abdominal ultrasound
- \* If lymphadenopathy do lymph node biopsy.
- \* Consider fine needle aspiration bopsy to rule out malignancy.
- \* If diagnosis remains unclear, give praziquantel and trial of malaria prophylaxis.

#### 3.14.4 Ascites

Diagnosed by finding abdominal distension with shifting dullness. May be a transudate (protein < 30 gm/l) or an exudate (protein > 30 gm/l).

##### CAUSES OF TRANSUDATES

- portal hypertension secondary to hepatic schistosomiasis, portal vein thrombosis or liver cirrhosis [4.10.13], hypoproteinaemia from malnutrition [4.2] and nephrotic syndrome [4.11.1] and cardiac disease [4.9.1] where there is severe cardiac failure or constrictive pericarditis.

##### CAUSES OF EXUDATES

- TB [4.5], infection (increased leukocytes in ascitic fluid) and malignancy such as Burkitt's lymphoma [4.17.1]. Lymphatic obstruction from TB and malignancy may give a chylous ascites (ascitic fluid looks like milk)

##### Investigations:

- \* Abdominal ultrasound
- \* Chest Xray.
- \* Ascitic tap [5.4.15] for protein, cells, acid fast bacilli.
- \* Urinalysis for protein to exclude nephrotic syndrome.
- \* Liver function tests.
- \* If diagnosis unclear - praziquantel and trial of TB treatment.
- \* If no response malignancy or chronic liver disease most likely.



### 3.14.5 Mass

Burkitt's lymphoma [4.17.1] often presents with irregular masses, that may involve spleen, liver, kidneys, abdominal lymph nodes or ovaries and there may also be jaw swelling. Renal masses may be caused by Wilm's tumour [4.17.4], neuroblastoma or hydronephrosis. A full bladder may result from bladder outlet obstruction or from neurological dysfunction as in paraplegia [4.12.8] so check the lower limb reflexes, power and tone. Occasionally TB lymphadenitis presents as irregular hard abdominal mass. Severe constipation with hard faecal masses is rare in Malawi.

- \* Ultrasound, and intravenous urogram (IVU) may help make the diagnosis.
- \* Urine culture, U&Es.
- \* Refer to surgeons if bladder distension or hydronephrosis.
- \* Fine needle aspiration [5.4.16] and trial of chemotherapy for suspected malignancy.

### 3.15 Weakness

Weakness of limb or limbs is an indication for referral. Acute weakness is urgent, but refer longstanding weakness to an outpatient clinic.

<b>History</b>	Speed of onset - Abrupt, rapid, slow, since birth Associated features - History of polio immunisation headache, mental retardation, fever, pain
<b>Examine</b>	Conscious level, meningism Temperature, heart rate, blood pressure Site - which limb(s) involved, level of weakness Tone - spastic or flaccid

**Hemiplegia** (Paralysis of arm, leg, and face on same side)

Hemiplegia of recent onset may be caused by malaria [4.1], meningitis [4.12.1], sickle cell disease [4.16.2], cerebral haemorrhage and emboli from congenital or rheumatic heart disease. However in most patients no cause is found. Children with hemiplegia should be considered for referral. Occasionally epileptics may experience a Todd's paralysis after a convulsion. This paralysis resolves fully within 24 hours.

Long-standing non-progressive hemiplegia may be the result of the above conditions or from birth trauma or asphyxia. Brain abscess [4.12.5] and

especially tumours [4.12.6] usually result in a slow and progressive deterioration, although acute presentation is possible when there is secondary hydrocephalus or haemorrhage into a tumour.

**Paraplegia [4.12.8]** (Paralysis of both legs - paraparesis is weakness) Paraplegia resulting from spinal cord compression is associated with spasticity of the limbs (increase tone and brisk reflexes). However there may be temporary flaccidity at the start. The most common non-traumatic cause in children in Malawi is Burkitt's lymphoma [4.17.1] with rapid onset. There may be accompanying jaw or abdominal swelling. Next most common is TB [4.5] where onset is slower with gibbus on examining the spine. In trauma the onset is abrupt at the time of injury.

**Beware a false history of trauma: preceding unrelated trauma may be said to be the cause by a relative wanting to explain the paraplegia. If trauma is the cause, weakness will occur at the time.**

Schistosomiasis [4.10.11] gives a slow onset and often the paraparesis is asymmetrical, epidural abscess gives a rapid onset with local tenderness over the spine, and in spina bifida paralysis is noted with the defect over the spine from birth.

If the limbs are floppy and reflexes are absent (flaccid paralysis), then poliomyelitis [4.7.3] is possible, though we hope it has been eradicated from Malawi, and Guillain-Barre syndrome and cauda equina damage are possible causes. In Guillain-Barre syndrome there is ascending paralysis of variable severity which usually recovers, but breathing may be impaired and the heart rhythm can be affected.

Children with recent onset paraplegia should be referred urgently for further investigation and acute flaccid paralysis has to be reported to the EPI programme [5.9].

- \* Consider lateral Xray of the thoracic spine.
- \* Consider LP (raised protein in Guillain-Barre, lymphocytosis in polio), but taking CSF from below a spinal lesion may make paraparesis complete or paraplegia permanent. Cisternal puncture may be wiser.
- \* Consider chest Xray.
- \* Myelogram, if available. (Delay LP till the time of the myelogram, if this is planned).

### Monoplegia (Paralysis of 1 arm or 1 leg)

Recent onset of weakness may be the result of polio [4.7.3], nerve damage (injection into the sciatic nerve) or pseudoparalysis (from fracture, dislocation, osteomyelitis, cellulitis, arthritis, congenital syphilis). Long-standing weakness of one limb may result from nerve damage at birth (Erb's or Klumpke's paralysis) or old polio.

### Floppy Child (General weakness without paralysis)

Hypotonia or floppiness without paralysis can be a feature of any acute illness. Down's syndrome and mental retardation commonly result in floppiness. Degenerative brain disorders may present with progressive generalised weakness. Children with protein energy malnutrition [4.2], rickets, and hypothyroidism [4.1.2] can all be floppy and lethargic. Developmental delay may be the presenting feature of such conditions and if the diagnosis is not obvious, and particularly if the condition is progressive, referral will be necessary. Remember HIV encephalopathy can present this way too.

## 3.16 Bone and Joint Complaints

Arthritis or arthralgia of recent onset needs urgent referral. Longstanding arthritis should be referred to an outpatient clinic.

<b>History</b>	Mode of onset - rapid or slow Site - which joints are involved
<b>Examine</b>	Swelling, warmth, pain, fever

NOTE: in arthritis there is joint pain with swelling, in arthralgia no swelling.

### Monoarticular Arthritis (One joint)

#### TRAUMA

Usually history of trauma with immediate pain and swelling of joint.

\* Xray joint or limb and seek an orthopaedic opinion.

#### SEPTIC ARTHRITIS [4.13.1]

Joint is red, hot and swollen, the child is ill, and joint aspirate gives pus.

\* Culture aspirate for salmonellae (in the wet season especially) or staphylococci.

\* Treat shoulder joint infections in children under 18 months with

chloramphenicol and gentamicin for salmonellae if very ill or oral ciprofloxacin, if there is only local disease. In infections of other joints, older children or in the dry season add flucloxacillin to gentamicin and chloramphenicol to cover staphylococci.

#### TB [4.5]

The symptoms usually develop over several weeks to months.

- \* Ask for history of TB contact.
- \* Xray joint.
- \* Aspirate joint [5.4.10] and culture for TB.
- \* Chest Xray.

Haemophilia and other bleeding diseases may cause haemarthrosis (blood in a joint) in elbows (infants) or the knees and ankles in a walking child.

- \* Ask about a family history of similar problems.
- \* Diagnosis depends on sophisticated investigations so refer for bleeding, clotting, prothrombin and partial thromboplastin time.

#### Hip

With hip problems pain may be referred to the knee. A limp in a younger child (under 5) may be due to transient synovitis (irritable hip) but the child should be admitted to exclude more serious conditions such as TB and septic arthritis. Perthe's disease, a form of avascular necrosis of the head of the femur, occurs in children aged 5-10 (and in sicklers) and slipped femoral epiphysis occurs in children over 10 years old.

- \* If high fever, refer for urgent joint exploration.
- \* If chronic, do hip and chest Xrays.
- \* Children with a painful hip should be referred for orthopaedic opinion.

#### Polyarticular Arthritis (several joints)

##### **Rheumatic fever** [4.9.2]

Arthritis is a major sign, whereas arthralgia is a minor sign of rheumatic fever. Typically the arthritis of rheumatic fever is flitting and involves ankle, wrist, knee and elbow joints. Look for other major criteria (carditis, chorea) and minor criteria (fever, streptococcal throat infection, raised ASO titre, past rheumatic fever, prolonged PR interval on ECG).

**Sickle cell disease** [4.16.2]

Younger children may present with swollen tender hands and feet ("hand foot syndrome"). The differential diagnosis includes syphilitic and tuberculous osteitis. Look for pallor and splenomegaly. Children with sickle cell disease are also at risk from septic arthritis and osteomyelitis.

**Syphilis**

Congenital syphilis may present with variably painful swollen limbs and joints. The age is typically between 4 months and 2 years. Do VDRL on mother and/or child.

**Other infections**

Meningococcal disease, rubella [4.7.4], infectious mononucleosis and brucellosis may cause a monoarticular or polyarticular arthritis.

**Juvenile rheumatoid arthritis** [4.13.4]

One or more large joints, or alternatively the small joints of the hand, may be mainly affected. The diagnosis is suggested by a long history of arthritis partly responsive to ibuprofen. Splenomegaly and fever may be noted.

### 3.17 Bleeding and Bleeding Disorders

Control epistaxis by firm pressure on the outside of the nostril that is bleeding, near the junction of cartilage and bone, for 5-10 minutes. Refer other types of bleeding urgently. Treat for shock if found [3.3]

<b>History</b>	Ask about - Bleeding from injections sites, mucous membranes and epistaxes. Any abnormal bleeding in male relatives (haemophilia) Past treatment with chloramphenicol or cotrimoxazole. Age - Congenital defects present earlier than acquired.
<b>Examine</b>	Check for shock [3.3]. Look at skin, mucous membranes including gums, <sup>1</sup> and conjunctivae, <sup>2</sup> for purpura, <sup>3</sup> petechiae, <sup>4</sup> ecchymoses, <sup>5</sup>

<sup>1</sup> Suggesting low platelets from marrow depression by drugs or in leukaemia.  
<sup>2</sup> Conjunctival haemorrhages occur in whooping cough [4.7.9] when venous pressure rises during coughing, in meningococcal septicaemia or meningitis [4.12.1] and in thrombocytopaenia [4.16.4].  
<sup>3&4</sup> Purpura is small round purple haemorrhages into the superficial layers of the skin. Petechiae are flame shaped bleeding points around small blood vessels in the skin and mucous membranes.  
<sup>5</sup> Ecchymoses are more extensive haemorrhages into skin and/or mucosa.

Associated features - Anaemia, lymphadenopathy, splenomegaly, weight loss.

**Local causes of bleeding** (more common)

Trauma such as wounds, dental extractions, circumcision, tonsillectomy, or secondary haemorrhage due to infection from umbilical cord stump and infected wounds, infected gums.

***Epistaxis***

in a child is usually due to his/her scratching blood vessels in Little's area of the nasal septum with a finger. It may also occur in a febrile illness such as URTI or typhoid.

- \* Apply local pressure for at least 10 minutes to wounds, do not rub otherwise this will dislodge the clot. Hold a bleeding nose firmly between thumb and forefinger.
- \* Treat infection if bleeding due to secondary haemorrhage.
- \* Occasionally a persistent epistaxis requires packing of the nose, usually just the anterior part in children, with gauze soaked in 1/1000 adrenaline solution.
- \* Rarely in epistaxis packing is not sufficient, and surgical cautery of the nasal septum is needed.
- \* Haematemesis may follow epistaxis, come from gastric erosions due to aspirin, or be from oesophageal varices (suggested by splenomegaly, hepatomegaly, ascites, jaundice).
- \* Melaena (altered blood in the stool) is from bleeding in the bowel (e.g. typhoid, peptic ulcer, Meckel's diverticulum).

**General clotting defects**

***Meningococcal septicaemia***

Petechiae or purpura, often with fever, sometimes meningitis or arthritis. Gram stain of scraping from lesion may show Gram negative diplococci. Treatment with intravenous drip and benzyl penicillin or chloramphenicol is URGENT as this condition can be rapidly fatal [4.12.1].

***Idiopathic thrombocytopaenic purpura*** [4.16.4]

Idiopathic thrombocytopaenic purpura (ITP) commonly presents between age 2-8 yrs. Platelet count is low (< 20,000), bleeding time prolonged and prothrombin time (PT) normal. Bone marrow shows megakaryocytes.

***Henoch Schonlein purpura*** [4.16.5]

FBC and platelets are normal. The purpuric rash is usually on the thighs, buttocks, and dorsa of feet, and there may be haematuria, and arthritis.

### 3 Clinical Presentations - Bleeding Disorders, Skin Lesions

#### **Haemorrhagic disease of the newborn [2.4.8]**

This is due to lack of vitamin K. Abnormal bleeding occurs on day 2-5 of life. Bleeding is often from the cord but may also occur as haematemesis, melaena, haematuria, oozing from injection sites, wounds (e.g. after circumcision) or intracranial bleeding. There is no purpuric rash. Prothrombin time is prolonged and platelets are normal.

#### **Haemophilia and Christmas Disease [4.16.3]**

These are hereditary diseases affecting males, but inherited from the mother's side. They present with bruising, haemarthroses after minor injury, and prolonged bleeding from incisions and dental extractions. Bleeding, clotting and prothrombin times are normal, but the partial thromboplastin time is prolonged, and corrected by factor VIII in Haemophilia and factor IX in Christmas Disease.

#### **Leukaemia [4.17.5]**

This is a malignancy of the blood. Normal blood cells, including platelets, are replaced by abnormal cells in the marrow. Patients are anaemic, have low or high WBC and are liable to infections and may bleed because of low platelets. A bone marrow examination is needed for diagnosis.

#### **Aplastic anaemia**

is suppression of bone marrow, by drugs such as chloramphenicol, cyclophosphamide, methotrexate or occasionally cotrimoxazole, or by Parvovirus B19 (in sicklers), or HIV, although often no cause is found.

## 3.18 Skin Lesions

### General approach<sup>1</sup>

#### **History**

##### ***When did lesions appear?***

**Congenital** - Usually noticed first in neonates, and include mongolian spot, haemangiomas, pigmented naevi.

**Acquired** - Appearing any time after birth, and are the majority of the skin lesions seen.

##### ***Is there itch or pain?***

**Intense itching** - Usually scratch marks on the skin. Commonly

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<sup>1</sup> Further details on the diagnosis of specific skin diseases in section 4.15.

caused by scabies (rule out in all cases of itching), chickenpox and onchocerciasis.

**Mild itching** - Eczema, tumbu fly, urticaria.

**Any preceding drugs?**

**Any systemic symptoms?**

**Examination**

**Where are the lesions?**

**Site** - Herpes simplex on lips, scabies between fingers, impetigo on scalp, face and around ears.

**Symmetrical** - Measles, pellagra, scabies, eczema.

**Asymmetrical** - Impetigo.

**Progression** - Chickenpox starts on the trunk, spreading to the face and limbs, while measles starts on the face, and going to the trunk later.

**How many lesions?**

**Many** - Usually form a rash e.g. measles, chickenpox, urticaria, also common papular rash seen in HIV infection, called papular pruritic eruptions or PPE.

**Few** - Are seen as isolated lesions e.g. ringworm, leprosy.

**Size and shape?**

**Small** - Heat rash, measles and chickenpox cause many small lesions.

**Large** - Leprosy and ringworm cause larger patches.

**Shape** - Most are round or nearly round, e.g. ringworm. Special shapes include shingles which is shaped according to the dermatome it occurs in, and larva migrans which is shaped like a worm.

**What are the edges like?**

**Well defined** - Pellagra, ringworm, leprosy, chronic ulcer.

**Poorly defined** - Eczema, kwashiorkor.

**Are lesions wet or dry?**

**Wet** - Acute eczema or impetigo (before crusts form).

**Dry** - Ringworm.

**Are they flat or raised?**

**Flat** (macules) - Vitiligo, pityriasis rosea.

**Raised** (papules) - Warts, measles, papular urticaria.

**Containing clear fluid** (vesicle) - Chickenpox and shingles (change from macule to papule to vesicle to pustule to scab).

**Containing pus** (pustule) - Impetigo, staphylococcal infections.



### 3 Clinical Presentations - Skin Lesions, Developmental Delay

#### **What is the colour compared to the healthy surrounding skin?**

**Red** - Commonly due to erythema<sup>1</sup> e.g. eczema. Rarer causes are purpura and petechiae<sup>2</sup> which are usually more serious.

**Paler** - Vitiligo, leprosy.

**Darker** - Single or few patches of darker pigmentation are benign "cafe au lait patches." Multiple patches can be neurofibromatosis which is a rare potentially serious hereditary condition.

## 3.19 Developmental Delay

Early identification<sup>3</sup> of treatable causes of delayed development is vital e.g. congenital cataracts, hypothyroidism. There is a critical period of development after which skills such as speech and sight may never be "learned." HIV infection should always be ruled out where there is no obvious cause.

### Delay in talking<sup>4</sup>

#### **Normal variation**

A few children of normal intelligence do not speak till their 3rd birthday. They appear alert and interested and will point to pictures of named objects and carry out instructions.

#### **Deafness**

May be congenital or acquired, complete or partial, and can seriously interfere with speech. Test the response of the child to sounds, including speech, both low and high pitched. The child must NOT see the sounds being made. Notice, if some speech is present, which sounds are defective (high tone deafness leads to failure to hear and therefore make "s" and "t" sounds and consonants at the ends of words). Acquired deafness may be caused by severe neonatal jaundice, meningitis, cerebral malaria or chronic otitis media.

#### **Mental retardation**

Other developmental milestones (walking, handling objects) are usually

<sup>1</sup> Secondary to inflammation becomes paler if pressed on with a glass slide.

<sup>2</sup> Remain red when pressed on with a glass slide.

<sup>3</sup> For normal milestones see section [1.3]

<sup>4</sup> Sometimes called "tongue tie" due to a misunderstanding of the cause.

also delayed, though not as much as speech. There may be Down's syndrome [2.5.4], microcephaly, hydrocephalus [4.12.2]. A history of birth asphyxia, abnormal neonatal crying or sucking, severe neonatal jaundice, cerebral malaria, meningitis, hypoglycaemia or severe dehydration may suggest the cause of brain damage.

### **Emotional deprivation**

May cause developmental delay, and is diagnosed from the social history, e.g. in orphans

### **Malnutrition**

Children who are seriously underweight, especially if they have kwashiorkor, will have delayed speech. This rather than the more obvious signs of malnutrition may be the presenting complaint.

### **Autism**

A child with this condition does not seem to respond well to other people. Speech, our main way of communicating, may be delayed or even absent, and they tend to like peculiar repetitive activities.

### **Delay in walking**

#### **Normal variation**

While on average, a child first walks at 13 months, there is considerable variation. Most normal children walk alone by 18 months.

#### **Malnutrition [4.2]**

Malnourished children are ill, apathetic, inactive, usually easily diagnosed.

#### **Cerebral palsy [4.12.7]**

Caused by brain damage, usually perinatal, resulting in hemiplegic or diplegic abnormalities. Affected muscles are usually spastic, but may be hypotonic. Occasionally there are abnormal movements (athetoid cerebral palsy). The diagnosis is made by looking for asymmetry of limb movements (upper and lower), and testing muscle tone and reflexes.

### *3 Clinical Presentations - Developmental Delay*

#### **Polio [4.7.3]**

Causes a hypotonic (flaccid) paralysis or weakness with depressed tendon reflexes and muscle wasting. Usually paralysis is asymmetrical, and larger muscles of the trunk and limb girdles are more severely affected than muscles of the hands.

#### **Chronic disease**

Many chronic diseases delay walking. e.g. cardiac disease, TB.

#### **Emotional deprivation**

The child who is not played with may show delay in walking, e.g. neglected babies and orphans.

#### **Mental retardation**

Is often, but not always, associated with delay in walking, and may also be associated with microcephaly, cerebral palsy and deafness (e.g. congenital Rubella). Usually hand use and speech are more delayed than walking, but Down's syndrome patients are often hypotonic so late to walk for two reasons.

#### **Blindness**

Leads to delay in walking, but the blindness itself is usually the presenting problem.

## 4 DISEASE MANAGEMENT

### 4.1 Malaria

The most common cause of disease and death in Malawian children, malaria is caused by protozoa called Plasmodia that infect man through the bites of anopheles mosquitoes (rarely from blood transfusion). Of four species, the most dangerous and most common in Malawi is *P. falciparum*, which can cause severe haemolytic anaemia and cerebral malaria but more usually fever, headache and malaise. The interval from infection to illness is usually 10, but may be as short as 7 days.

#### Standard Treatment

With increased parasite resistance to sulphadoxine-pyrimethamine (SP), the first line treatment in Malawi for uncomplicated malaria is now lumefantrine-artemether (commonly known as Coartem). This comes in combined tablets of 20 mg artemesinin and 120 mg lumefantrine. Its absorption is improved if taken with fatty food, such as milk.

**Dose:** aim at 12-18 mg/kg/dose of the combination twice daily for 3 days.

This corresponds to:

- 1 tablet a dose for children of 5-14 kg (age under 3),
- 2 tablets for children of 15-24 kg (age 3-8),
- 3 tablets for children of 25-34 kg (age 9-14) and
- 4 tablets for children over 35 kg and adults.

#### Problems with lumefantrine-artemesinin (LA):

Vomiting - this may occur, especially in a febrile child. Repeat the LA dose, if a child vomits within an hour of taking a dose.

No serious side effects are known, but dizziness, palpitations, abdominal, muscle and joint pain, headache, and skin rash may occur.

At present resistance to LA is unlikely, but if blood films from a child not improving clinically show asexual (ring) forms of malaria over 48 hours after treatment is started, give second line treatment with amodiaquine and artesunate, which come as tablets of 100 mg amodiaquine and 40 mg of artesunate. Aim at 10 mg/kg/dose of amodiaquine and 4 mg/kg/dose of artesunate once daily for 3 days.

So for each of amodiaquine and artesunate the dose is:

- 1/2 tablet for babies of 5-6.4 kg (under 6 months),
- 1 tablet for children of 6.5-11.9 kg (6/12 to 2 years),
- 2 tablets for children of 12-24.9 kg (age 2-8 years),
- 3 tablets for children of 25-34.9 kg (age 9-14) and
- 4 tablets for children over 35 kg and adults.

### Severe Malaria

Malaria is severe when the child's life is at risk. In Malawi the two most common forms of severe malaria are severe anaemia and cerebral malaria.

Watch out for the following danger signs:-

- \* the child is too weak or sick to take medicine by mouth
- \* the child has persisting reduced level of consciousness (Blantyre coma score) suggesting cerebral malaria.
- \* the child has more than 1 convulsion within 24 hours
- \* there is severe anaemia (Hb < 5 g/dl or HCT < 18%)

#### Blantyre Coma Score

SCORE	MOTOR RESPONSE TO PAIN	VERBAL RESPONSE	EYES TO OBJECT
2	Localises pain	Appropriate cry	
1	Withdraws limb	Inappropriate	Directed gaze
0	None	None	Do not follow

Total score maximum is 5 for fully conscious

### Management of Cerebral Malaria

- \* Give quinine IV initially in a dose of 20 mg/kg/dose<sup>1</sup> in 10 ml/kg 5% dextrose (or half Darrow's dextrose) over 3-4 hours, followed every 12 hours by IV quinine 10 mg/kg/dose (in 5% dextrose) over 3 hours. When patient numbers are too great for nursing staff available to manage many IV drips, IM quinine 10 mg/kg stat and at 4 and 12 hours may be a better option, but beware the risk of hypoglycaemia.<sup>2</sup> When the child can take orally, stop quinine and give LA.
- \* Give anticonvulsants to control fits (paraldehyde or diazepam to arrest

<sup>1</sup> When giving quinine at this high loading dose, check patient has not previously had quinine IM before arriving. If he has within the last 8 hours, give 10 mg/kg/dose IV over 3 hours.

<sup>2</sup> See National Guidelines on Malaria Management, 2008

a convulsion, and phenobarbitone to prevent more fits) [3.4].

- \* Look for and treat hypoglycaemia (give 1.0 ml/kg of 50% glucose, best diluted with 4 ml/kg of 5% dextrose, IV stat, then continue 5% dextrose or 1/2 Darrow's dextrose to prevent rebound hypoglycaemia).
- \* If working in a health centre, give the first dose of quinine (10 mg/kg)<sup>1</sup> IM stat, before transferring the patient to hospital<sup>2</sup>. In preparing quinine injection, draw up the contents of a 300 mg ampoule (1 ml) of quinine into a syringe containing 5 ml of sterile water or saline to give 50 mg/ml. The same dose should be repeated after 12 hours if the patient is still not transferred.
- \* Give good nursing care, including fluids appropriate for an unconscious patient IV or by naso-gastric tube, and physiotherapy to prevent contractures in spastic limbs and hypostatic pneumonia. Teach the guardian physiotherapy if nurses are few.
- \* During recovery of all severe malaria children teach guardians the LA childhood dosage and emphasise the importance of taking a child to a health unit at the first appearance if any of the danger signs above.
- \* Record a coma score

### Prophylaxis

This is recommended for certain groups of children:

- Expatriates from non-malarial countries.
- Those with sickle cell disease, since already prone to haemolysis.
- Children with recurrent (> 3) episodes of febrile convulsions, until they are over 6 years old.
- Those who have had splenectomy
- Those who are immunosuppressed from cytotoxic drugs.
- Patients with hyperreactive malarial splenomegaly (tropical splenomegaly syndrome).

The recommended drugs for prophylaxis in categories of Malawian children who need it is proguanil (Paludrine) daily and chloroquine weekly. Do not give routine malaria prophylaxis to the general public, as this is likely to promote drug resistance through drug pressure.

<sup>1</sup> If the child cannot be weighed estimate the weight by use of the charts in the appendix [5.1.3] or use the formula "weight (in kg) = age (in years) × 2 + 8."

<sup>2</sup> If you do this, state in the referral letter the time the dose was given, as there is a risk that, if given without dextrose, the quinine may induce hypoglycaemia, and the receiving officer should know when to give the next dose of quinine IV.



**Daily dose of proguanil for prevention:**

- 1/4 tablet for babies under 1 year, (or for children not yet walking),
- 1/2 tablet for children aged 1 to 4, (or children walking but not at school)
- 1 tablet for children of 5 to 8 years,
- 1 1/2 tablets for children of 9 to 14 years,
- 2 tablets for older children and adults.

**Weekly dose of chloroquine for prevention**

- 1/4 tablet under 6 months, (or for children not yet sitting)
- 1/2 from 6 months to 2 years (for children sitting but not walking),
- 1 tablet 2 years to 8 years (or for children walking but not at school),
- 1 1/2 tablets 9 years to 14 years (or for children at school),
- 2 tablets over 14 years and adults.

## 4.2 Malnutrition

Malnutrition affects many children in Malawi [1.1.1]. Malnutrition results from inadequate quantity and/or quality of food. The major form of malnutrition is Protein Energy Malnutrition (PEM), which is the result of insufficient energy intake for the growing child, with lack of protein, but also minerals and vitamins, especially iron and vitamin A.

Malnourished children have weight loss, and most will be below the path to health in the health passport, unless the child was previously very large, or has very marked oedema.

Severe PEM is diagnosed when there is bilateral nutritional oedema or the weight for height of the child < 70% of normal [See weight for height table section 5] These criteria are preferred to older classifications<sup>1</sup> of severe malnutrition based on weight for age, mainly because weight for

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<sup>1</sup> Older categories were

Marasmus - < 60% of expected weight for age (WFA).

Kwashiorkor - < 80% of expected WFA with oedema.

Severe marasmus - < 50% of expected WFA.

Marasmic kwashiorkor - < 60% of expected WFA with oedema.

Children with marasmus have no oedema, whereas those with kwashiorkor do. In kwashiorkor the amount of oedema varies in severity from feet only, to generalised oedema with ascites. There is loss of hair pigment and the skin may be peeling and have areas of hypo- and hyper-pigmentation and ulceration. Other typical features of kwashiorkor are apathy, misery and diarrhoea.



height reflects acute malnutrition, which requires medical attention, whereas low weight for age can be due either to acute or chronic underfeeding, the latter causing stunting in height, more a social and economic problem. Weight for height also avoids uncertainties over ages of children whose dates of birth are unknown, but the disadvantage is the difficulty of measuring height accurately.

### The Energy Needs of Healthy Children

100 kcal/kg/day in infants and toddlers

88 kcal/kg/day at 6 years

72 kcal/kg/day at 10 years

In children with bilateral oedema exclude causes such as nephritis, nephrotic syndrome, and heart failure, and in children without oedema but failing to thrive chronic heart [4.9.1 & 4.9.5] or renal disease [4.11]. In all malnourished children check history and examination for evidence of tuberculosis [4.5] and HIV infection [4.3]. Assess whether malnutrition is severe, requiring admission or can be treated at a nutrition clinic. Less severely malnourished children need extra feeding to bring daily calorie intake to over 100 Kcal/kg/day, with careful education of guardians on how to improve feeding, especially on the monitoring and need to continue feeding of children who have diarrhoea or infections. Also educate parents on child spacing. Maintain breast feeding whenever possible, when mother is HIV negative. Treat any intercurrent infections appropriately, and immunise fully, especially against measles, to prevent cross-infection at nutrition clinics. Income generating projects may be valuable, but are beyond the scope of this Handbook.

### Energy Content of Some Foods

Full strength milk	80 kcal/100 ml
F75	77 kcal/100 ml
F100	100 kcal/100 ml

### Severe Protein Energy Malnutrition (PEM)

Severe PEM is diagnosed when there is bilateral nutritional oedema of any degree, or weight for height is below 70%. This requires admission to a nutritional rehabilitation unit or ward.

The history must cover birth weight, feeding and weaning, past illnesses, family health and social, educational and economic circumstances.

Initial assessment requires an accurate weight, height, mid upper arm circumference (MUAC), temperature, pulse and breathing rates. Assess the coma score and mental state for alertness or apathy and appetite. Record the degree of oedema, presence of any skin ulceration, presence and severity of vomiting and diarrhoea and nature of stools. Examine eyes for xerophthalmia and keratomalacia (vitamin A deficiency), mouth for stomatitis, mucous membranes, nail beds and palms for pallor, skin for infections and scabies, lymph nodes and parotids for enlargement, breathing for cough, respiratory rate and dyspnoea and heart for rate and any murmurs or gallop rhythm.

Be particularly careful in assessing dehydration. In malnourished children loose skin and sunken eyes may be due to loss of fat, and dry mouth from mouth breathing, so these signs can be misleading. Malnourished children generally retain extra sodium and water, so only give ReSoMal or IV fluids for dehydration if the guardian gives a history of four or more watery stools a day recently and clearly says the eyes have become more sunken lately. Giving extra fluids, especially IV, has great dangers if there is no shock of causing pulmonary oedema and killing the child.

In managing severely malnourished children, treat any intercurrent infections, and treat for hypoglycaemia<sup>1</sup> and septicaemia any child with hypothermia, depressed coma score or hypovolaemic shock (slow capillary refill [3.3], cold extremities, weak fast pulse). Treatment is I.M. broad spectrum antibiotics (e.g. ampicillin and gentamicin, or chloramphenicol, cefotaxime or ceftriaxone), sugar water by mouth or NG tube, and rest.

If you are sure there is shock from recent diarrhoea, give fluids for dehydration cautiously, and preferably use oral ReSoMal [5.5]. First mark the liver edge on the abdomen and record weight, heart rate and sounds and respiratory rate, and monitor these half hourly to assess if dehydration is improving or pulmonary oedema developing. (Stop extra fluids when weight lost since diarrhoea started has been regained, or if liver size increases by 1 cm, the breathing rate increases by 5/minute, if dyspnoea

<sup>1</sup> Treat hypoglycaemia with 1.0 ml/kg of 50% glucose, preferably diluted with 4 ml/kg of 5% dextrose, IV stat, followed by 5% dextrose or half Darrow's dextrose drip to prevent rebound hypoglycaemia.

#### 4 Disease Management - Malnutrition

develops or a triple heart rhythm). But preferably give no extra fluids and start on F75 [5.6] feeds aiming to give at least 80% of the target volumes initially (see table below). If the child fails to take this, tube feed until the child takes target volumes. After an initial period (usually about 5 days) when oedema is lost (in kwashiorkor) or weight begins to rise (in marasmus) change to F100 [5.6] feeds. Continue breast feeding if possible where mother is HIV negative. Also give vitamin A 100,000 units once in babies under 6 months, 100,000 units on days 1, 3 and 14 to those between 6 and 12 months, and 200,000 units on days 1, 3 and 14 to children over 1 year and folic acid 5 mg once to all. Put all children with severe PEM without signs of infection, on oral amoxicillin till 4 days after the initial F75 period. Check weight, oedema and respiratory rate and record any diarrhoea daily, temperature twice daily, and MUAC weekly.

#### Target volumes of F75 feeds for children of different weights

<u>Weight</u>	<u>Vol/kg/day</u>	<u>Vol/24 hours</u>	<u>Vol/feedx8/day</u>	<u>Vol/feedx6/day</u>
2.2 kg	150 ml	300 ml	35 ml	(50 ml)
2.25 kg	150 ml	350 ml	40 ml	(60 ml)
2.5 kg	150 ml	375 ml	45 ml	(60 ml)
3.0 kg	145 ml	430 ml	50 ml	(70 ml)
3.5 kg	140 ml	490 ml	60 ml	80 ml
4.0 kg	140 ml	560 ml	70 ml	90 ml
4.5 kg	135 ml	600 ml	75 ml	100 ml
5.0 kg	135 ml	650 ml	80 ml	110 ml
5.5 kg	135 ml	750 ml	90 ml	125 ml
6.0 kg	135 ml	800 ml	100 ml	135 ml
7-8 kg	130 ml	1000 ml	125 ml	160 ml
8-9 kg	130 ml	1200 ml	150 ml	200 ml
9-10 kg	125 ml	1300 ml	165 ml	215 ml
10-11 kg	120 ml	1350 ml	170 ml	225 ml
11-12 kg	120 ml	1400 ml	175 ml	230 ml
12-13 kg	120 ml	1500 ml	190 ml	250 ml
13-14 kg	120 ml	1600 ml	200 ml	265 ml
14-15 kg	120 ml	1750 ml	215 ml	290 ml

#### Nasogastric tube (NGT) feeding.

Mothers often resist NGT feeding. Their experience has shown them that children with NGTs frequently die. This is true if NGTs are inserted only

in terminally ill children, but the high mortality is not related to the NGT. An NGT should be inserted as soon as it is seen that the child's calorie intake is insufficient. The minimum requirement is about 75 Kcal/kg/day, which is equivalent to about 100 ml/kg/day of F75. Experience has shown that mothers accept the NGT, and even ask for it, if an NGT is inserted in all seriously malnourished children on admission, because they soon see its good effect.

### Complications of PEM

- \* Diarrhoea and vomiting: continue feeding, add ReSoMal 30 ml/watery stool only if there is good evidence of dehydration (weight loss, signs of shock, eyelid retraction showing white sclera below the iris during sleep).
- \* Infection (often without fever in PEM): treat any infection - pneumonia [4.8.14], or septicaemia with benzyl penicillin 50,000 units/kg 6 hourly and gentamicin 7.5 mg/kg/dose once daily, or chloramphenicol 25 mg/kg q.i.d.
- \* Hypoglycaemia (often with sepsis, sometimes after starting rifampicin if also suffering from TB): give 1.0 ml/kg 50% dextrose, preferably diluted with 4 ml/kg 5% dextrose, IV stat, continue a slow 5% dextrose drip, give frequent feeds and treat for sepsis. Where rifampicin has precipitated hypoglycaemia, give prednisolone 5 mg daily for 2 weeks.
- \* Hypothermia (often with sepsis): warm up and keep warm at night, treat for sepsis and hypoglycaemia.
- \* Hypokalaemia (can give ileus, heart failure): give high potassium fruits, occasionally IV KCl 20 mmol in 1 litre of 5% dextrose at 20 ml/kg over 4 hours.

**Never give KCl undiluted IV - beware sudden heart arrest!**

- \* Anaemia: give folic acid and ferrous sulphate when oedema has gone. Rarely is anaemia severe enough in PEM for transfusion, which has to be given very cautiously and not more than 10 ml/kg.
- \* Heart failure (in recovery from kwashiorkor from rapid reabsorption of

**Severely malnourished children often rapidly deteriorate, so regular ward rounds by medical staff are essential.**

oedema): do not give ORS or IV fluids, limit intake to 80 ml/kg/day, and give frusemide (1 mg/kg/dose daily or b.d. orally) and KCl (1 mmol/kg/dose q.i.d. orally).

\* Cancrum oris: treat with penicillin and metronidazole, and refer for surgery when weight is > 80% weight for height.

After the initial phase, i.e. when all oedema has gone in kwashiorkor and appetite has returned, or when a marasmic child has started to gain weight, change feeds from F75 to F100 or "Chiponde," [5.6], which will increase the calorie intake for the same volume. Once the child is taking the target volume of F100 without any complications such as fever, diarrhoea, dyspnoea or tachycardia, allow the child to eat as much as wanted, to give rapid gain. Chiponde can be continued on discharge.

#### Discharge Criteria:

Often family and social conditions require flexibility in the timing of discharge, but oedema must have disappeared in kwashiorkor with gain of weight to 80% weight for height. Make good arrangements for outpatient follow up and food supplements.

### 4.3 AIDS (Acquired Immuno-Deficiency Syndrome)

Since the first edition of this Handbook, prevalence of AIDS and its impact on children have increased immensely, but the coming of anti-retroviral therapy (ART) has injected hope into a previously disastrous situation and that has changed AIDS from an almost taboo subject to allow much freer discussion, as it can be treated, though not cured. AIDS is a disease of damaged immunity, caused by infection with the Human Immunodeficiency Virus (HIV), and leading to complicating infections, often with *Streptococcus pneumoniae*, *salmonellae* or TB, but sometimes unusual organisms (opportunistic infections) and some tumours. AIDS implies infection with HIV, but there is in addition immune deficiency. ART is not of proven value for HIV infection until immunity is impaired to the extent that AIDS can be diagnosed. It is therefore very important to define the basis of a diagnosis of AIDS in a patient, before considering ART. In young children this is more difficult than in adults. For a fuller discussion, you should consult the current edition of the Ministry of Health Guidelines on the Treatment of AIDS. Here we summarise the most relevant aspects of diagnosis and treatment for paediatrics in Malawi.

HIV's genetic material is a ribonucleic acid (RNA) which is surrounded by a protein coat. It has a cycle of reproduction that involves several stages of attaching to receptors (mainly) on CD4 lymphocytes, fusing with the cell membrane, passing into the cell, penetrating the nucleus, producing a deoxyribonucleic acid (DNA) mirror image that integrates into the cell DNA, and then reorganising the chemistry of the cell to produce and release many virus particles. Antiretroviral drugs (ARVs) interfere with different stages of this cycle, especially the action of reverse transcriptase which makes the DNA mirror image from HIV RNA. RNA is much less stable than DNA in our genes, and therefore it mutates rapidly, which is particularly important in the development of variants that resist ARVs.

Infection with HIV is acquired by children most often from the mother through the placenta or at birth, but may come through breast milk, blood transfusion, unsterile injections, traditional incisions, sexual abuse, or, in older children, early sexual activity. Infection is followed, after about 6-12 weeks in most adults and older children, by the presence of antibodies to the virus, but children under 18 months old may have acquired antibodies through the placenta from the mother before birth. The diagnosis of HIV infection in children may be suspected from the family and social history and from abnormalities in the history and examination that are found in different stages of HIV infection as set out below, but must be confirmed by a combination of tests for antibodies to HIV.

The diagnosis of HIV has many serious health, family and social consequences, and full counselling must be given to guardians and to (older) children as well in terms they can understand. Remember HIV infection is a sensitive, confidential issue, and guardians should be counselled privately and compassionately about it. There is a need to listen, explore their knowledge and fears and to be non-judgmental. It is important to inform guardians of the availability of ART, to give hope in what is otherwise a very negative situation. The diagnosis of HIV infection may lead to ART, but only when there is a diagnosis of AIDS (or in children under 18 months of presumptive AIDS, as finding HIV antibodies is not conclusive for HIV infection at that age). Criteria for diagnosing AIDS are set out below. In most of Malawi clinical features will be used to stage immune deficiency, but laboratory counts of the CD4 lymphocytes, those primarily damaged by HIV and involved in immunity,

#### 4 Disease Management - AIDS

can help. Where CD4 counts cannot be done, a total and differential white count to give a total lymphocyte count can help indicate immunodeficiency.

#### Stages of HIV infection in its progress to AIDS in children

Stage 1 The infection has not damaged immunity no symptoms or persistent generalised lymphadenopathy (PGL)

Stage 2 Minor signs of impaired immunity:

skin papules, extensive warts, or molluscum contagiosum, repeated mouth ulcers,  
firm, non-tender parotid enlargement, herpes zoster, repeated or chronic upper respiratory tract infections, fungal nail infections.

Stage 3 Moderate signs of impaired immunity:

weight persisting below the normal range (health passport weight chart) for 3 months in spite of extra feeding and without other explanation (such as congenital heart disease or other abnormality),  
unexplained persistent diarrhoea for over 2 weeks,  
unexplained fever over 37.5°C for over a month,  
persistent oral thrush after 8 weeks old and without preceding antibiotic treatment, oral hairy leukoplakia,  
ulcerating gums,  
TB lymphadenopathy, pulmonary TB,  
severe recurrent pneumonia, lymphoid interstitial pneumonitis,  
neutropaenia (< 500/c.mm.) or low platelets (< 50,000/c.mm.)

Stage 4 Severely impaired immunity – findings that strongly suggest AIDS:

Seriously under weight – below 70% weight for age or height, or oedema both feet in spite of treatment and feeding  
or Pneumocystis carinii pneumonia (PCP)  
or repeated severe bacterial infections other than pneumonia or cryptosporidiosis or isosporiasis with diarrhoea over 4 weeks  
or cryptococcal meningitis  
or cytomegalovirus infection of retina  
or thrush (candidiasis) extending to oesophagus or trachea  
or extrapulmonary TB other than lymphadenopathy

or Kaposi's sarcoma (not Burkitt's lymphoma, which is usually unrelated to AIDS).

Where specialised laboratory investigations are available the CD4 count can be used to define immune staging. For children over 5 years, the total CD4 cell count is used, but below 5 years the percentage of lymphocytes that are CD4 cells is used.

<u>Stage</u>	<u>Lympho</u> <u>count</u>	<u>CD4</u> <u>count</u>	<u>&gt; 5 years</u>	<u>5-3 years</u>	<u>3-1 years</u>	<u>&lt; 1 year</u>
1	< 2000		> 500	> 25%	> 30%	> 35%
2	< 2500		499 - 350	25 - 20%	30 - 25%	35 - 30%
3	< 3000		349 - 250	20 - 15%	25 - 20%	30 - 25%
4	< 4000		< 250	< 15%	< 20%	< 25%

#### Criteria for considering ART in children

HIV positive children over 18 months old who are in stage 4, or in stage 3 (especially with a low CD4 count, or low total lymphocyte count, if CD4 counts are unavailable) or stage 2 with a low CD4 or total lymphocyte count or who are PCR positive [2.4.7.1].

PCR negative HIV positive ill children under 18 months may be thought to have "presumptive AIDS," but the HIV test must be rechecked after 18 months. They qualify for presumptive AIDS with a positive HIV test and being in stage 4 or having oral candidiasis (after age 2 months and without prior antibiotics) and either severe pneumonia or severe sepsis.

**Don't forget the mother needs counselling, advice and perhaps treatment and contraception, when you are treating a child.**

#### General Management

Treat complications (TB [4.5], thrush [4.10.7], pneumonia [4.8.14], meningitis [4.12.1], infective diarrhea [4.10.2], cryptococcal meningitis [4.12.2], Kaposi's sarcoma [4.17.2]) and malnutrition [4.2]) first, as set out elsewhere in this handbook.

Do not start ART as an emergency, especially if TB is diagnosed as rifampicin and one of the anti-retroviral drugs (ARVs), nevirapine (NVR) interact.

Immunise children with HIV infection with pentavalent, polio and measles vaccine, unless already very ill. BCG vaccine is controversial, but it will usually have been given before the diagnosis is reached.



#### 4 Disease Management - AIDS

Pay special attention to nutrition (watch growth), hygiene, and avoidance of contact with infectious illnesses, such as chickenpox and measles.

Where possible treat children with HIV infection as outpatients to minimise the risks of cross-infection.

Give all infants of HIV positive mothers from 6 weeks old and all HIV positive children cotrimoxazole in appropriate daily dosage to prevent *Pneumocystis carinii* pneumonia (PCP). If a child with AIDS has a reaction to cotrimoxazole, give daily dapsone.

#### Specific Treatment - anti-retroviral therapy (ART)

ART aims to prolong good quality of life and to allow normal or nearly normal growth and development and education (in school age children).

**ART must only be given by fully ART trained and certified personnel.**

Staff running the ART clinic must have had full training in all details of such clinics and be certified for ART.

Antiretroviral drugs (ARVs) must always be given in combination to slow down the development of resistance to individual drugs. Treatment must be carefully supervised, documented and there must be regular counselling of the guardian and the child, if old enough to understand.

The standard starting combination of drugs is Stavudine (d4T), Lamivudine (3TC) and nevirapine (NVP), normally in a combined tablet called "Triomune." NVP must be started cautiously in once daily dosage for the first 2 weeks, because of the risk of severe skin reactions on higher initial dosage. So for the first two weeks combined d4T, 3TC and NVP is given each morning and combined d4T and 3TC in the evening. If there are no toxic reactions after the first 2 weeks the three drug combination is given twice daily, a month's treatment being issued at a time.

Dosages are given in the accompanying table: tablets used contain d4T 30 mg, 3TC 150 mg with or without NVP 200 mg.

**Weigh the child on ART at every clinic visit so that drug doses are carefully adjusted.**

<u>Weight Range</u>	<u>No. of tablets</u>	<u>Daily d4T aim 2 mg/kg</u>	<u>Daily 3TC aim 8 mg/kg</u>	<u>Daily NVP aim 7 mg/kg</u>
< 5 kg	1/4 once daily	1.5 mg/kg	7.5 mg/kg	10 mg/kg
5 - 8 kg	1/4 twice daily	1.9 - 3	9.4 - 15	6.25 - 10
8 - 12 kg	1/2 a.m., 1/4 p.m.	1.9 - 2.8	9.4 - 14.1	4.2 - 6.25
12- 14 kg	1/2 a.m., 1/2 p.m.	2.1 - 2.5	10.7 - 12.5	7.1 - 8.3
14 - 19 kg	3/4 a.m., 1/2 p.m.	2 - 2.7	9.9 - 13.4	5.3 - 7.1
19 - 26 kg	3/4 a.m., 3/4 p.m.	1.8 - 2.4	8.7 - 11.8	5.8 - 7.9
26 - 30 kg	1 a.m., 3/4 p.m.	1.8 - 2	8.8 - 10.1	5 - 5.8
> 30 kg	1 a.m., 1 p.m.	< 2.0 mg/kg	< 10 mg/kg	< 6.7 mg/kg

### Drug toxicity is a significant problem.

Skin reactions and jaundice may be caused by NVP (sometimes severe Stevens-Johnson syndrome, especially if NVP is started in high dosage) and will then require replacement of NVP by Efavirenz (EFV) but this requires extra care as EFV may also give skin reactions, and its safety in children under 3 years old has not been established, so in them NVP toxicity may require ART to be stopped. If there is a severe skin reaction or jaundice, stop Triomune, but for 2 weeks continue d4T and 3TC without NVP, since NVP has a long half life and will persist, effectively giving monotherapy.

Peripheral neuropathy and less often pancreatitis and lactic acidosis and lipodystrophy may be caused by d4T. Mild peripheral neuropathy may be managed with amitriptyline, with additional phenytoin or carbamazepine if necessary, but more severe neuropathy, pancreatitis and lactic acidosis require a change from d4T to zidovudine (AZT), which has the serious risk of causing anaemia.

Fortunately 3TC seldom gives toxicity, and mainly gastrointestinal symptoms that can be managed symptomatically.

### Drug interactions are important to remember.

NVP interacts with rifampicin, NVP metabolism being increased, with greater risk of viral drug resistance developing, so ART should generally not be started till 2 weeks after the rifampicin phase of TB treatment is finished. But with the new use of rifampicin (with isoniazid) for maintenance TB treatment, Triomune may be started at that stage in full dosage, without the initial 2 weeks of reduced dosage NVP as rifampicin will reduce NVP blood levels. (But when giving INH in patients on ART

remember to give pyridoxine to reduce peripheral neuropathy risk from possible interaction of d4T and INH). NVP also interacts with ketoconazole so the drugs should not be given together.

d4T and AZT are pharmacologically incompatible and must not be given together.

Drug resistance can only be diagnosed if a child has been complying fully with ART for at least 6 months and has either developed a new AIDS clinical stage 4 feature or has a fall in CD4 count to below 50% of its peak value. If drug resistance is suspected specialist advice must be sought. The child may then, if resistance is definite, be changed to combined didanosine (ddI) with abacavir (ABC) and perhaps AZT (in which case regular Hb checks are needed) or lopinavir/ritonavir (LPV/r) (Kaletra).

## 4.4 AIDS related syndromes and diseases

A number of conditions and diseases are associated with HIV/AIDS: their presence may suggest HIV infection or in some cases define the stage of AIDS. The list below is not comprehensive but concentrates on conditions diagnosable in Malawi.

### 4.4.1 Persistent Generalised Lymphadenopathy (PGL)

To qualify for this diagnosis lymphadenopathy must involve at least 3 groups of nodes, be symmetrical, be non-tender with at least 2 nodes over 1.5 cm in diameter and last over a month. As PGA occurs at an early stage of HIV infection, there are often no systemic symptoms like fever, weight loss or diarrhoea.

It is important to remember that a number of more minor viral infections may cause lymphadenopathy, especially involving the suboccipital and submandibular nodes, including Rubella [4.7.4], infectious mononucleosis, occasionally hepatitis A [3.10].

Tuberculosis in children quite often causes non-tender lymph node enlargement, especially of the cervical nodes, but occasionally elsewhere and rarely general lymphadenopathy. In the later stages, the nodes become matted and may soften and drain. BCG can sometimes lead to local non-tender lymph node enlargement, usually in the axilla.

Other bacterial infections usually lead to tender lymphadenopathy,

often in one or two groups of nodes related to a primary skin focus of infection, as can the organisms of lymphogranuloma venereum, syphilis, plague, and various borreliae and rickettsiae and trypanosomiasis may be associated with posterior cervical adenopathy.

Other causes of lymphadenopathy include lymphomas (Hodgkin's and non-Hodgkin's but seldom Burkitt's) and Kaposi's sarcoma.

#### 4.4.2 Parotid enlargement

Enlarged parotids are quite common in older children with HIV infection, usually non-tender and bilateral, and are often associated with rather slower disease progress.

But bilateral parotid enlargement may be found in healthy individuals, and can occur in mumps and some coxsackie virus infections when the parotids are painful and tender. Occasionally mumps [4.7.2] is unilateral, but septic parotitis must be remembered (especially in chronically ill patients). Burkitt's lymphoma [4.17.1] may involve a parotid and benign parotid tumours may also cause unilateral parotid enlargement.

#### 4.4.3 Oral candidiasis

Oral thrush (candidiasis) is very common in children with HIV/AIDS. But remember that neonates, especially preterm may get thrush, and thrush also often follows many antibiotics that kill off competitor bacteria in the mouth. On its own thrush is not an AIDS defining illness, though when combined with severe pneumonia or sepsis in an infant it may strongly suggest a diagnosis of (presumptive) AIDS. Whether or not there is HIV infection, it is usually well managed with topical nystatin or alternatively gentian violet. When candidiasis progresses to involve the oesophagus or trachea, AIDS is suggested. Ketoconazole may then be needed, but remember it cannot be given to patients receiving Triomune.

#### 4.4.4 Lymphoid interstitial pneumonitis (LIP)

This is thought to be caused by the Epstein Barr virus where there is some degree of immunosuppression (stage 2). It usually presents as chronic cough over the age of one, and may be accompanied by lymphadenopathy, parotid enlargement, and clubbing. Try to exclude pulmonary and military TB if possible, and consider other causes of chronic cough such as cardiac failure, whooping cough, asthma and bronchiectasis (which may complicate LIP).

#### 4.4.5 Widespread skin infection

with warts virus or *Molluscum contagiosum* suggests stage 2 immunosuppression in HIV infection, as does any Herpes zoster (shingles) infection in a child or a very severe chickenpox infection.

Certain other infections or diseases define stage 4 AIDS: some are discussed in more detail elsewhere in this book. They include *Pneumocystis carinii* pneumonia (PCP) [4.8.15], Cryptococcal meningitis [4.12.2], and Cytomegalovirus retinitis (with pigmented and white patches in the retina).

### 4.5 Tuberculosis

Tuberculosis (TB) is caused by *Mycobacterium tuberculosis*, a slow growing bacillus with unusual staining characteristics. The organism grows better in oxygen, survives acid and drying (so may persist in dust) but is killed by heat and ultraviolet light. It usually enters the body by inhalation, but may be swallowed. After entry bacteria multiply unhindered in local lymph nodes. Occasionally spread by the blood stream occurs in the weeks before partial immunity (sensitisation) develops. Once partial immunity develops the mantoux test becomes positive. Thereafter spread of mycobacteria within the body is limited, but inflammation occurs around the bacteria, and this may lead to cure or walling off of the infection, or may produce necrotic caseous material (like thick pus, but without local signs of inflammation).

In young children disease usually results from progress of the initial infection, but in older children there may be the common adult pattern of reactivation of a past infection with scarring and cavity formation in the lungs. Most disease occurs in the lungs and related lymph nodes, but cervical and abdominal lymph nodes may be involved. There can be fluid exudates in the pleural, pericardial and peritoneal cavities. The disease can involve the bones (especially the spine), joints (especially the hip and knee), meninges, skin, intestines and kidneys.

When associated with AIDS, depending on the degree of immunosuppression, the pattern of disease may be atypical, and mycobacteria may spread with limited immune response and little caseation.

## Clinical Presentation

A child with tuberculosis may present with general symptoms, specific symptoms, or a precipitating disease (such as malnutrition, measles or HIV) or treatment that has reduced resistance to TB (such as steroids for nephrotic syndrome or cyclophosphamide for Burkitt's lymphoma).

### Making the Diagnosis

In paediatrics the diagnosis of TB is seldom completely certain, because the bacteria are infrequently found in sputum or other specimens. It is important to make the right diagnosis, both because TB is very treatable, and because treatment may be toxic. Try therefore to collect as much evidence for the diagnosis as possible.

#### *Family or contact history:*

Ask repeatedly, and try to prove TB in possible contacts by sputum smears.

#### *General Symptoms*

These may include fever, weight loss, malaise, anorexia, lack of energy, night sweats.

#### *Specific Symptoms*

These depend on the system involved, and may include cough, sputum, chest pain, dyspnoea, swellings (e.g. neck glands), back deformity and/or paraplegia (TB spine), hip pain or limp (TB of hip or knee) headache, confusion and cranial nerve palsies (TB meningitis) and abdominal distension (from ascites) or abdominal masses (lymphadenopathy).

#### *Treatment Failures*

Investigate for TB children with recurrent pneumonia or failure to thrive, who do not respond to adequate treatment.

*Mantoux, Heaf (or PPD) tests.* These are currently not generally available in Malawi unfortunately, but BCG can be used to suggest sensitisation, which will cause a local lump within 72 hours.

Reaction to mantoux or Heaf test in 48-72 hours (> 10 mm if no BCG, > 15 mm after BCG) shows sensitisation, and implies mycobacteria entered the body 6 or more weeks before. In children under 5 this often means active TB. Take care with test technique and dosage, and beware possible false negatives in malnutrition, after measles, with HIV, on steroids and in miliary TB.

#### *Look for organisms*

in sputum in older children, aspirates of pleural, ascitic and pericardial fluid, biopsy, and CSF (though mycobacteria are usually scanty in these).

Needle aspirate from lymph nodes for ZN staining of smears may show TB.

#### *Xrays*

Though seldom diagnostic, Xrays may give some evidence. Suggestive appearances are miliary lung infiltrations, collapsed vertebrae with loss of disc space, hilar adenopathy with lobar collapse, especially in the right upper or middle lobes, and chest X-ray changes that are more dramatic than clinical chest signs.

#### *Response to TB treatment*

Often treatment is itself a test, so try to measure response (weight, fever) to assess if it is effective. However response may be slow. Give a full course of TB treatment even in non-responders to avoid development of drug resistance.

### **Treatment**

Abbreviations used for TB drugs are:

E - ethambutol,                      H - isoniazid,                      R - rifampicin,  
S - streptomycin,                      Z - pyrazinamide.

In all treatment regimens for TB the 3 essential features are REGULARITY of treatment, COMBINED treatment, and PROLONGED treatment. This needs supervision, education and organisation.

#### *Current treatment*

The treatment of TB in adults and children was changed in Malawi in 1997 and again in January, 2007. Currently standard treatment is to give rifampicin and isoniazid for 6 months accompanied by pyrazinamide and ethambutol for the first 2 months all once daily. Previously DOT (Directly Observed Treatment) was given three times a week. Streptomycin is now kept as a reserve drug. Standard drug treatment is now more powerful and used in TB meningitis, but TB meningitis should be treated for 7 months, as previously.

Resistant TB is diagnosed when full TB treatment is completed without default but the disease remains active, especially if it is sputum +ve.

Treatment is with daily SRHZE for 2 months then RHZE for 1 month and 5 months RHE.

#### *Treatment for defaulters:*

Defaulters, by definition did not receive a full treatment course, so are not to be considered as having drug resistant TB. They should either continue (interruption < 2 months) or repeat (interruption > 2 months) the interrupted treatment regimen.

### Summary of TB Treatment Regimens for Children

(as recommended by the National Tuberculosis Control Programme<sup>1</sup>)

<u>Standard Regimen</u>	<u>First 2 months</u>	<u>Later 4 months</u>
All forms, except Meningitis, not drug resistant	RHZE daily	RH daily
Meningitis	RHZE daily	RH daily for 5 months

### Drug Doses & formulations for children with TB & Drug Toxicity

<u>Drug</u>	<u>Daily dose</u>	<u>Thrice weekly dose</u>	<u>Toxicity</u>
E Ethambutol (100 mg tabs)	15 mg/kg	25 mg/kg	optic neuritis red-green colour blindness
H Isoniazid (30 mg in RHZ tablets)	5-10 mg/kg	15 mg/kg	jaundice, rash neuropathy – give pyridoxine 1 mg/kg t.i.d.
R Rifampicin (60 mg in RHZ tablets)	10 mg/kg	10-15 mg	nausea, vomiting jaundice, red urine rapid metabolism of contraceptive pill steroids and nevirapine
S Streptomycin (1 vial = 1 gm)	20 mg/kg	20 mg/kg	ataxia, deafness rash, fever
Z Pyrazinamide (150 mg in RHZ tablets)	30 mg/kg	50 mg/kg	jaundice, gout arthritis

### Dosage for RHZ & E tablets or sachets

<u>Weight</u>	<u>RHZ daily</u> (R60/H30/Z150)	<u>E daily</u> (100 mg)	<u>RHtablets</u> (R60/H30)
< 7kg	1 tablet	1 tablet	1 tablet
8-9 kg	1½ tablets	1½ tabs	1½ tabs
10-14 kg	2 tablets	2 tablets	2 tablets
15-19 kg	3 tablets	3 tablets	3 tablets
20-24 kg	4 tablets	4 tablets	4 tablets
25-29 kg	5 tablets	5 tablets	5 tablets

<sup>1</sup> Manual of National Tuberculosis Control Programme in Malawi, Ministry of Health 6th edition, 2007.



#### 4 Disease Management - TB

<u>Weight</u>	<u>(R150/H75/Z400/E275)</u>	<u>(R150/H75)</u>
30-37 kg	2 tablets	2 tablets
38-54 kg	3 tablets	3 tablets

#### Management of Skin Reactions to TB Drugs

Thiacetazone caused many skin reactions, but is not now used, so streptomycin is the common cause.

Severity	Symptoms	Management
mild (child well)	itching	give antihistamine, observe
moderate (child sick)	fever, rash	stop TB drugs, give antihistamine. When better change regimen.
Severe (child very ill or mucosa involved).	fever, widespread rash, mucosal sores	stop TB drugs, give antihistamine give steroids 2 mg/kg once daily till rash gone, then start one drug at a time $\frac{1}{4}$ dose, then $\frac{1}{2}$ , then full daily.

#### TB and AIDS

TB often complicates AIDS, and treating them together poses problems that have not been entirely solved. Some of the drugs can have similar toxic effects (e.g. nevirapine, isoniazid and rifampicin can all cause jaundice and stavudine and INH can both cause peripheral neuropathy). Then again treating AIDS in a patient on TB treatment can cause "immune reconstitution syndrome" when the patient's immune reaction against TB increases (more inflammation, more caseation). Finally rifampicin increases the rate at which some ARVs are metabolized, notably nevirapine, levels of which in the blood may drop by 30%, causing concern that the virus may more rapidly develop resistance to ARVs.

As our experience increases policy may change, but at present the policy is in patients with TB to wait till the first two months of TB treatment have been completed before starting ARVs. In patients who develop TB while already on ARVs TB treatment should be given in the normal way without stopping ARVs. All patients receiving INH and

stavudine should get pyridoxine to reduce the risk of peripheral neuropathy.

## TB Prevention and Control

### Contact Tracing

Relatives of children diagnosed with TB should have sputum examined, if they have productive coughs. (Do not accept the guardian's or patient's word that the cough is not productive until they have actually tried coughing hard in the mornings and not produced sputum). Treating such sources of TB is very important for preventing further spread. All children with sputum positive adults should be screened for TB. Children under 5 years who are close TB contacts but who are found free of TB should receive INH prophylaxis (5 mg/kg daily for 6 months).

### Immunisation

All newborn babies receive BCG. It is effective in preventing about 60% of TB meningitis and miliary TB. BCG should be avoided in babies with symptomatic HIV infection, i.e. AIDS.

## 4.6 Measles

A very infectious viral illness, transmitted by droplet spread, with an incubation period of 12-14 days. The characteristic rash and mucosal inflammation occur at the time immunity to the virus is developing, and the child is usually non-infectious 48 hours after the rash has developed. The rash is preceded by fever, cough and sometimes diarrhoea. Koplik's spots, fine white dots with a narrow red surround on the inside of the cheeks may be seen at the onset of the rash. The rash appears first behind the ears, spreads to the face and upper trunk, and then to the lower trunk and limbs. It is maculo-papular, and darker than the normal skin. During healing the rash typically desquamates markedly.

Measles may be complicated by secondary bacterial or viral infection of lungs, larynx, and middle ear. Vitamin A metabolism is upset, and corneal damage (xerophthalmia and keratomalacia) may occur. Important late complications are malnutrition and susceptibility to TB.

### Management of measles

\* There is no specific treatment for measles. Patients are often over treated, because features of the viral infection are misinterpreted as

#### 4 Disease Management - Measles

complicating illness. Thus nystatin has no effect on Koplik's spots, and tetracycline eye ointment does not cure the viral conjunctivitis, though it may satisfy relatives and persuade them not to instil herbal medicines in the eyes.

- \* Give vitamin A (100,000 u if < 1 year and 200,000 u if > 1 year) to all cases and repeat the next day and a week later.
- \* The guardian should be taught supportive treatment in milder cases.
- \* Manage fever with paracetamol and extra fluids and diarrhoea with ORS.
- \* Feeding is very important, and, to prevent lactation failing, advise spoon feeding of expressed breast milk if there is stomatitis, and frequent high energy feeds during recovery.
- \* Clean eyes with boiled and cooled water and advise against the use of herbal medicines.
- \* Advise mother how to recognise complications such as dyspnoea, dehydration, ear discharge and convulsions, and then to return to hospital.
- \* Admit child if there are complications or if child is under weight (check the health passport).
- \* Give antibiotics for pneumonia [4.8.14] and otitis media [4.8.7].
- \* Treat laryngitis [4.8.2] with IM dexamethasone 0.3 mg/kg 6 hrly for 24 hours. If not effective intubation and tracheostomy may be needed in an intensive care unit (ICU) setting.
- \* Give IV fluids for severe dehydration.
- \* Give anticonvulsants if there are fits.
- \* Give extra feeding if there is weight loss.
- \* After discharge the child should attend an Under 5 clinic for 3-6 months for growth monitoring.
- \* Give health education on the importance of immunisation.

#### Prevention of Measles

- \* All children who have not suffered documented measles infection should be given measles vaccine at nine months old or as soon as possible thereafter. Take the chance of any health service contact with children to promote immunisation against measles.
- \* All children admitted between 6 and 9 months old to a ward or nutrition centre during a measles outbreak should be given measles vaccine on admission, unless they have already suffered from

documented measles. This applies also to young relatives of admitted children staying with them.

- \* When children with measles are admitted, isolate them if possible, and check that all other children over 6 months old already admitted, or coming for admission thereafter are immunised.
- \* Immunisation of children against measles should only be postponed if the child is acutely and seriously ill (e.g. with depressed consciousness from meningitis or cerebral malaria or severely immunosuppressed by cytotoxic therapy for malignancy) in view of the very real risk to life of measles occurring in an already sick child.

## 4.7 Other Infectious Diseases

### 4.7.1 Chickenpox (Varicella)

This is a highly infectious viral illness spread by droplet infection with an incubation period of 10-20 (commonly 14-15) days. It is usually a mild, self-limiting disease, but can be very serious in immunocompromised children (e.g. AIDS, or on cytotoxic drugs). Patients are infectious from 2 days before the rash appears till all vesicles have crusted. Note that babies born to a mother developing chickenpox from 5 days before deliver to 2 days after is in danger of severe chickenpox.

There is mild fever and scattered, superficial, often slightly oval lesions, mainly on the trunk. Lesions progress rapidly from papules to vesicles (blisters), to pustules, to scabs and are at different stages at one time. Occasionally complications occur, including cerebellar ataxia, encephalitis and viral pneumonia.

The painful condition, herpes zoster, is caused by reactivation of latent varicella virus in patients who previously had chickenpox. In herpes zoster the lesions are like those of chickenpox but more densely clustered, and usually confined to one or a few dermatomes. In immunocompromised patients chickenpox can be extremely severe, even fatal, and herpes zoster often affects multiple dermatomes.

- \* Aciclovir is specific treatment for chickenpox and herpes zoster, but this has to be started at the onset of the rash. As chickenpox is normally a mild illness, only use aciclovir in known immunocompromised patients (e.g. with AIDS, on cytotoxic treatment for malignancy or high dose steroids) within one day of starting itchy rash after known contact with chickenpox or herpes zoster, or starting to

develop herpes zoster. Do not give aciclovir to otherwise healthy children with chickenpox or after the rash of chickenpox or herpes zoster is well developed.

**Do not give aspirin to children with chickenpox or herpes zoster, because of the risk of Reye syndrome.**

- \* Relieve itch with antihistamines e.g. chlorpheniramine (Piriton) or topical calamine lotion.
- \* Keep child's fingernails clean to reduce risk of infecting rash.
- \* Relieve fever and malaise with paracetamol, but avoid aspirin, because of the risk of Reye syndrome.
- \* Immunosuppressed patients should be kept well away from infected children.
- \* Secondary infection of skin lesions may need treatment with flucloxacillin.

#### 4.7.2 Mumps

Mumps is a viral illness that is moderately infectious and spread by droplet infection with an incubation period of 14-28 (usually 16-18) days. The illness usually presents with fever and a painful swelling of both parotid glands, though only one parotid or one or both submandibular and sublingual glands may be involved.

- \* There is no specific treatment for mumps.
- \* Relieve pain of parotitis or orchitis by paracetamol.
- \* The occasional case of pancreatitis or oophoritis may necessitate IV fluids with pethidine when pain is severe.
- \* The occasional case of meningoencephalitis is usually mild and needs only analgesia. Rarely anticonvulsants are needed.

#### 4.7.3 Polio

This is caused by one of three strains of polio virus, with an incubation period (to paralysis) of 2-5 weeks. Infection is by the faecal oral route, and may be asymptomatic, or cause a febrile illness, followed a few days later by a meningoencephalitis with possible fever, headache, neck stiffness, muscle pain and tenderness (and some lymphocytes in the CSF). In a small number of cases progressive flaccid paralysis then

develops over 2-3 days. Paralysis is mainly in proximal limb muscles rather than the muscles of the hands and feet, but is often distributed widely and asymmetrically. Sometimes the muscles of breathing and swallowing are involved. Spontaneous complete recovery is possible, but there is more often residual paralysis, which, if not well managed by physiotherapy and splinting, can lead to marked limb deformities and incapacity.

- \* Analgesics (usually paracetamol) may be given in the acute stage.
- \* Rest the child and avoid IM injections in the acute phase.
- \* After the first two weeks, passive and active physiotherapy can prevent contractures and strengthen unparalysed muscles. Splinting of paralysed limbs in the position of function of joints helps to prevent deformity.
- \* Six months later, once it is clear how much natural recovery will occur, splints and orthopaedic surgery can help mobilise seriously affected patients.
- \* If breathing and swallowing are affected, death is likely to occur. Tracheostomy is the only possible procedure here to keep the airway clear and allow adequate ventilation. Tube feeding and nursing in an intensive care unit at a central hospital are needed.
- \* Prevention of polio is almost completely effective with three doses of oral polio vaccine at monthly intervals (provided the cold chain is effective). Our policy is for an extra "zero" dose at birth so that 4 doses are given. Immunise contacts.
- \* Current attempts to eliminate polio in the world make it important to send stools for viral studies in any child with acute flaccid paralysis or weakness to the EPI Department, MOH, Lilongwe [5.9].

#### 4.7.4 Rubella (German Measles)

Rubella is a viral infection, spread by droplet infection. It usually causes a mild infection, with occipital lymphadenopathy and in fair skinned children a faint pink macular rash. Clinically apparent cases are infectious from about a week before to a week after the rash appears, but babies with congenital Rubella are commonly infectious for many months.

Infection in pregnancy can cause congenital abnormalities (microcephaly, microphthalmos, cataract, deafness, mental defect, persistent ductus arteriosus) or a syndrome of hepato-splenomegaly, anaemia, jaundice and purpura.

- \* There is no specific treatment for rubella.
- \* Give paracetamol for fever and occasional post rubella arthritis.
- \* Keep cases away from women in the child bearing age group.

#### 4.7.5 Diphtheria

An illness caused by strains of *Corynebacterium diphtheriae* that produce diphtheria toxin, with an incubation period of 2-6 days. There are two types:

**Toxic form** - with cardiac failure, poor myocardial contraction, a rapid, weak pulse and low BP.

**Obstructive form** - where a necrotic membrane can spread from the throat to cover and obstruct the larynx. There are swollen, tender cervical lymph nodes and occasionally neurotoxicity (usually paralysis of the soft palate muscles, causing regurgitation of feeds through the nose). The illness is preventable by immunisation with pentavalent vaccine.

- \* The essential treatment is antidiphtheritic antitoxin 20,000 units IV, which must be given as soon as possible (not usually available).
- \* Give penicillin to kill the bacteria (erythromycin if penicillin allergic).
- \* Laryngeal obstruction is an indication for tracheostomy and referral to an intensive care unit setting (24 hour nursing staff, oxygen, suction and facilities for emergency reintubation).

#### 4.7.6 Syphilis

Syphilis is caused by *Treponema pallidum* and may infect about half an infected mother's babies. It may cause death of the unborn foetus (macerated still birth), neonatal disease (including jaundice, anaemia, hepatosplenomegaly and skin peeling of the palms and soles), or it may only show signs later. In infancy it most commonly presents as osteitis (usually multiple and often involving the ends of long bones, resembling arthritis of knees, ankles, elbows or wrists with pain and pseudoparalysis). There is usually failure to thrive and often anaemia, hepatosplenomegaly and cough. A wide-spread rash may appear like eczema, or "snuffles" - discharge and bleeding from both nostrils, or ulcerations near the anus or of the palate. Later in childhood signs can include abnormal teeth (peg-shaped incisors and moon molars), skull bossing, depressed nasal bridge, fine scarring at the corners of the mouth and inflammation of the cornea. The diagnosis is confirmed by a positive VDRL test (which can be done on mother in small babies).

- \* Treat baby and both parents with benzyl penicillin. To the baby give 50,000 u/kg/dose 6 hrly for 10 days (15 days if any signs of nervous system involvement). Because of the danger that the mother may abscond with child before the 10 day course is completed, it is wise also to give an initial dose of 50,000 u/kg of benzathine penicillin stat IM to the child and 1.2 mega to each parent.
- \* Severe osteitis may require POP splints, and severe anaemia may require blood transfusion. Health education to the parents on the dangers of STDs, including AIDS, should always be given.

#### 4.7.7 Tetanus<sup>1</sup>

Tetanus is caused by *Clostridium tetani*, an anaerobic Gram positive bacillus that forms spores which resist boiling. Spores occur widely, and may contaminate wounds, where they germinate into non-invasive bacilli that produce a neurotoxin causing increased tone and muscle spasms. In older children the presentation is usually with stiffness, spasms and trismus (jaw spasm). Diagnosis is clinical and usually easy, but differentials include meningitis and rabies.

- \* First sedate the child with IM or preferably rectal paraldehyde, followed by IM phenobarbitone, and then oral phenobarbitone and diazepam. Regulate the dose by response to treatment. Large doses are often needed but watch for respiratory depression. Chlorpromazine (Largactil) IM can also be used for sedation.
- \* Give antitetanus serum (ATS) 10,000 unit IM stat, if available.
- \* Give benzyl penicillin 50,000 u/kg/dose 6 hrly for 5 days.
- \* Nurse the child in a dark, quiet place to reduce the number of spasms.
- \* When the spasms are severe, IV half strength Darrow's is needed to maintain fluid intake and provide easy access for IV diazepam.
- \* Feed by nasogastric tube if unable to swallow without spasms. It is important to give adequate calories, as the disease may be prolonged and the child may lose much weight.
- \* Watch for and treat complicating pneumonia.
- \* Tetanus does not confer immunity so give tetanus toxoid on admission and repeat after 1 and 6 months.
- \* Educate the guardian about the cause of tetanus and the value of pentavalent vaccine and of tetanus toxoid in pregnancy to prevent neonatal tetanus.

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<sup>1</sup> See section 2.4.7 for management of neonatal tetanus.



#### 4.7.8 Typhoid

A bacterial illness caused by *Salmonella typhi*, a Gram negative bacillus with an incubation period of 10-14 days (but as wide a range as 4-23 days has been reported). Infection is usually caught by swallowing contaminated food or water. It is now uncommon in Malawi.

Onset is often gradual with a sustained and increasing fever, and other symptoms such as cough, slight diarrhoea (especially children) or constipation. Later there is wasting, mental confusion, listlessness, abdominal pain and guarding in the right hypochondrium and a relative bradycardia. Complications include perforation of the terminal ileum, haemorrhage from ileal ulcers and local abscesses.

- \* Obtain, if possible, blood, stool and urine cultures prior to starting treatment to confirm the diagnosis. The Widal test is almost useless. The WBC is characteristically normal to low.
- \* Give chloramphenicol (25 mg/kg/dose 6 hrly) for 14-21 days. Initially give IV (IM if IV impossible) and then orally when any ileus has resolved.
- \* Alternative drugs are ciprofloxacin (10-15 mg/kg/dose b.d.), and ceftriaxone. Where these are not available, IV ampicillin<sup>1</sup> (50 mg/kg/dose 8 hrly), oral amoxicillin (30 mg/kg/dose 8 hrly), and cotrimoxazole (30 mg/kg/dose orally 12 hrly) can be tried.
- \* Treat ileal perforation by surgical closure, peritoneal lavage, drip and suction, and IV chloramphenicol. IV metronidazole, if available, is given also to combat anaerobes released into the peritoneal cavity.
- \* Intestinal haemorrhage (a less common complication than perforation) requires blood transfusion.
- \* Careful IV fluid therapy, good nursing care (to prevent pressure sores), mouth cleaning (to prevent septic parotitis) and extra feeding during recovery are important.
- \* In patients with co-existing schistosomiasis, cure of typhoid may only occur after the schistosomiasis has been treated with praziquantel.

#### 4.7.9 Whooping Cough (Pertussis)

This is caused by a bacterium, *Bordetella pertussis*, with an incubation period of 7-10 days. At first it is indistinguishable from a mild coryza [4.8.1], but an increasingly persistent cough develops in bouts, which is

<sup>1</sup> IV Ampicillin is necessary if oral absorption is uncertain, but is less effective than amoxicillin, when used orally.

followed by the typical "whoop" and often vomiting of food and thick mucus. Cough may continue for up to 3 months, is typically worse at night, and can lead to wasting from vomiting. Secondary complicating infections, especially pneumonia may occur. It is particularly serious in small infants, many of whom have apnoeic attacks rather than whooping. It does not generally respond to antibiotics, partly because the bacteria do not invade the tissues, but live in very thick mucus in the bronchi and nasopharynx, into which antibiotics penetrate with difficulty, and partly because symptoms are due to toxin damage to the mucosa of the bronchi that persists long after the infecting bacteria have disappeared.

- \* Antibiotics (erythromycin preferably, or chloramphenicol) can abort attacks of whooping cough<sup>1</sup> if given VERY early (during the coryza phase, before the "whooping" cough is apparent).
- \* Treat secondary pneumonia [4.8.14] or acute otitis media [4.8.7] with antibiotics.
- \* Maintain nutrition, compromised by vomiting and exhaustion after bouts of coughing (refeed babies after vomiting).
- \* Cough mixtures are useless. Frequent and severe coughing spells can be helped by phenobarbitone 5 mg/kg/dose orally 12 hrly, although this is controversial, as it sedates.
- \* Whooping cough is preventable by pentavalent vaccine.

#### 4.7.10 Trypanosomiasis

In Malawi trypanosomiasis (sleeping sickness) is caused by *Trypanosoma rhodesiense*, a flagellate protozoan spread from wild game to man by the bite of the tsetse fly. Recently cases have occurred in Rumphi, Kasungu and Nkhosakota Districts, so think of this diagnosis in patients from there. A painful hard nodule occurs at the bite site after 2-7 days. This heals completely but slowly after about 2 weeks. By 3 weeks after the bite, trypanosomes appear in the blood, causing an illness characterised by irregular fever, headache, rash, lymphadenopathy (especially cervical) and peripheral oedema. The illness may further develop by invasion of the nervous system, with changed behaviour, headache, neck stiffness, day time sleepiness, tremors, paralysis, depression of mental functions, and carditis with cardiac failure. The diagnosis is confirmed by finding trypanosomes in the blood film, CSF or lymph node aspirate.

<sup>1</sup> Antibiotics are NOT justified in the treatment of ESTABLISHED whooping cough. In practice it is the patient's unimmunised or partly immunised sibling who should be treated.

#### 4 Disease Management - Respiratory Diseases - Coryza, Croup

- \* Before nervous system invasion occurs, treat with suramin. Give a test dose of 10 mg IV first in case of rarely seen hypersensitivity shock. Then, if there is no adverse reaction, give 5 doses of 20 mg/kg IV at intervals of 5-7 days. Before each dose check for proteinuria which indicates renal toxicity. Follow up the patient to check the CSF.
- \* Melarsoprol is needed to cure nervous system infection. After 2 to 3 preliminary doses of suramin, give melarsoprol, in a dose of 3.6 mg/kg by slow IV injection. Give a series of 3 daily injections at weekly intervals for a total of 3 courses i.e. total of 9 injections. Melarsoprol may have to be obtained specially, because not normally stocked now in Malawi. It is also effective against trypanosomes in the blood.
- \* Encephalopathy is the most serious toxic effect of melarsoprol and should be treated with dimercaprol (BAL). It has been shown that there is less risk of encephalopathy if prednisolone is given at a dose of 1 mg/kg/dose once daily for the first 2 weeks of melarsoprol treatment and then tailed off over the next 1-2 weeks.
- \* Melarsoprol may also cause exfoliative dermatitis, and renal and liver damage.
- \* Confirm cure by follow up clinical examination, blood and CSF tests.

### 4.8 Respiratory Diseases

#### THE UPPER RESPIRATORY TRACT

##### 4.8.1 Coryza (URTI)

- \* No antibiotics are needed for this viral infection.
- \* Small babies should have their noses cleaned with cotton wool to relieve obstruction that interferes with breathing and feeding.
- \* Paracetamol can be given for fever over 38.5°C or for headache in older children.
- \* If there is fever, also treat malaria [4.1].
- \* Treat any complicating acute otitis media [4.8.7].

##### 4.8.2 Croup

Croup is usually acute viral laryngotracheobronchitis. Measles may cause an acute laryngitis which presents similarly with hoarseness and a barking cough. The differential diagnosis includes other causes of stridor (i.e. obstruction to air flow through the upper airway) such as: acute epiglottitis, diphtheria, infectious mononucleosis, inhaled foreign body

and congenital causes such as laryngomalacia.

\* Assess the severity of croup (see table below):

<u>Sign/Symptom</u>	<u>Mild</u>	<u>Moderate</u>	<u>Severe</u>
Stridor	when aroused	heard at rest	marked/decreased
Retractions	absent/slight	moderate to severe	severe
Air entry	normal	normal	decreased
Pulse	increased	tachycardia (> 160)	tachycardia
Resp.rate	normal	raised	60/min or reduced
Restlessness	absent	absent	present/irritable
Cyanosis	absent	absent	present

\* Observe MILD AND MODERATE CASES for deterioration.

\* FOR SEVERE CASES

- Give oxygen.
- Maintain hydration with IV fluids if needed.
- Treat laryngeal inflammation with dexamethasone 0.3 mg/kg/dose IM or IV 6 hrly for 24 hours (longer administration or higher dosage is not recommended). Intubation or tracheostomy may be needed, preferably in an ICU setting.
- Adrenaline 1.0 ml of a 1/1000 solution diluted with 1.0 ml sterile saline delivered by nebuliser through a face mask at frequent intervals (half hourly) can help temporarily, if transfer to an ICU setting is not possible.
- Antibiotics are ONLY needed if secondary bacterial infection is suspected, in which case give benzyl penicillin and chloramphenicol.

#### 4.8.3 Acute Epiglottitis

This is a bacterial infection, caused by Haemophilus influenzae, which presents with a rapid onset of severe stridor, dyspnoea, toxicity and drooling saliva..

- \* Give preferably ceftriaxone, or chloramphenicol (initially IV or IM, later orally or ampicillin initially (IV or IM), and then oral amoxicillin.
- \* Airways obstruction is frequently severe, and intubation or

**If you suspect acute epiglottitis, only attempt to inspect the epiglottis when all preparations are made for intubation of the child, with oxygen available, as otherwise spasm of the epiglottis may cause death.**

tracheostomy is often needed. However inspection of the epiglottis or larynx can cause laryngeal spasm and DEATH. So before trying to inspect or intubate, carefully prepare in theatre with oxygen, a functioning laryngoscope, suction and a range of endotracheal tubes.

- \* The management is otherwise similar to that for croup [4.8.2].
- \* Pentavalent vaccine should now prevent most cases.

#### 4.8.4 Acute Sinusitis

This is a bacterial infection which complicates coryza in older children. It is usually caused by *Streptococcus pneumoniae* and gives fever and local pain (headache).

- \* Treat with amoxicillin or cotrimoxazole for 5-7 days.
- \* For relief of pain and fever, give paracetamol.

#### 4.8.5 Acute Tonsillitis

Caused by beta haemolytic group A *Streptococcus pyogenes*, it presents with fever, sore throat and usually tender tonsillar glands at the angle of the jaw. In small children abdominal pain may occur. Complications include peritonsillar and tonsillar gland abscesses, rheumatic fever [4.9.2] and acute glomerulonephritis [4.11.2]. The differential diagnosis includes diphtheria and infectious mononucleosis.

- \* Give amoxicillin for a full 10 days to eliminate *Streptococcus pyogenes* and prevent complicating rheumatic fever.
- \* If compliance with this rather long course of treatment seems unlikely, give benzathine penicillin IM stat.
- \* Give erythromycin for 10 days if allergic to penicillin, or preferably azithromycin for 3 days (better compliance).
- \* Note that if a marked skin rash appears on amoxicillin it is likely that the tonsillitis was from infectious mononucleosis, caused by the Epstein Barr (EB) virus, and not necessarily allergy to amoxicillin.
- \* Give paracetamol for initial pain and fever.

#### 4.8.6 Foreign Body Inhalation<sup>1</sup>

There is often a history of sudden choking and subsequent dyspnoea in a previously well small child. Acute epiglottitis is the chief differential diagnosis.

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<sup>1</sup> See also sections 3.2.1 and 5.3.1 for Xray appearances of foreign body inhalation.

- \* Occasionally a large foreign body in the larynx can be removed in an older child immediately by Heimlich's manoeuvre (forceful squeezing of the chest by the clinician standing behind the child with a clenched fist grasped by the other hand pressing immediately below the child's sternum) or by laying the smaller child prone with head tilted down and striking the back of the lower chest with the heel of the hand up to five times.
- \* Otherwise refer urgently for removal by bronchoscopy. If the foreign body is vegetable (e.g. groundnut), it is likely to disintegrate and be inhaled further into the small bronchi causing lung collapse, secondary infection, lung abscess or later bronchiectasis.
- \* Following bronchoscopy, give vigorous chest physiotherapy and antibiotics such as benzyl penicillin, cotrimoxazole or chloramphenicol.

#### 4.8.7 Acute Otitis Media

An acute, usually bacterial infection of the middle ear, which may follow coryza, measles or whooping cough. There is usually pain, fever and deafness. Diarrhoea, vomiting and febrile convulsions may occur.

- \* Give cotrimoxazole for 10 days. Amoxicillin is an alternative.
- \* Relieve pain and fever with paracetamol.

#### 4.8.8 Chronic Suppurative Otitis Media

A chronic painless discharge from the ear, complicating acute otitis media. It is seen if the acute infection is not well treated early. Secondary bacterial invaders enter the ear, making cure difficult.

- \* Gently dry mop with cotton wool<sup>1</sup> until the cotton comes out clean from the ear. Repeat four times a day.
- \* Give antibiotics only when the guardian has been taught to dry mop competently (antibiotics are not effective without the use of dry mopping), and preferably after bacterial culture and sensitivities of the pus have been obtained<sup>2</sup>. Blind guessing of appropriate antibiotics often gives disappointing results. It is occasionally necessary to use gentamicin.

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1 A small twisted piece of cotton wool is inserted as a "wick" to absorb discharge at frequent intervals.

2 A mixture of secondary invaders such as pseudomonas, klebsiella, E. coli, streptococci or staphylococci is often found in the pus.

- \* Check that the child has been immunised against tetanus, and give a booster dose of tetanus toxoid 0.5 ml IM if necessary.

#### 4.8.9 Retropharyngeal Abscess

Usually it occurs in infants or small children, and is secondary to infection in the oro- or nasopharynx which spreads to lymph nodes in the retro-pharyngeal space. The abscess develops gradually, and presents with stridor from partial airway obstruction, drooling of saliva because of inability to swallow, neck retraction and tender tonsillar nodes with fever. Bulging of the back of the throat may be seen on gentle inspection or felt on digital palpation of the throat but do not examine if epiglottitis is suspected. A lateral Xray of the soft tissues of the neck will show an increased "space" between the pharynx and cervical vertebrae.

- \* Treatment is surgery with good anaesthesia and endotracheal intubation. Intubation is best continued for 1-2 days after surgery in an ICU.
- \* Give flucloxacillin, chloramphenicol and metronidazole to cover staphylococci, streptococci and anaerobes.

### THE LOWER RESPIRATORY TRACT

#### 4.8.10 Asthma

Asthma is a disease of recurrent but reversible bronchospasm and bronchial obstruction. It commonly runs in families, and may be associated with eczema. Children often present with recurrent cough, which is typically worse at night, or in cool cloudy weather. Wheeze may not have been noted. Difficulty in breathing with coughing may be seen. Frequently the child will have been given repeated courses of antibiotics for an incorrect diagnosis of pneumonia or "wheezy bronchitis." Bronchial obstruction is due to spasm of smooth muscle, increased sticky bronchial secretions and mucosal oedema. (Bronchial muscle develops at about a year old).

Treatment involves drugs that reduce the muscle spasm, either by acting directly on the muscle (e.g. aminophylline) or by counteracting the chemicals causing the bronchoconstriction. These drugs have limited effectiveness under 1 year old.

### SEVERE ASTHMA

If the child is too breathless to talk, cry or feed, or is cyanosed or has

poor respiratory effort, exhaustion, a silent chest, or low coma score, treat for very severe asthma. If possible admit to a special care area and monitor oxygen saturation with a pulse oximeter.

- \* Give oxygen at 1-2 litres/minute.
- \* Give salbutamol by nebuliser, 2.5 mg if age < 3, 5 mg if age > 3.
- \* Insert an IV line with 5% dextrose and give hydrocortisone 4 mg/kg IV stat (or dexamethasone 0.3 mg/kg IM).
- \* 30 minutes after the nebuliser, if no improvement, repeat nebulised salbutamol.
- \* If necessary after a further 30 minutes repeat this again.
- \* If still no improvement, give aminophylline 5 mg/kg<sup>1</sup> (maximum 300 mg) slowly IV over 20 minutes. (Too fast can lead to vomiting, headaches, convulsions and cardiac arrhythmias).
- \* If needed repeat the aminophylline 5 mg/kg slowly IV 6 hourly, or by slow IV infusion to give this dosage.
- \* Repeat the hydrocortisone 6 hourly if the child still cannot swallow.
- \* Give prednisolone 2 mg/kg orally once daily for 3 days when able to swallow.
- \* Start on antibiotics if there is good evidence of chest infection (fever without malaria, bronchial breathing, consolidation on chest Xray).

### SEVERE ASTHMA

If a child has dyspnoea (chest indrawing, use of accessory muscles in breathing, grunting or audible wheeze) but no signs of very severe asthma as above, treat for severe asthma.

- \* Give salbutamol by nebuliser as above half hourly or by metered dose inhaler with spacer (5 puffs into spacer and allow child to take 20 normal breaths through the spacer).
- \* If salbutamol is not available, give aminophylline 5 mg/kg<sup>1</sup> IV over 20 minutes.
- \* Give prednisolone 2 mg/kg orally daily for 3 days.
- \* Reassess 30 minutes after starting treatment, and if dyspnoea persists, give oxygen 1-2 litres/minute.
- \* Repeat salbutamol hourly if necessary..
- \* If dyspnoea improves, continue salbutamol regularly by metered dose inhaler 5 puffs hourly or orally 0.1 mg/kg/dose t.i.d. for 3 to 5 days.

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<sup>1</sup> If the child has been on oral aminophylline or taking erythromycin, do not exceed 5 mg/kg/dose.



### MILD ASTHMA

If a child is wheezing without dyspnoea, and has a past history of wheezy episodes, treat with an oral bronchodilator, preferably salbutamol, or, if that is not available, aminophylline. Ask about precipitating causes (coryza, smoke, allergy to pollen or dust, allergy to food or drugs) and when possible avoid these. Many asthmatics wheeze with exercise and should be advised to take a bronchodilator before expected exercise. Persistent wheeze is best managed by a "preventer" inhaled steroid, such as beclomethasone (Becotide), if available, once or twice daily. The use of inhalers for salbutamol or steroid requires careful training of the guardian and child to be effective.

#### 4.8.11 Acute Bronchiolitis

This viral illness presents in young infants (3-6 months) with wheeze, dyspnoea, over expansion of the chest, often with palpable liver and spleen, and bilateral crepitations in the lungs. Often outbreaks occur in the wet season, and several infants may be admitted at one time. Usually the cause is Respiratory Syncytial Virus (RSV), which is very infectious and transmitted on the hands of attendants as well as by droplets. Hand washing is therefore most important in preventing spread within the ward. Antibiotics are not effective.

- \* Where there is cyanosis, restlessness or marked dyspnoea, give oxygen at 0.5-1 litres/minute.
- \* Maintain hydration with IV fluids if needed, but avoid over hydration.
- \* Antibiotics are often given because of difficulty ruling out pneumonia as an alternative diagnosis, and cotrimoxazole is probably first choice [4.8.14].
- \* Keep suspected cases away from infants under 6 months old with other diseases.

#### 4.8.12 Acute Bronchitis

Usually a viral infection, presenting with cough, crackles, and sometimes wheeze. Antibiotics are not effective and beware of diagnosing asthma [4.8.10] as "bronchitis".

- \* If fever > 38.5°C (usually mild), give paracetamol and enough fluids.
- \* Where secondary bacterial infection is suspected, give cotrimoxazole for 5 days. An alternative antibiotic is amoxicillin for 5 days.

#### 4.8.13 Bronchiectasis

This is a chronic dilatation of the bronchi in part or all of a lobe of lung, caused by suppurative damage to the bronchial wall. Frequently it follows a period of lung collapse and infection e.g. after whooping cough, LIP [4.4.4] or an inhaled foreign body. The characteristic features are of chronic productive cough, with recurrent episodes of fever and discoloured sputum. Often there is finger clubbing.

- \* The main treatment is regular postural drainage and chest physiotherapy to evacuate sputum from the chest. This must be taught competently to the parents of the child, and needs to be performed 3 to 5 times a day.
- \* If sputum is discoloured (yellow, green), contains blood, or the child has tachypnoea, dyspnoea, chest pain or fever, treat secondary infection with antibiotics. Choice of antibiotics depends on which one has been recently been used. Ten day courses are often needed, and alternatives include cotrimoxazole, amoxicillin, chloramphenicol and erythromycin.
- \* Rarely, if the bronchiectasis is severe and proven by bronchography to be localised to one lobe or lung, surgical removal of the affected lobe or lobes may be recommended.

#### 4.8.14 Pneumonia<sup>1</sup>

Pneumonia is an infection of the lung tissue. The causes are many, including viruses, bacteria, mycoplasma and *Pneumocystis carinii*. The most common bacterial causes are *Streptococcus pneumoniae* (pneumococcus) and *Haemophilus influenzae*, less often staphylococci and klebsiellae. As well as cough and fever there may be dyspnoea and lateralised chest pain. Herpes simplex on the lip is common in lobar pneumonia. The pattern of inflammation may be lobar (usually caused by *S. pneumoniae*, but also by klebsiellae or *M. tuberculosis*), patchy or perihilar. Complications of pneumonia include pleural effusion, empyema, and less commonly pericarditis, pneumothorax, pneumomediastinum and subcutaneous emphysema. [See also 3.2.1]

The most sensitive respiratory signs are rapid breathing, and dyspnoea (nasal flaring, subcostal recession, intercostal indrawing, grunting). Rapid breathing is defined as a rate of > 60/min if under 2 months old, > 50/min if 2 months-1 year old, > 40/min if 1-4 years old. More specific

<sup>1</sup> For the management of neonatal pneumonia see sections 2.4.3 and 2.4.7.

signs, like reduced breath sounds, bronchial breathing, and fine crepitations, are difficult to detect and not always present in children.

#### MANAGEMENT OF PNEUMONIA IN CHILDREN OVER 2 MONTHS OLD

Management depends on classification of pneumonia as pneumonia, severe or very severe pneumonia.

- \* *For pneumonia* (tachypnoea but no dyspnoea) treat as an outpatient with cotrimoxazole 18-30 mg/kg/dose of combined drug (3-5 mg/kg/dose of trimethoprim) b.d. or amoxicillin 15 mg/kg/dose 8 hourly for 5 days. Advise return if not improving in 48 hours.
- \* Treat wheeze if present.
- \* *For severe pneumonia* (tachypnoea and dyspnoea, but no danger signs) admit to hospital and give benzyl penicillin 50,000 units/kg/dose q.i.d. for 5 days and perhaps oxygen. Change to chloramphenicol if not improving after 36-48 hours. These children may also need antimalarials, treatment of wheeze. Keep up fluid and food intake.
- \* When children with severe pneumonia improve and can take drugs orally, switch to oral antibiotics like amoxicillin or cotrimoxazole.
- \* *For very severe pneumonia* (dyspnoea, and danger signs - cyanosis, depressed consciousness, inability to drink, convulsions or malnutrition) give chloramphenicol 25 mg/kg/dose q.i.d. or (for staphylococci), flucloxacillin 25 mg/kg/dose q.i.d. for 7 days. Give oxygen and check pulse oximetry. Exclude other severe disease like meningitis and treat malaria and wheeze if needed.
- \* If children respond poorly suspect other disease like HIV infection, heart failure, bronchospasm, foreign body, TB, *Pneumocystis carinii* or staphylococcal pneumonia. (Xray may show pneumatoceles, suggesting staphylococcal pneumonia, or empyema [5.3.1]).
- \* *Mycoplasma pneumonia* occurs in older children, has a less clear presentation often with few signs, and responds best to azithromycin or erythromycin.
- \* In children thought to have HIV, who do not respond to standard treatment, suspect *Pneumocystis carinii* infection and give cotrimoxazole in high dosage of 60 mg/kg/dose b.d. (or equivalent divided into 3-4 doses a day) for 3 weeks. If at all possible such a child (or the mother if the child is under 18 months) should have an HIV test, because, if the test is negative, the diagnosis should not be made. Also investigate for TB (see Section 4.4). Continue lifelong

cotrimoxazole prophylaxis (30 mg/kg/dose of combined drug or 5 mg/kg/dose of trimethoprim daily) to prevent reinfection.

#### MANAGEMENT OF PNEUMONIA IN CHILDREN UNDER 2 MONTHS OLD

Pneumonia in small infants is very dangerous, and requires admission, whether or not tachypnoea (respirations > 60/minute) is accompanied by dyspnoea. Otherwise management is similar to pneumonia in older children except that the appropriate antibiotics are benzyl penicillin<sup>1</sup> combined with gentamicin (in the first week 5 mg/kg/dose daily for 5 days but 7.5 mg/kg/dose daily later) [see Section 2.4.7].

#### 4.8.15 *Pneumocystis carinii* (now renamed *Pneumocystis jirovecii*) pneumonia (PCP)

Pneumonia caused by this (probably fungal) organism is seen in immunosuppressed patients, mostly infants with AIDS (it is an AIDS defining illness). It tends to present with cough, tachypnoea, dyspnoea and hypoxia (cyanosis or low pulse oximeter reading) with relatively few chest signs. Chest Xray may show a varied picture but often interstitial perihilar infiltrates.

Diagnosis in Malawi is clinical, where there is a clear history of HIV exposure, with acute bronchiolitis and bacterial pneumonia the most important differentials.

Treatment is high dosage cotrimoxazole (30-40 mg/kg/dose of combined drug or 5-7 mg/kg/dose of trimethoprim 3 or 4 times daily, aiming at 120 mg/kg of combined drug or 20 mg/kg trimethoprim daily) for 3 weeks. Continue lifelong cotrimoxazole prophylaxis with 30 mg/kg/dose of combined drug or 5 mg/kg/dose of trimethoprim daily. Give oxygen. Patients still dyspnoeic and hypoxic on treatment should be considered for oral prednisolone 2 mg/kg/dose daily for at least 2 weeks, thereafter tapered off over a week. The prognosis, however, is poor

#### 4.8.16 Pleural Effusion and Empyema

These can be complications of bacterial pneumonia and TB and may cause dyspnoea and persistent fever, reduced chest movement, very dull percussion note, reduced breath sounds and sometimes bronchial breathing. Confirmation can be made by chest Xray [5.3.1] or ultrasound.

<sup>1</sup> In the first week of life benzyl penicillin 50,000 units/kg/dose can be given 12 hourly, thereafter 6 hourly.

#### 4 Disease Management - Cardiovascular Disease - Heart Failure

- \* Pleural effusions require aspiration [5.4.11], sometimes repeatedly, and treatment of underlying pneumonia or TB.
- \* Empyema needs urgent insertion of a wide pleural tube for under-water-seal drainage. [5.4.12]
- \* In addition antibiotics are needed according to culture results of the pus. Often chloramphenicol or flucloxacillin will be needed for 10 days, but TB can be the cause and will need TB treatment [4.5].

### 4.9 Cardiovascular Diseases

#### 4.9.1 Cardiac Failure

Cardiac failure is failure of the heart to pump enough blood for the needs of the body. The causes are multiple, including

- a need for increased cardiac output (e.g. in anaemia)
- increased work of the heart (e.g. in hypertension)
- damage to the valves of the heart (rheumatic heart disease)
- abnormal arrangements of the heart pumps and their communications (various congenital heart diseases)
- damage to the heart muscle (e.g. in viral, including HIV, and rheumatic myocarditis)
- restriction of heart movement (e.g. pericardial effusion, from TB or Kaposi's sarcoma, and constrictive pericarditis in TB)
- fluid overload (e.g. renal failure, glomerulonephritis, excess hydration, especially if there is malnutrition)

Effects of heart failure can be predominantly poor forward pumping of blood (weak pulse, low blood pressure) or accumulation of fluid behind the pump (e.g. pulmonary oedema, engorged neck veins and liver, oedema of the legs). The most specific sign of heart failure in a child is triple rhythm, but more often seen are tachycardia, dyspnoea and hepatomegaly. Digoxin and diuretics are the main drugs used to treat heart failure from all causes.

#### DIGOXIN

This is potentially a lethal drug. Caution is needed for preterm babies and children with kwashiorkor or myocarditis who are particularly sensitive to digoxin. It is not effective if heart failure is caused by pericardial effusion. Calculate doses very carefully. Nurses should check the dose before giving it, and check with the prescriber if in doubt.

- \* To digitalise the patient, give a dose of 0.005 mg (5 micrograms) /kg/dose 8 hrly for 3 doses.
- \* For maintenance treatment give 0.010 mg (10 micrograms)/kg daily.
- \* In children on digoxin watch for toxic signs: loss of appetite, nausea, vomiting, slow heart rate, extrasystoles, especially coupled beats, yellow vision. Be particularly careful if blood potassium may be low as in malnutrition or children on high dose frusemide.

#### DIURETICS

- \* The most commonly used diuretic is frusemide, given IV, IM or orally (1-2 mg/kg/dose once or twice daily). Use IV frusemide in acute pulmonary oedema. It is a powerful short-acting diuretic, so avoid giving just before sleep. Long term use causes potassium deficiency, and potassium supplements (slow K tablets, bananas) are needed.
- \* Alternatively for maintenance give daily oral hydrochlorothiazide 25 mg (smaller children) or 50 mg (larger children).
- \* In resistant cases add spironolactone in a dose of 0.5-1 mg/kg/dose 8 hrly to frusemide. Spironolactone also reduces potassium loss.
- \* An alternative drug now sometimes available is an ACE inhibitor such as captopril, but this should only be started in hospital as it may interact with diuretics, dropping blood pressure.
- \* In severe cases ensure bed rest, with the patient propped up on pillows, oxygen at 2 litres/minute intranasally and salt restriction.
- \* Treat any causal or intercurrent illness, especially anaemia.

#### 4.9.2 Rheumatic Fever

This is caused by an immune type inflammatory reaction resulting from beta haemolytic streptococcal infection of the throat. The joints, brain, subcutaneous tissue, and all three layers of the heart (endocardium, myocardium and pericardium) may be affected.

The diagnosis is a clinical one, and depends on finding 2 major, or 1 major and 2 minor, "Jones" criteria.

##### *Major Jones criteria*

Carditis, flitting transient arthritis, chorea, subcutaneous rheumatic nodules and erythema marginatum (last two rare).

##### *Minor Jones criteria*

Fever, arthralgia (in the absence of arthritis), prolonged PR interval on ECG (in the absence of carditis), evidence of a streptococcal infection e.g.

by throat culture or ASO titre, and a past history of rheumatic fever.

- \* Give penicillin as for tonsillitis [4.8.5] and thereafter benzathine penicillin<sup>1</sup> IM regularly monthly.
- \* Penicillin prophylaxis should be continued until at least the age of 20 in all children who have had rheumatic fever, but should be life-long for children who have had any evidence of carditis or heart valve damage.
- \* Bed rest is advisable while the patient is febrile, has active arthritis or carditis.
- \* High dose aspirin (20-25 mg/kg/dose 6 hrly orally) is very effective for arthritis and may control carditis.
- \* Cardiac failure may require digoxin (given with caution) and frusemide [4.9.1]. In heart failure check heart and breathing rates regularly.
- \* Prednisolone 2 mg/kg/dose orally once daily can control carditis if cardiac failure persists, but exclude TB first, as latent TB is common among Malawian children and may become active during steroid treatment. If TB is diagnosed give full TB treatment with the prednisolone. Steroid treatment is likely to be needed for a month, before being gradually tailed off.
- \* Treat chorea with oral haloperidol 25-50 micrograms/kg daily, or phenobarbitone.
- \* Long term follow up after rheumatic fever is needed for prevention of recurrences and management of complicating chronic valve damage (usually mitral incompetence and stenosis, also aortic incompetence). These may appear some time after acute rheumatic fever.
- \* Always carefully document the evidence for rheumatic fever in the health passport, since secondary prevention must be continued so long, and fortunate patients have no residual signs but are still liable to recurrent acute rheumatic fever.

### Prophylaxis against infective endocarditis

If valve damage is present, then secondary infective endocarditis is a risk [4.9.4].

- \* Educate parents to care for the child's teeth, by not giving foods high in sucrose (sugar, sweets, soft drinks, biscuits), by good dental care

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<sup>1</sup> Alternatives to benzathine penicillin are amoxicillin 125-250 mg 12 hrly or, if there is penicillin allergy, use erythromycin. However problems of poor compliance are more likely with these oral regimens.

hygiene, and by informing a dentist who has to fill or extract a tooth that the child has heart disease, so that antibiotic cover can be given.

- \* Antibiotic cover should be started 1 hour before any dental surgery, and should be repeated 6 hours afterwards. Give benzyl penicillin (25,000 u/kg IM) before and 6 hours after the procedure. In a child on maintenance low dose penicillin there is a risk that organisms resistant to benzyl penicillin may be present so give ampicillin 25 mg/kg/dose IM or amoxicillin 50 mg/kg/dose orally 1 hour before and 6 hours after, combined with gentamicin 7.5 mg/kg IM before.

#### 4.9.3 Pericardial effusion

Severe pericardial effusion may cause "cardiac tamponade" by compressing the heart and preventing it pumping blood well. There are signs of cardiac failure, especially tachycardia and hepatomegaly with raised jugular venous pressure. The pulse is weak, systolic blood pressure low and heart sounds faint. Lesser effusions may cause chest pain. The effusion may be caused by TB, malignancy (especially Kaposi's sarcoma) or occasionally be a complication of pneumonia or trauma.

- \* Chest Xray shows a large globular heart but the best investigation is cardiac ultrasound.
- \* tap the effusion [5.4.13] for laboratory investigation and relief of tamponade.

#### 4.9.4 Infective Endocarditis<sup>1</sup>

This is a bacterial infection of a heart valve (endocardium). Causative organisms are usually only mildly virulent. Infection is secondary to rheumatic or congenital disease of the heart. Any septic lesion allowing entry of bacteria to the blood may lead to infection, but the most common source is the teeth, especially if there is caries or gingivitis.

- \* The prevention of infective endocarditis in patients with either congenital or rheumatic heart disease is important [4.9.2].
- \* The diagnosis of active infective endocarditis should whenever possible be made by repeated blood culture, which will then give a guide to appropriate antibiotic treatment.
- \* If blood culture is not possible, benzyl penicillin 4 hrly with gentamicin daily for 4 weeks will often be effective. Alternatively ampicillin IM 4 hrly and gentamicin daily for 4 weeks may be given.

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<sup>1</sup> Also known as subacute bacterial endocarditis (SBE).



#### 4 Disease Management - Cardiovascular Disease - Hypertension

- \* Treat cardiac failure [4.9.1]. Correct anaemia by transfusion if severe. Milder anaemia will only be corrected by control of the infection.
- \* Heart surgery may be needed to correct primary congenital abnormality or to replace severely damaged valves after the infection has been cured.

#### 4.9.5 Hypertension

Hypertension occurs most commonly secondary to post-streptococcal glomerulonephritis, but also secondary to other renal disease, coarctation of the aorta, steroid treatment, Wilm's tumour and other rare causes. All children with suspected renal disease should have their BPs taken. When there is hypertension, fluid retention and heart failure is likely to develop, and will not improve without treatment of the hypertension. To obtain the correct BP it is necessary for the BP cuff to cover 2/3 of the length of the arm above the elbow. Too narrow a cuff will give a falsely high reading.

The following table is a guide of when to treat raised BP

#### ***Blood pressure values***

Age	Acceptable values (mmHg)		Dangerous values (mmHg)	
	systolic	diastolic	systolic	diastolic
< 1 yr	70-105	40-70	110	75
1-5 yrs	80-110	50-75	115	80
6-8 yrs	85-115	50-75	120	85
9-12 yrs	90-125	55-80	130	90
12-14 yrs	100-135	60-85	140	95

#### ***Paediatric Dosages of Antihypertensive Drugs***

DRUG	PRESENTATION	DOSE
Hydrochlorothiazide	tablets 50 mg	25-50 mg once daily
Methyldopa	tablets 250 mg	5 mg/kg/dose increasing to 10 mg/kg/dose 6-12 hrly
Captopril	12.5 mg tablets	test dose of 0.1 mg/kg then 0.1 to 0.3 mg/kg/dose two or three times daily
Propranolol	tablets 40 mg	0.25-1 mg/kg/dose 6 hrly Only use if there is no heart failure.

#### 4.9.6 Congenital Heart Disease

There are very many types of congenital heart disease but only a few types are reasonably common. The main presentations are:

- Cardiac failure, with possibly a murmur on examination.
- Failure to thrive and a heart murmur is found.
- A heart murmur may be found during an unrelated illness.
- More rarely babies are cyanosed<sup>1</sup> from birth (e.g. from tricuspid atresia or univentricular heart) or become cyanosed during the first year of life (Fallot's tetralogy or a variant).
- Some children present with infective endocarditis as a complication of previously undiscovered congenital heart disease.

The common congenital heart diseases are ventricular septal defect (VSD) with a pansystolic murmur to the left of the lower sternum (not heard so well in the axilla, whereas mitral incompetence murmurs are heard well there), pulmonary stenosis, where there is an ejection systolic murmur in the pulmonary area with a single or narrowly split second pulmonary sound, and persistent ductus arteriosus (PDA), which presents in infancy with an ejection systolic murmur in the pulmonary area and split pulmonary second sound and bounding pulses. In older children with PDA there is a characteristic continuous "machinery" murmur. Less common conditions are atrial septal defects, Fallot's tetralogy (VSD, pulmonary stenosis, right ventricular hypertrophy and overriding by the aorta of the VSD) and coarctation of the aorta (heart failure, hypertension and weak, delayed femoral pulses).

- \* Only PDA is potentially treatable surgically in Malawi.<sup>2</sup> Other patients may warrant cardiac surgery outside the country, but this is difficult to arrange. However several important things can be done to help patients with congenital heart disease:
- \* Immunise them fully (especially give measles immunisation before referral of non-urgent patients for assessment).
- \* Prevent anaemia, by educating guardians in the proper and prompt treatment of malaria.
- \* Encourage guardians to avoid sweets, biscuits, and fizzy drinks, to reduce dental caries and the risk of infective endocarditis [4.9.4].
- \* Treat cardiac failure [4.9.1]

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<sup>1</sup> A useful test to differentiate cyanotic heart disease from respiratory causes of cyanosis in neonates, is to give oxygen for 30 minutes, which is ineffective in cyanotic heart disease.

<sup>2</sup> See 2.4.3 for medical treatment of neonatal PDA.

## 4.10 Gastrointestinal Diseases

### 4.10.1 Fluid Balance See section 5.5 for details.

Fluid therapy is specially important in infants and children, as they have a high water and electrolyte turnover, so abnormalities occur rapidly. Fluids can be given orally, or by naso-gastric tube, or intravenous [5.4.2] or intraosseous [5.4.3] drips. Dehydration may be due to lack of fluid intake, vomiting, diarrhoea or any other abnormal fluid losses. Avoid overhydration in hospital, especially in the malnourished.

**Fluid requirements = maintenance + replacement requirements**

#### Maintenance Requirements

These vary with age [5.5].

#### Replacement Needs - Assessing Dehydration

Replace fluids according to degree of dehydration, assessed as set out below: (WHO diarrhoea management wall charts are available in most health centres & Under 5 clinics, to which you should refer if in doubt):

Signs	Severity of dehydration	Management
2 or more of lethargy or coma sunken eyes unwilling/unable to drink skin pinch goes back v. slowly	severe	Child needs IV or NG tube fluids. Refer to hospital giving ORS on way. If cholera around, give erythromycin.
2 or more of restless, irritable sunken eyes drinks eagerly, thirsty skin pinch goes back slowly	some	give 70 ml/kg ORS in 1st 4 hours, and reassess. If breast feeding, continue, if not, give 100-200ml water
insufficient of above signs to classify as dehydrated	none	teach mother to give extra fluids, continue to feed, to look for danger signs.

In babies under 2 months any degree of dehydration shows need for hospital referral and the likely need for antibiotics for sepsis [2.4.7].

**Remember accurate weighing before and during rehydration can clearly define severity of dehydration. "No dehydration" is less than 50 gm/kg or 50 ml/kg loss and severe to over 100 ml/kg.**

**NO SIGNS = Education**

- \* Explain to mother how to treat diarrhoea at home.
- \* Give your child more fluid than usual - dilute porridge, soup, weak tea, fruit juice, rice water, breast milk (do not stop breast feeding) or milk diluted with an equal part of water.
- \* Give your child food - as much as he wants, 5-7 times a day, Easy to digest foods, with high potassium content e.g. bananas, are good.
- \* Watch for signs of dehydration - educate the mother about what to ASK, LOOK and FEEL for, to bring the child back if there are signs of dehydration, or diarrhoea persists for another 2 days.
- \* In addition, after each loose stool, give the child extra fluid (as much as he can tolerate). If the child is vomiting, give smaller amounts more frequently and slowly.

**Check that the guardian actually gives the ORS**

- \* To prevent diarrhoea in future continue exclusive breast feeding until 6 months and introduce clean, freshly prepared and nutritious weaning foods then. Dispose of children's stools, like all stools, into a latrine or by burying. All the family should wash hands after defecating and before eating or preparing food.

**Remember that in severely malnourished children, especially those with kwashiorkor, it is very difficult to assess dehydration well, and err on the side of caution in using any IV fluids. Try to rehydrate with oral ReSoMal solution [4.2, 5.5]**

**SOME SIGNS = Health centre ORS treatment**

- \* Give as much ORS as the child can tolerate. Make sure the mother has a suitable container (cup, tin) and a spoon (for an infant). Suggested

#### 4 Disease Management - Gastrointestinal Disease - Managing Dehydration

volumes are shown below (approximately 50 ml/kg every 4-6 hours):

<u>Weight</u>	<u>Volume in 4-6 hours</u>
3 - 6 kg	200 - 400 ml
6 - 9 kg	400 - 600 ml
9 - 13 kg	600 - 800 ml
13 - 20 kg	800 - 1000 ml
20 - 40 kg	1000 - 2000 ml

- \* Reassess the child after 4-6 hours:
- \* If condition is better, continue breast feeding 4-6 hourly and allow home with ORS. If not breast feeding give 100-200 ml clean boiled water 4-6 hourly with the ORS.
- \* If condition is the same, continue above regime under supervision.
- \* If condition is worse consider rehydration by drip.
- \* Educate the mother as for mild dehydration.

**The smaller the baby, the more important is exact fluid control.**

#### **MARKED SIGNS = Rehydration by drip**

- \* If IV or intraosseous [5.4.2 & 5.4.3] drip is possible, use half strength Darrow's dextrose. At all ages give 100 ml/kg over 4-6 hours (25 ml/kg/hour). If shocked (weak pulses, cold peripheries, floppy) give first 20 ml/kg over 10-20 minutes.
- \* To make sure that only the prescribed amount of fluid is given, empty out surplus fluid from the bag before starting the drip.
- \* If a drip is not available, give ORS solution (20 ml/kg/hr) under close supervision, if the baby will swallow. If the baby is NOT swallowing, give fluids by nasogastric tube 20 ml/kg/hour, stopping if the abdomen is becoming distended, and arrange for transfer to a referral unit.
- \* Reweigh dehydrated children following treatment.

**Frequently reassess dehydrated children.  
Remember there are risks in IV rehydration [5.5].**

#### 4.10.2 Gastroenteritis [see also 3.12]

Gastroenteritis causes diarrhoea and vomiting. Diarrhoea can be defined as 4 or more loose stools a day. It is usually infective, when there will often be a family or contact history. Most cases are caused by viruses, with rotavirus being most common in children. These, and most bacterial cases are self-limiting, so specific treatment is only indicated if there is invasive bacterial illness or in the case of cholera, when erythromycin can reduce diarrhoeal fluid loss and infectivity.

- \* Assess and treat dehydration [4.10.1]. Apart from rehydration, there is no specific treatment for gastroenteritis, and antibiotics are not indicated
- \* Breast feeding should be continued both to promote lactation, and to give additional fluid with a lower sodium content. Continue other feeding also. Children under 2 years old and not breast fed need to be given additional water between ORS doses (otherwise there is some danger of ORS administration leading to hypertonic dehydration).
- \* Specific treatment is needed for malaria [4.1], giardiasis [4.10.6], amoebic dysentery [4.10.4], invasive *Salmonellae* (ciprofloxacin or chloramphenicol) or *Shigella* dysentery [4.10.5]. If diarrhoea is chronic (> 2 weeks) give a dose of vitamin A (200,000 i.u. capsule).

#### **HYPERTONIC DEHYDRATION**

This is a difficult complication to manage. Suspect it on finding a rather doughy feel of the skin, or a well preserved pulse in a baby who has acidosis, is drowsy or convulsing. Check U&E to confirm if possible.

- \* Rehydrate gradually, because the blood-brain barrier slows electrolyte adjustments between blood and brain. Convulsions may result from too rapid rehydration.
- \* Observe for convulsions and anuria (can be caused by cerebral and renal vein thromboses respectively).
- \* Treat convulsions in the usual way [3.5].
- \* Acidosis is common in hypertonic and other severe dehydration. It will usually improve when the circulation is restored. Rarely, when acidosis is severe, sodium bicarbonate solution (1 ml/kg of 4.2% solution) may be given diluted in the intravenous fluid. The high sodium in this bicarbonate can worsen hypertonic dehydration.

### 4.10.3 Cholera

This is caused by one of three strains of motile curved bacteria called *Vibrio cholerae*. The organisms are not invasive, but multiply in the intestines, and produce toxins that cause profuse diarrhoea ("rice-water stools") and vomiting, leading rapidly to severe dehydration. There is no fever.

- \* Diarrhoea and vomiting is usually severe with rapid dehydration and shock. Thus rapid IV infusion of Ringer's Lactate (which has sodium and potassium concentrations similar to normal serum levels) is recommended for resuscitation. If half Darrow's dextrose is given instead, the rapid infusion of 17 mmol/l of potassium may lead to cardiac arrest. Ringer's lactate contains no dextrose and if used for long term maintenance may lead to hypoglycaemia if the child is not feeding. Thus half Darrow's dextrose should be used after the initial rapid resuscitation.
- \* The volume and duration of intravenous therapy depend on repeated reassessment of dehydration, especially noting mental alertness, capillary refill, pulse strength and urine output. Urine output may be difficult to assess in the presence of watery diarrhoea. In milder cases ORS alone may be enough.
- \* Give erythromycin orally 12.5 mg/kg 6hrly for 3 days to shorten disease and infectivity.
- \* Give guardian health education and doxycycline 300 mg stat orally.
- \* Wash hands with soap and water. Disinfect floors, beds, pots, and clothing. Isolate of patients to prevent spread of the disease.
- \* Inform environmental health staff, who will trace contacts, identify source and carry out preventative measures.

### 4.10.4 Amoebic Dysentery

This is caused by *Entamoeba histolytica*, a motile protozoon that produces resistant cysts, and should be differentiated from non-pathogenic *Entamoeba coli* by detailed microscopic characteristics. It may cause diarrhoea with blood and mucus, may also invade to cause an abscess of the large bowel wall (amoeboma), liver abscess, and even penetrate the diaphragm into the pleural cavity. In dysentery amoebae may be seen on direct microscopy of fresh stools, but in invasive disease, usually only cysts can be found, and are more easily missed.

- \* Give high dose metronidazole (15 mg/kg/dose orally 8 hrly) for 5 days.

Toxicity includes nausea and vomiting, which it is especially important to avoid in malnourished children needing feeding, so laboratory proof of this disease, which is not common in Malawian children, is advisable before treatment.

- \* Give health education on faeces disposal, hand washing and food hygiene.

#### 4.10.5 Bacillary Dysentery (Shigellosis)

Bacillary dysentery is caused by Gram negative bacilli called Shigellae. Typically the disease is acute with fever, abdominal pain, and blood and mucus in the stools. Usually the illness is brief and self-limiting, but occasionally it is severe or prolonged and needs specific treatment. One species, *Shigella shigae* (and also some genetically related enteropathic *E. coli*) may produce a toxin that can cause acute renal failure [4.11.5]

- \* If it is persistent (> 5 days) or if the child's general health is poor, give nalidixic acid 50 mg/kg/day in 3-4 doses for 5 days. Ciprofloxacin 20 mg/kg/dose once daily for up to 3 days has the advantage of being easier to ensure compliance,
- \* Resistance to many antibiotics, even nalidixic acid, occurs.
- \* Health educate on faeces disposal, hand washing and food hygiene.
- \* Carefully look for reduced urine output not related to dehydration in bacillary dysentery, and check U&Es if seen. Manage acute renal failure as in 4.11.5.

#### 4.10.6 Giardiasis

This is caused by *Giardia lamblia*, a flagellate protozoon that produces cysts. Infection is often asymptomatic, but diarrhoea, abdominal discomfort, and malabsorption can occur. The cysts are often hard to find in the stools, and treatment may have to be tried as a diagnostic test.

- \* Give metronidazole 7.5 mg/kg/dose 8 hrly for 3 days. This may cause nausea and vomiting, so avoid in malnourished children. Treatment of giardiasis should not be routine in diarrhoeal illness.<sup>1</sup>
- \* Also treat other children in the family to prevent reinfection, and give health education on hygiene (hand washing with soap and water, disposal of faeces and boiling of drinking water).

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<sup>1</sup> However the difficulty of making the diagnosis on stool microscopy, makes a trial of treatment of giardiasis in chronic diarrhoea (> 2 weeks) justifiable.



#### 4.10.7 Thrush

This is caused by a yeast, *Candida albicans* that most often affects the mouth in children, especially small babies, malnourished children and in children who have had antibiotics that have reduced the normal bacterial population of the mouth. The vagina and perianal area may also be involved. In HIV infection thrush may spread from the mouth to the oesophagus.

- \* Treat with 0.5% gentian violet paint (cheap and visually impressive, but messy) applied twice or thrice daily, or with nystatin suspension (more expensive but colourless) 100,000 u/ml, 1 ml orally 6 hrly.
- \* Severe thrush in AIDS patients requires ketoconazole 3 mg/kg/dose daily, but do not combine with rifampicin, INH or nevirapine.

#### 4.10.8 Roundworms (*Ascaris lumbricoides*)

These are very common large intestinal worms whose eggs are passed in stools, and if swallowed hatch in the stomach to give larvae that migrate through the blood stream via the lungs back to the intestine. Occasionally the migrating larvae may cause cough, wheeze and eosinophilia (suggestive of asthma). Often the adult worms give no symptoms, but sometimes a mass of entangled worms can cause intestinal obstruction, or worms may migrate into and block the bile duct, gall bladder or pancreatic duct.

- \* Treat with a single dose of albendazole

#### 4.10.9 Hookworm

These are small worms that live attached to the duodenal mucosa, from which they suck blood. Eggs are passed in stools, hatch into larvae in the ground, and after a free-living phase of development can enter through a person's skin (usually the feet), migrate through the circulation and lungs and return to the duodenum. When iron intake is inadequate and the number of hookworms large, they may cause iron deficiency anaemia, but do not cause direct symptoms.

- \* Treat with a single dose of albendazole. Give iron for anaemia.

#### 4.10.10 Strongyloides

These are small intestinal worms which have a complicated life cycle, being capable of spreading from person to person through larval forms excreted in the stools, but also able to cause auto-infection by invasion

of larvae through the intestinal wall. Serious disease occurs when immunity is suppressed (diarrhoea, bleeding from the bowel), due to rapid multiplication of the parasites, and secondary bacterial septicaemia

\* Treat with high dose albendazole (400 mg 12 hrly for 5 days), or a single dose of ivermectin, 150-200 mg/kg.

\* Consider testing for HIV infection if patient very ill.

#### 4.10.11 Intestinal Schistosomiasis

Two types of schistosomes are found in Malawi: intestinal schistosomiasis (*S. mansoni*), and urinary schistosomiasis (*S. haematobium*) [4.11.4]. Free swimming cercariae (found in water containing certain snails, the other host) penetrate the skin. The adult worms live in the veins of the bowel or bladder, and cause damage from granulomata that form round eggs they lay. *S. mansoni* can cause diarrhoea with bleeding, liver cirrhosis with portal hypertension, splenomegaly and hypersplenism, pulmonary disease leading to pulmonary hypertension and tricuspid incompetence. Ova may also reach other sites e.g. spinal cord and cause paraplegia of gradual onset. Ova can be found by microscopy of stools, but more reliably on rectal snip [5.4.8]

\* Treat both forms of schistosomiasis with a single dose of praziquantel 40 mg/kg given orally at bed time (to avoid the side effect of dizziness).

#### 4.10.12 Reye Syndrome

Reye syndrome is a rare illness presenting with convulsions and coma and liver damage (vomiting and hypoglycaemia, but usually no jaundice). It may be caused by an unusual reaction to aspirin during certain viral illnesses (chickenpox and influenza). The syndrome only occurs in childhood. Full recovery is possible but there is a high mortality. Aspirin is not recommended now in Malawi for children under 12. Use paracetamol instead, but aspirin can be given to children over 12 or used to treat rheumatic fever or rheumatoid arthritis. Aspirin should, however, be avoided during chickenpox and influenza epidemics.

\* Treatment is symptomatic, with control of hypoglycaemia and electrolyte disturbance by IV infusions, and nursing care for coma, including keeping a clear airway, controlling convulsions, regular turning to prevent pressure sores and physiotherapy to prevent contractures.

#### 4.10.13 Cirrhosis

Fibrotic scarring of the liver may follow severe liver damage from hepatitis, some herbal treatments, *Schistosoma mansoni*, and rarer conditions. It often presents with leg oedema and abdominal distension from ascites. There may be vomiting of blood from dilated oesophageal veins (varices).

- \* Look for fluid thrill and shifting dullness in the abdomen.
- \* Diagnostic ascitic tap will show a transudate.
- \* Treat symptomatically with diuretics, potassium supplements (bananas) and high protein diet (beans, groundnuts, fish).
- \* Transfuse blood for severe haematemesis.

## 4.11 Renal Diseases

### 4.11.1 Nephrotic Syndrome

This consists of oedema, massive proteinuria and low serum albumin, and is caused by excessive leakage of protein through the renal glomeruli. Other features are raised serum cholesterol and sometimes haematuria.

There are many possible causes, but in children the only treatable variety is "minimal change" in which there is usually no haematuria or hypertension. As precise diagnosis requires kidney biopsy (not usually done in Malawi), a trial of treatment is usually necessary. Untreated the disease will lead to death from infection or renal failure.

- \* Consider chest Xray before starting prednisolone, which may activate TB. If the Xray is positive, treat for TB along with the prednisolone.
- \* Give prednisolone initially in a dose of 2 mg/kg/dose orally once daily, changing to a single dose on alternate days after the first month. It may take up to 2 months to work, though usually if the nephrotic syndrome is responsive, some reduction in proteinuria occurs within 4 weeks.

**Always check blood pressure before starting steroids in nephrotics**

- \* Watch for hypertension as a result of prednisolone.
- \* Once the urine becomes clear of protein for 5 days, tail off the steroids, but be prepared to reintroduce them one or more times if there is a relapse.
- \* Initially restrict salt and water intake, to prevent increasing oedema and ascites.
- \* Give frusemide only if oedema is so bad it interferes with breathing or passing urine. If you use frusemide, watch for electrolyte disturbances and shock. Frusemide is NOT routine, as this may cause hypovolaemic shock in nephrotic syndrome (where the intravascular volume is already significantly reduced).
- \* Give a high protein diet.
- \* Amoxicillin may be needed as pneumococcal infections, including pneumococcal peritonitis are very common because of antibody loss in the urine.
- \* Most typical ("minimal change") nephrotics recover, though this may take months, and include relapses. It is important to teach a relative

early how to test the urine for protein and record the results, and to attend long term follow up at the hospital.

- \* If there are repeated relapses, oral cyclophosphamide may be of value, but should be given under specialist supervision.

Unfortunately many nephrotics are atypical (e.g. may have red cells in the urine, or become hypertensive with prednisolone) and the less typical they are, the less likely are they to respond to treatment. The atypical patients probably don't have "minimal change" nephrotic syndrome, but some other form (e.g. due to hepatitis B, quartan malaria, glomerulonephritis).

#### 4.11.2 Glomerulonephritis

Acute glomerulonephritis is an immune reaction, usually to a beta haemolytic streptococcal infection of the skin (often secondary to scabies) or throat. It presents as an acute illness with some combination of oedema (usually facial), oliguria, haematuria and hypertension. The majority of patients recover spontaneously in a few weeks, but some die from the acute effects of hypertension or go into acute or chronic renal failure. Rarely glomerulonephritis may progress to the nephrotic syndrome.

- \* Treat scabies, if present.
- \* Give 10 days of amoxicillin (to eliminate streptococci from skin or throat) – or benzathine penicillin for tonsillitis.
- \* Restrict oral water and sodium intake to reduce the oedema. Restrict protein intake if urea is elevated.
- \* Reduce potassium intake (no fruit and vegetables) while urine output is reduced. Later during recovery there may be a diuresis in which salt and potassium are lost and need replacement.
- \* Complications, such as hypertensive convulsions, cardiac failure [4.9.1], and renal failure [4.11.5], may need further treatment.

#### 4.11.3 Urinary Tract Infection (UTI)

Infection may involve the bladder or the upper tract with kidney infection. In girls such infections are common without predisposing cause, but in boys more often infection may be secondary to abnormality of the urinary tract, especially if there is bladder neck obstruction, or may be associated with ureteric reflux. Symptoms may be acute with urinary frequency, dysuria and fever, but are often vague, with poor general

health and perhaps vomiting or diarrhoea and failure to thrive. Diagnosis requires urinary microscopy, and preferably culture. Recurrent infections may require further investigation to detect correctable abnormalities, and prolonged suppressive antibiotic treatment to prevent recurrences that can lead to renal damage and failure.

- \* Give cotrimoxazole for 7-10 days or perhaps amoxicillin, nitrofurantoin (dosage of 1.5 mg/kg/dose 6 hrly) or ciprofloxacin 5-10 mg/kg/dose b.d.
- \* Check for recurrences for many months thereafter.
- \* In relapsing patients long term low dose cotrimoxazole or nitrofurantoin (0.5 mg/kg/dose 6 hrly) may be needed.
- \* Treat any underlying schistosomiasis.
- \* If there are repeated relapses, ultrasound or an IVU may show a surgically correctable abnormality (hydronephrosis or hydroureter). Ureteric reflux is an associated and probably causative lesion, treatable by prolonged low dose cotrimoxazole rather than surgery.

#### 4.11.4 Urinary Schistosomiasis

Urinary schistosomiasis (bilharzia) is a helminth infection caused by *Schistosoma haematobium* worms that live in the veins of the bladder, and lay eggs which cause damage by the inflammatory granulomata they excite. Such damage is primarily in the bladder, but may affect the genital tract (fallopian tubes and uterus etc). Haematuria is a common result, and ureteric obstruction with renal failure can occur, and as a late complication (after childhood) bladder cancer.

- \* Examine urine microscopically for ova, preferably a mid morning specimen taken after exercise.
- \* Treat with a single dose of praziquantel 40 mg/kg orally (at bed time to avoid the side effect of dizziness).

#### 4.11.5 Renal Failure

May be acute, when the causes can be classified as prerenal (following shock from blood or fluid loss), renal (as in acute glomerulonephritis, *Shigella shigae* bacillary dysentery [4.10.5], haemolytic-uraemic syndrome [4.11.6] and herbal remedies), or postrenal (obstruction of the urinary tract e.g. from calculi, or from neurological disease leading to retention) or may be chronic from various diseases.

- \* If there is shock (prerenal cause), treat [3.3], but avoid overhydration.

#### 4 Disease Management - Renal Diseases - Renal Failure, HUS

- \* If there was shock that has been corrected (capillary refill time, pulse, blood pressure restored) but there is no urine on catheterisation, give frusemide 2 mg/kg IV stat.
- \* As acute renal failure may be reversible, maintain the child's biochemistry as near normal as possible until recovery occurs. Such patients should if possible be transferred to a central hospital where biochemical monitoring and possible peritoneal dialysis can be done.
- \* Prevent serum potassium rising above 6.5 mmol/l, by restricting fruit, vegetable, meat and fizzy drink intake.
- \* Restrict salt and water intake (limit to 10 ml/kg/day plus urine output).
- \* Keep a careful fluid intake and output chart and weigh child daily.
- \* Reduce the rate of rise in urea, by giving adequate calories with a low protein diet.
- \* Check U&Es frequently.
- \* Treat any infection vigorously to reduce fever and catabolism, which release potassium from the cells. (Avoid antibiotics excreted in urine).
- \* If these measures fail peritoneal dialysis may be indicated, so refer the patient to a specialist centre where this is possible before the child's condition is critical.

#### **Drugs in renal failure**

Great care must be taken over prescribing medicines. Many drugs are excreted through the kidneys and accumulate when urinary output is reduced, and others are presented as sodium or potassium salts, contributing to the accumulation of these electrolytes.

- \* Usually safe: chloramphenicol, erythromycin, penicillin, phenytoin, rifampicin
- \* Use with care and reduce dose: amoxicillin, cotrimoxazole, isoniazid, phenobarbitone
- \* Drugs to avoid: digoxin, gentamicin, streptomycin, doxycycline

#### 4.11.6 Haemolytic-uraemic syndrome (HUS)

This illness, affecting mainly children under 5, is caused by a toxin (verotoxin), produced by some *E. coli* bacteria and *Shigella shigae*, that damages small blood vessels in the intestine and kidney. Red blood cells passing through the damaged vessels break down. The illness starts with diarrhoea, sometimes bloody, then there is a febrile illness and

haemolytic anaemia, petechiae from low platelets, and renal failure. Recovery is usually complete if the child survives the acute renal failure.

- \* Transfuse if anaemia is severe [3.9.1].
- \* Treat for acute renal failure [4.11.5].
- \* Check for hypertension [4.9.5].
- \* Observe strict hygiene (handwashing, safe disposal of faeces) to prevent bacterial spread.

## 4.12 Nervous System Diseases

### 4.12.1 Meningitis

Meningitis (more accurately meningo-encephalitis) is an infection involving the meninges and brain. In Malawi bacterial infections with *S. pneumoniae*, *N. meningitidis*, and *H. influenzae* are most common causes. In infants salmonellae, and in neonates group B streptococci and coliforms are also important causes. Other causes include TB, various viruses, trypanosomiasis in its later stages, and in the immunosuppressed a yeast, *Cryptococcus neoformans*.

Symptoms vary with the acuteness of the illness and age of the child, and may include fever, convulsions, headache, neck stiffness, cough and vomiting. Meningococcal meningitis may be accompanied by widespread skin haemorrhages and shock.

Diagnosis is by lumbar puncture (LP) for examination of the CSF, except if the lumbar sin is infected, there is severe dyspnoea, or a coma score of zero or cranial nerve lesions, suggest raised intracranial pressure with a danger of coning.<sup>1</sup> In such cases wait till presumptive treatment has given some improvement. But such a delay, will result in loss of diagnostic accuracy, and may make the differentiation of partly treated pyogenic from tuberculous meningitis very hard. Remember that non-viral meningitis is a severe, life-threatening infection, and treatment should not be delayed if LP is not immediately possible.

- \* If you suspect meningitis the patient must be referred quickly to a hospital for urgent treatment, but FIRST:
  - \* Do an immediate LP, if at all possible, and send the CSF in a sterile container with the patient for cells, Gram stain, protein and glucose.
  - \* Give benzyl penicillin 50,000 u/kg/dose IV or IM stat and chloramphenicol. If not available give whatever antibiotics you have.

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<sup>1</sup> Also avoid LP if the patient has a non-functioning shunt for hydrocephalus [4.12.2],



#### 4 Disease Management - Neurological Disease - Meningitis

- \* In hospital give benzyl penicillin (50,000 u/kg/dose 4 hrly) and chloramphenicol<sup>1</sup> (25 mg/kg/dose 6 hrly), both by IV bolus, while awaiting the result of the tests. When the patient improves (conscious, no fever, headache or vomiting, and is able to feed), change to oral chloramphenicol only. (For neonatal meningitis see 2.4.7).

#### SPECIAL SITUATIONS

- \* If chloramphenicol is in short supply, give benzyl penicillin for *N. meningitidis* or *S. pneumoniae*, and ampicillin for *H. influenzae*, but preferably ceftriaxone IM, as it is now often ampicillin resistant.
- \* If progress on benzyl penicillin and chloramphenicol is poor, give ceftriaxone (daily IM or IV) or cefotaxime (given 6 hourly IV), if either is available. Outside central hospitals ceftriaxone and cefotaxime may not be available. Then use IV ampicillin.<sup>2</sup>

#### DURATION OF TREATMENT

<i>N. meningitidis</i>	- 7 days
<i>S. pneumoniae</i>	- 14 days
<i>H. influenzae</i>	- 10-14 days (depending on progress)
No bacteriological diagnosis	- 10-14 days (depending on progress)
Neonates	- 3 weeks [2.4.7]

- \* If staff are short, prolonged IV infusions carry risks of fluid overload and drip sepsis, so change to oral chloramphenicol when tolerated.
- \* Avoid too much IV fluid, which can give cerebral (brain) oedema.
- \* Give anticonvulsants for several days to all meningitis patients who convulse [3.4].
- \* Give paracetamol while there is fever or pain, if the child can swallow.
- \* Ensure good nursing care, especially in the unconscious patient:
  - \* Keep a clear airway (position of patient, suction).
  - \* Control fluid intake with care not to give too much by IV or NGT.
  - \* Maintain food intake by NGT if necessary, especially if long in coma.
  - \* Prevent pressure sores by turning comatose children.
  - \* Clean the mouth well to prevent parotitis in the comatose.

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<sup>1</sup> Chloramphenicol is the current recommended antibiotic, as it is effective against most organisms which cause meningitis, and its use avoids multiple injections and prolonged IV infusions.

<sup>2</sup> Ampicillin enters the CSF better than benzyl penicillin, but now seldom kills *H. influenzae*.

- \* Give physiotherapy - postural drainage and percussion of chest to prevent and treat hypostatic pneumonia, and passive limb movement for paresis or paralysis to prevent contractures.
- \* Explain about the disease to guardians otherwise confidence is lost and they may abscond.
- \* If progress is poor recheck LP to see if inflammation persists.

### Normal and abnormal CSF values

The table shows normal CSF values and typical values for bacterial, viral and TB meningitis.

	<u>Normal</u>	<u>Bacterial meningitis</u>	<u>Viral meningitis</u>	<u>TB meningitis</u>
<b>Protein</b> (mg/dl)	20-40	100-500	50-200	100-500
<b>Glucose</b> (mg/dl)	30-50	usually < 40 (may be 0)	normal	moderately low (< 50)
<b>White blood cells</b> (/c.mm.)	0-5	100-60000	20-200	20-500

### Note:

1. In neonates up to 50 white blood cells (mainly lymphocytes) and protein up to 150 mg/dl may be normal.
2. In bacterial meningitis the white cells are mainly polymorphs, whereas in viral and TB meningitis the white cells are mainly lymphocytes. However polymorphs may predominate very early on in the course of viral and TB meningitis and in partially treated bacterial meningitis lymphocytes may predominate.
3. CSF findings in brain and epidural abscess are similar to those seen in viral meningitis.
4. Early meningitis may have only minor CSF abnormalities.

### 4.12.2 Cryptococcal meningitis

Meningitis caused by a fungus, *Cryptococcus neoformans*, occurs almost exclusively in AIDS patients (rarely in patients on high prolonged steroids or cancer chemotherapy) and is an AIDS defining disease. It may be acute or gradual in onset, not always with much fever, and is likely to present with mental changes, headache and perhaps cranial nerve lesions,

occasionally convulsions. CSF usually shows some increase in protein and reduced glucose. Cells are usually modestly increased and mainly lymphocytes, but the critical examination is to make an Indian ink preparation to show the fungi and their capsules on microscopy.

In AIDS treatment is not curative, but control of the infection may be gained with fluconazole 6-12 mg/kg/dose daily IV initially then orally. After 4 weeks of treatment, recheck the CSF and consider reducing the dose to 6 mg/kg/dose daily orally for life to prevent recurrence. After cryptococcal meningitis has been controlled, start ARVs.

#### 4.12.3 Hydrocephalus

Hydrocephalus is an abnormal and excessive accumulation of CSF, usually caused either by blockage of the circulation of the CSF or by impaired reabsorption. It may be congenital (often associated with spina bifida) or acquired, usually secondary to meningitis or perhaps brain tumour or intracerebral haemorrhage. Onset before age 2 increases head size<sup>1</sup>.

- \* Hydrocephalus can be treated surgically by a shunt.<sup>2</sup> This drains CSF from a lateral ventricle to the peritoneal cavity.
- \* Refer patients to a paediatrician for assessment of suitability.
- \* Patients with shunts who have headache, vomiting, ataxia, cranial nerve lesions or visual problems may have a blocked shunt and a raised intracranial pressure. Do NOT do an LP on such patients as death from coning may occur. Start treatment for meningitis and refer to a central hospital.

#### 4.12.4 Epilepsy

A condition of recurrent intermittent abnormal electrical activity of the brain. It may be "idiopathic" (i.e. the underlying cause is not understood, though there may be a family tendency), or secondary to previous brain damage (birth asphyxia, head injury, cerebral malaria or meningitis) or to congenital brain abnormality. The pattern of epilepsy may be of generalised convulsions with loss of consciousness and posture ("grand mal" attacks), or may have partial forms, where posture is not lost and only part of the body is visibly affected. A type of epilepsy sometimes seen in children is "petit mal," in which the child appears to have periods of inattention, and perhaps minor lip or eyelid movement.

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<sup>1</sup> See section 5.1.4 for normal head circumference values.

<sup>2</sup> Shunts give a number of problems (blockage and infection).

- \* Epilepsy, whether idiopathic or secondary to brain damage, can usually be controlled completely or largely by the regular use of the anticonvulsants, phenobarbitone or phenytoin. Carbamazepine and valproate are more expensive alternatives.
- \* Many parents, however, have more faith in traditional treatments, and to achieve good results time must be spent discussing antiepileptic treatment.
- \* Make it clear that treatment is not curative, but preventive, and has to be regular and long term.
- \* Tell parents that the dose of anticonvulsant may have to be increased, and the drug changed, if not fully effective.
- \* Get the guardians to keep a record of convulsions to guide the dosage of drug, and to convince them of efficacy. Guardians also need to know drug overdose and toxic effects.
- \* Make arrangements for epileptics to obtain regular drug supplies without long waits in queues to improve compliance (e.g. run a special clinic). Treatment is usually for at least 3 years, and may be lifelong.
- \* Drug changes should be gradual, with introduction of the new drug in stages at intervals of about a week, as the old one is reduced.
- \* Give phenobarbitone 3-8 mg/kg/dose once daily, preferably before bed. Although theoretically it should be started at the lowest dose, to obtain a convincing effect rapidly, it may be wise to start at 5 mg/kg/dose once daily. Overdose leads to sleepiness and poor school performance, and a few patients may develop allergic skin rashes (stop treatment immediately). A few children become hyperactive, destructive and aggressive on phenobarbitone, and have to be changed to phenytoin.
- \* Phenytoin dosage is 4-8 mg/kg/dose once daily. Overdose causes ataxia (clumsiness and a tendency to trip), or nystagmus. Other toxic effects include gum hypertrophy, breast enlargement, increased hair, abnormal calcium metabolism, folic acid deficiency anaemia and skin rashes. Fortunately none is common.
- \* Petit mal epilepsy is unusual. The patient does not fall down or twitch, but seems to have short periods of apparent loss of awareness of what is happening, and may blink the eyes. Drugs of choice, if available, are valproate (20-40 mg/kg/day) or ethosuximide (15-50 mg/kg/dose nocte). Side effects include nausea, dizziness, headache (ethosuximide) and increased appetite, low platelets (valproate).

#### 4.12.5 Brain Abscess

Brain abscesses usually occur secondary to chronic perforated otitis media, compound fractures of the skull, or congenital cyanotic heart disease. They present with symptoms and signs of increased intracranial pressure (headache, vomiting, unsteadiness and cranial nerve palsies), but there may be meningism, fever, and focal neurological damage. Various organisms, including anaerobes, may be the cause.

- \* Surgical drainage of the abscess should be done as soon as possible. However this is difficult in Malawi with limited diagnostic facilities for localisation. The underlying cause (chronic discharging ear, previous compound depressed fracture) and neurological signs must be taken into account when localising the probable abscess site for surgery.
- \* Culture any accessible pus to guide antibiotic use. Initially give high dose benzyl penicillin (50,000 units/kg/dose IV 4 hrly) and chloramphenicol (25 mg/kg/dose 6 hrly) because of the latter's good penetration. Give gentamicin when there is a chronic discharging ear or metronidazole when anaerobes are suspected.
- \* Temporary control of raised intracranial pressure may be necessary, by using dexamethasone to reduce cerebral oedema in a dose of 0.3 mg/kg IV stat with 0.2 mg/kg/dose 6 hrly IM.

#### 4.12.6 Brain Tumour

Brain tumours present with symptoms and signs of increased intracranial pressure (vomiting, headache, papilloedema and pupillary changes with brain herniation). Other presentations include cerebellar ataxia. When symptoms are due to secondary obstructive hydrocephalus, rather than the growth of the primary tumour, they may be relieved temporarily by drainage of CSF from a lateral ventricle. Otherwise the tumour itself is rarely treatable, except that the single most common intracranial tumour in Malawian children is Burkitt's lymphoma [4.17.1]. Tuberculoma - not a real tumour - is a rare though treatable cause (with chemotherapy as for TB meningitis [4.5]).

- \* Burkitt's lymphoma may respond well to intrathecal methotrexate 12.5 mg/m<sup>2</sup> surface area, given with intrathecal hydrocortisone and IV cyclophosphamide for Burkitt's lymphoma [4.17.1] outside the nervous system.

#### 4.12.7 Cerebral Palsy (CP)

A non-progressive neurological damage of the motor system in childhood, usually originating at birth or soon after. Causes include congenital brain abnormality, birth asphyxia, neonatal hypoglycaemia, kernicterus, early head injury, cerebral malaria or meningitis. Though the damage is static, its apparent effects may change during childhood as different parts of the brain begin to function and behaviour matures. Cerebral palsy can be spastic (stiff), hypotonic (floppy) or athetoid (abnormal movements), and may show a hemiplegic, monoplegic, diplegic or quadriplegic distribution.

- \* Skilled physiotherapy improves posture, tone and mobility and prevents contractures. Unfortunately there is reduced intelligence in many (but not all) cases, which makes physiotherapy more difficult.
- \* Look for other deficits, such as deafness or poor vision.
- \* Treat epilepsy if present.
- \* Detailed assessment of the child's neurological problems is needed. Counselling of the parents on the realistic prospects of the child is important to reduce disappointments and prevent waste of their resources in the search for miracle cures.

#### 4.12.8 Paraplegia

Paraplegia [3.14] means paralysis of both lower limbs (incomplete forms are more correctly called paraparesis) and may be flaccid, when the cauda equina or peripheral nerves are affected, or spastic when the long tracts in the cord are damaged, with absent inhibition of spinal reflexes (though early in acute cord damage legs may be flaccid). The most common paediatric cause in Malawi is Burkitt's lymphoma, followed by spinal TB, Guillain-Barre syndrome, trauma, schistosomiasis, transverse myelitis and possibly polio.

- \* Give specific treatment for the cause if available (e.g. cytotoxic drugs for Burkitt's lymphoma [4.17.1], short course TB treatment for TB spine with paraplegia, praziquantel for schistosomiasis, high dose antibiotics for epidural abscess).
- \* If the exact diagnosis has not been made it is reasonable to give praziquantel (relatively non-toxic), but only give the other treatments when there is good evidence for the diagnosis.
- \* For TB spine surgery is usually not indicated in Malawi and plaster casts are not effective.

- \* Patients in whom the cause of paraplegia is unclear, should be referred early for specialist advice on whether a myelogram is indicated, with a view to possible surgical decompression.<sup>1</sup>
- \* Ensure good nursing care. Turn the child regularly to prevent pressure sores over the trochanters and sacrum (and teach guardians to do so).
- \* Prevent maceration of the skin from incontinence of urine. In the male use Paul's tubing or a home-made substitute attached to the penis. In the female catheterisation may be necessary, though that carries serious risk of urinary infection, and may require continuous cotrimoxazole treatment.
- \* Ensure regular full passive movement at the ankle, knee and hip joints.
- \* If available, ask a physiotherapist to help in mobilising the patient.

## 4.13 Disease of Bones and Joints

### 4.13.1 Septic Arthritis

Salmonella is the most common cause in children under 2 years during the wet season and also common in HIV infected children. At other times of the year and in older children staphylococci are most common and in neonates group B streptococci and staphylococci are very common. These age and seasonal patterns determine antibiotic choice.

- \* Aspirate the affected joint for Gram stain, culture and sensitivities before starting antibiotics.
- \* Give chloramphenicol for 2 weeks. Modify treatment if culture results are reported. Staphylococci may need flucloxacillin or salmonellae require ceftriaxone or ciprofloxacin, if response to treatment is poor.
- \* Give paracetamol for pain.
- \* Rest the joint in the position of function (at right angles for the elbow and ankle, at 5-10 degrees of flexion for the knee) using a plaster of Paris cast.
- \* Xray the joint and adjacent bones (out of plaster) after 2 weeks looking for osteomyelitis. If osteomyelitis is not present, 2 weeks of antibiotics are enough.
- \* In infants and in all septic arthritis of the hip and shoulder, there is often adjacent osteomyelitis and antibiotics for 4 weeks should be given [4.13.2]. Remember congenital syphilis is a possible cause of "arthritis" (really osteitis of adjacent bone) in infants [4.7.6]

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<sup>1</sup> When a myelogram is likely, do not do a preliminary lumbar puncture.

#### 4.13.2 Osteomyelitis

In acute osteomyelitis appropriate antibiotics need to be given in high dosage for at least 4 weeks. The organism should be identified by blood culture in the very early stage, when there is only high fever, or by aspiration, or drilling of the infected bone when the presence of pus is indicated by tenderness or swelling. Most commonly the infecting organism will be a staphylococcus or salmonella (treated with flucloxacillin, or both chloramphenicol and gentamicin, or ciprofloxacin or ceftriaxone). Xray bone changes in osteomyelitis are not seen before 10 days, so take Xrays after 2 weeks to rule out osteomyelitis.

- \* Give the same dose of flucloxacillin or chloramphenicol as for septic arthritis [4.13.1], but for 4-6 weeks.
- \* Splint affected limb with a plaster of Paris cast in position of function.
- \* In small babies osteomyelitis often resolves completely, but in older children there can be a chronic phase of sequestrum (dead bone) formation, which needs to be removed surgically once the involucrum (new bone shaft) is well enough developed to bear weight and not fracture.

**Remember congenital syphilis can give chronic osteitis in infancy [4.7.6]**

- \* Check Xrays after 3-4 months to confirm that the involucrum is adequate.
- \* Give antibiotics again at the time of operation.

#### 4.13.3 TB of the Spine

TB of the spine is almost always best treated conservatively with TB treatment, even if there is paraplegia [4.5, 4.12.7].

- \* There is no proven value in plaster of Paris casts in this condition. Although surgery may relieve pressure on the spinal cord and allow recovery of paraplegia more rapidly, there is the danger of secondary bacterial infection unless surgery is done under strictly aseptic conditions, which are difficult to achieve in Malawi.

#### 4.13.4 Juvenile Rheumatoid Arthritis

An inflammatory disease of unknown, probably auto-immune, cause, which may affect multiple, especially large synovial joints. It often



initially presents with a systemic illness (fever, rash, hepatosplenomegaly and weight loss) before the arthritis appears. Serology for rheumatoid factor is usually negative.

- \* Give ibuprofen (5-7 mg/kg/dose 6 hourly) or aspirin (10 mg/kg/dose up to 25 mg/kg/dose 6 hrly if necessary). Aspirin should always be given with food and is safe in children over 12, but should be avoided in younger children during outbreaks of chickenpox and influenza because of the risk of Reye syndrome.
- \* Splint involved joints in the position of function, using POP casts, to prevent crippling deformities. Apply splints at night, removing them for part of the day, to allow some movement to occur at the joints and reduce stiffening.
- \* If ibuprofen and aspirin are ineffective, give indomethacin, after consultation with a paediatrician.
- \* Refer severe, crippling cases to a paediatrician.

## 4.14 Endocrine Diseases

### 4.14.1 Diabetes Mellitus

Diabetes mellitus is almost always of the insulin dependent type in children. Presentation is acute or subacute, with a history of polyuria, thirst (polydipsia) and recent loss of weight. There is often a precipitating cause such as pneumonia (or other infection). On examination there may be dehydration, acidosis, ketosis, abdominal pain, vomiting and thirst. Diabetic ketoacidosis is a medical emergency presenting with acidosis, vomiting, abdominal pain, dehydration, hyperventilation and clouded consciousness or coma.

#### DIABETIC KETOACIDOSIS

- \* Take blood for glucose and U&Es.
- \* Severe dehydration is invariably present, and its correction [5.5] is as important as insulin. Give IV Ringer's lactate or normal saline at 15-20 ml/kg in 1 hour, and then 10-15 ml/kg/hour until acidosis is corrected (slower breathing, breath not ketotic, no ketones in the urine). Calculate first 24 hour needs as in section 5.5, but a good rough guide for children over 6 years is to give 1 litre of fluid in the first hour, 1 litre in next 2, and 1 litre in next 3 hours. If there is coma, replace deficits more slowly in 48 hours to avoid brain oedema.

- \* When the child has passed urine, add KCl to IV infusion at 3 mmol/kg/24 hrs, changing to oral K supplements when able to feed.
- \* Give soluble insulin (0.5 u/kg) s.c. stat, even if the usual daily dose has been given.
- \* Then give insulin 6 hrly 0.5 u/kg, if blood glucose > 20 mmol/l, 0.4 u/kg for glucose 15-20 mmol/l, 0.3 u/kg for glucose 10-15 mmol/l, 0.2 u/kg for glucose 5-10 mmol/l and 0.1 u/kg for glucose 3-5 mmol/l.
- \* Once blood glucose is < 300 mg/dl (15-20 mmol/l) change IV fluids to half Darrow's dextrose, and continue soluble insulin.
- \* The next day give the usual daily dose of insulin. In a newly diagnosed diabetic, give insulin 0.5-1 u/kg/day,  $\frac{1}{3}$  dose as soluble and  $\frac{2}{3}$  dose as lente (protaphane)
- \* Adjust insulin doses according to subsequent results of urine tests<sup>1</sup> for glucose and ketones (acetone), with changes of no greater than 10% of the total daily insulin each day. Aim for negative urine tests, a fasting blood sugar of 100 mg/dl (5.5 mmol/l) and an evening (4.30 pm) blood glucose of 150 mg/dl (8 mmol/l)

**Do not change the insulin dose by more than 10% of the daily total.**

#### ADJUSTING INSULIN DOSES

TYPE	Begins to Act	Peak Effect	Duration
Soluble	< 1 hr	2-4 hrs	6-8 hrs
Lente (protaphane)	2-4 hrs	8-12 hrs	18-24 hrs

The above table shows that the urine tests before breakfast, supper and bed time reflects the action of protaphane insulin, whereas the urine test before lunch reflects the action of soluble insulin. Therefore:

- \* if there is glycosuria before breakfast, supper, and bed time increase the protaphane, and if tests are negative, reduce the protaphane.
- \* if there is glycosuria before lunch, increase the soluble, and if tests are negative, reduce the soluble.
- \* Insulin orders should therefore be made after the results for glucose in the urine in the previous 24 hours are known. The results before lunch and supper are essential for this.

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<sup>1</sup> Ideally the urine should be tested for glucose and ketones before breakfast, lunch, supper, and at bed time.

## EDUCATION

The long term management of diabetic children is difficult. It is essential that both children and family receive education before the child is sent home. Poor control of diabetes is usually associated with poor understanding of the disease by the patient and family. Points which should be covered include:

- \* General - this life long disease can't be cured, but can be controlled, as long as the patient attends clinics regularly. Advise patients to wear shoes if possible to minimise foot complications.
- \* Diet - complex carbohydrates are better than refined (i.e. eat any vegetables and fruits, root vegetables, ngaiwa, fish, meat and eggs, but avoid sugar, juicy drinks, biscuits, white bread and polished rice).
- \* Urinalysis - teach urine testing and how to read results.
- \* Insulin - teach how to measure and draw up insulin doses into the syringe (it may be necessary to mark the syringe). Insulin should be stored in a fridge or an earthenware jar in a cool shady place standing in water, and the needles and syringes kept clean.
- \* Hypoglycaemia - the patient should be made to experience a hypoglycaemic attack in hospital and told how to deal with them (i.e. always to carry some sugar or snack).

### 4.1.2 Cretinism

Cretinism is also known as congenital hypothyroidism of which there are two main types. The first type (classical) is due to inadequate thyroid function in the baby secondary to iodine lack, biochemical abnormality of the thyroid or absence of thyroid gland. In the first 2 situations there may be a goitre. This type presents with retardation both of linear growth and mental development. The second type (neurological), is due to a deficiency of maternal thyroid hormones in the early part of pregnancy (before the fetal thyroid has developed), and causes irreversible brain damage.

Cretinism may presents as prolonged neonatal jaundice, constipation, a hoarse cry and slow development. Early diagnosis of cretinism is difficult but very important if treatment is to be effective in preventing mental retardation. It is equally important to convince the guardians of the need for lifelong treatment with thyroxine by careful explanation. Treatment of cretinism so changes the appearance of the child, that the diagnosis is no longer apparent after some months of treatment.

Therefore it is very important to document the diagnosis with thyroid function tests, Xrays of epiphyses or photographs of the patient before treatment. Xrays of the epiphyses show delayed development and abnormal appearances.

- \* Give levothyroxine initially 5 micrograms/kg/dose once daily, rising to 50 mcgs (0.05 mg) in infancy, and 100 mcgs (0.1 mg) by 5 yrs.
- \* Record weight and height to compare with normal values, and assess the child's growth (record in the health passport) and development as guides to dosage. Unfortunately, unless the diagnosis is made and treatment is started very early, normal growth may occur without normal mental development.

## 4.15 Skin Diseases

### 4.15.1 General Principles of Treatment

Many skin conditions can be treated without knowing the specific diagnosis e.g. since many are inflammatory, general measures in the treatment of inflammation are often very effective.

- \* If severe and acute use milder degrees of treatment (powder, wet dressings with boiled cool water, lotions and emulsions).
- \* If sub-acute use lotions and creams.
- \* If dry and chronic use ointments.
- \* If you can't think of what to do, then it is often best to do nothing, except advise rest and rely on the ability of the skin to heal itself.

### 4.15.2 Congenital Malformations

#### *MONGOLIAN SPOT*

These are very common in dark skinned babies, present as a blue-black patch over the lumbo-sacral area, which fades with time and requires no specific treatment.

#### *SALMON PATCHES AND PORT WINE STAINS*

These are capillary haemangiomas, occurring on the face, neck, and sometimes on limbs (unilateral). If associated with mental retardation and epilepsy, this could be due to the Sturge-Weber syndrome. They shrink with time, but don't disappear. There is no treatment.

#### *STRAWBERRY MARKS*

These are due to haemangiomas that appear 2-4 weeks after birth, as reddish nodules. They grow for the first year, then gradually shrink and

disappear by 5 years. The main problems are maternal anxiety, and cosmetic embarrassment. There is no specific treatment, but it is important to follow up the child and repeatedly reassure the mother, to prevent unnecessary surgery and traditional treatments. Refer children with very large haemangiomas to a paediatrician who may use steroids.

#### 4.15.3 Changes in Pigmentation

##### *ALBINISM*

Albinism is an inherited autosomal recessive disease. Absence of the pigment melanin in the skin results in white hair, pink pupils, white skin which is easily burnt by the sun, and later goes on to develop solar keratoses, melanoma and squamous cell carcinoma. Early death is common. There are also often photophobia and nystagmus.

\* Protection of the skin (with hats, long sleeves and trousers, gloves and sun-screen creams) and of the eyes (with sunglasses) from the sun is vital. Refer to ophthalmologists.

**Take every chance to advise albinos to protect their skin with hats, long sleeves, long trousers, gloves and eyes from sunlight.**

\* Advise to seek indoor jobs and activities.

##### *VITILIGO*

This is a common acquired disease of unknown cause, presenting with round or oval pale patches of pale skin, which enlarge at a very variable rate (harmless, save for cosmetic embarrassment).

\* There is no treatment, except to protect the patches from sun.

##### *PITYRIASIS ALBA*

This is a common condition, presenting as lighter, slightly scaly patches. Lesions are usually on the face and heal without treatment.

#### 4.15.4 Bacterial Skin Infections

##### *IMPETIGO*

This commonly occurs on face as blisters which easily rupture, leaving oozing and then crusting. Satellite lesions may occur. Group A streptococci are the main cause, with later colonisation by staphylococci.

\* Treat localised milder lesions with soap and water, or GV paint.

\* In addition treat spreading lesions with erythromycin or flucloxacillin.

### *BULLOUS IMPETIGO*

Here the blisters are thick-walled, do not rupture that easily, and are usually due staphylococci.

- \* Soften crusts with emulsifying ointment (containing sulphur salicylic acid), then remove crust with normal saline soaks and apply GV paint.
- \* Give erythromycin or flucloxacillin.

### *BULLOUS IMPETIGO OF THE NEWBORN*

This is a highly contagious form of bullous impetigo which presents in infants (often in epidemics) and is caused by penicillinase producing staphylococci. The staphylococcal scalded skin syndrome is a severe form of this disease caused by an exotoxin producing sub-strain of staphylococci. The skin rapidly goes red, then forms big blisters, which burst shedding the top layer of skin. This condition can be rapidly fatal, though with prompt treatment recovery is complete within a week.

- \* Treat with flucloxacillin IM or IV (if not available use gentamicin).
- \* Maintain hydration and electrolyte balance.

### *ECTHYMA*

Lesions follow trauma and are due to infection of the total thickness of the skin (by staphylococci or streptococci), which sloughs off forming an ulcer with red margins, and then heals slowly with scarring.

- \* Remove crusts as for impetigo, and apply GV paint.
- \* Treat with flucloxacillin.

### *ERYSIPELAS*

Erysipelas is an acute streptococcal infection of the upper layers of skin. It presents with systemic signs (fever and chills), and a rapidly spreading erythematous, painful rash with a well defined edge. The skin sheds off causing superficial ulcers which often become secondarily infected. The condition can be rapidly fatal unless treated with systemic penicillin.

- \* Treat with benzyl penicillin IM. Expect improvement within 2 days.

### *FOLLICULITIS*

This is due to infection of the upper parts of the hair follicle, and presents as tiny pustules with a central hair and surrounding erythema. In babies it is most common on the scalp, and in children on the legs. It is extremely common in Malawi due to the use of greasy ointments (vaseline), which block glands around the base of the hairs.

- \* Stop the use of greasy substances that probably caused the problem in the first place.
- \* Apply a drying agent (calamine lotion with sulphur added), or paint all

lesions with GV paint.

- \* Use flucloxacillin if lesions are extensive and deep.

#### *FURUNCULOSIS*

These are painful boils due to infection of the whole hair follicle including the sebaceous gland.

- \* If single and for the first time, no specific treatment is indicated.
- \* If recurrent or multiple, treat with flucloxacillin for at least 2 weeks.
- \* If large and fluctuant, incision and drainage is indicated.

#### *STY*

A sty (external hordeolum) is an infection of the glands of the eyelid. *Staphylococcus aureus* is the usual cause.

- \* Apply warm compresses.
- \* Tetracycline eye ointment.
- \* If abscess forms, incise and drain and give flucloxacillin.

### 4.15.5 Fungal Skin Infections

#### *TINEA CAPITIS*

This is a common superficial fungal infection of the head which affects mainly children. It spreads directly between affected people, via combs, brushes, or from infected animals and pets. It presents as round patches, with very clear and slightly raised margins. The centre of the patches can be grey, scaly with broken hairs (non-inflammatory type), or contain crusts and pustules (inflammatory type). Very chronic deep infection (favus) can lead to permanent hair loss.

- \* Treat with griseofulvin (10 mg/kg/dose orally once daily for 2 weeks). Lesions usually heal spontaneously after months or years. Whitfield's ointment is not effective, but may perhaps reduce transmission.

#### *TINEA CORPORIS*

This is a superficial fungal infection of the body which starts as a small slightly itchy red papule that grows slowly with raised margins and contains small papules, pustules, and scaling. Healing is from the centre, giving the lesion the typical ring-like appearance. Lesions develop over weeks and months and can be secondarily infected after scratching.

- \* Treat with a twice daily sparing application of Whitfield's ointment for at least 4 weeks. This is usually effective, though slow, and relies on good compliance. Oral ketoconazole is an alternative, especially in the immunosuppressed, but avoid combining it with nevirapine, rifampicin, INH or artemether.

### *CANDIDIASIS (thrush)*

Thrush is an infection caused by yeasts, *Candida albicans*. Usually confined to the skin (giving a red moist and raw appearance), nails, mucous membranes (white dots or a thick white matting), and gastrointestinal tract, but can be systemic and spread to multiple deeper organs.

*Candida* is an opportunistic infection, predisposed to by moisture and maceration (e.g. in nappies), reduced immunity (e.g. malnutrition, HIV infection, severe systemic illness), antibiotics and steroid treatment.

Oral candidiasis is an infection of the mucous membranes of the mouth, and is very common in the newborn. It presents as discrete white patches that leave a red raw appearance if scraped off.

Angular cheilitis (perleche) presents as erythema, fissuring, maceration and soreness at the angles of the mouth.

Nappy rash presents with red, raw skin and there are "satellite" lesions that spread beyond the main rash.

- \* Treat skin, nail and mucous membranes with GV paint.
- \* Treat oral candidiasis with nystatin oral suspension.
- \* Reverse conditions that may have led to development of the infection (stop steroids, treat malnutrition, expose skin to the air).
- \* Griseofulvin has no effect and is strictly contraindicated but ketoconazole is an alternative, especially in the immunosuppressed, but avoid combining it with nevirapine, rifampicin, INH or artemether.

### 4.15.6 Viral Skin Infections

Many viruses cause rashes in children: measles, rubella, varicella, roseola infantum, erythema infectiosum, as well as echo and coxsackie viruses. Only those that cause skin problems are considered here.

#### *HERPES SIMPLEX*

About 90% of the population before 10 years of age are infected with this virus. The primary attack is often painful, but may also pass unnoticed. Recurrent attacks are less severe and occur at times of stress and reduced immunity (e.g. with meningitis or pneumonia). Lesions are on skin or mucous membranes of mouth, lips and genitals, and are preceded by an itching, burning sensation 1-2 days before. These start as a small red patch, and within hours develop into a small group of vesicles, that soon rupture forming crusts. They heal without scarring usually within a few days. Recurrences occur at the same site.



#### 4 Disease Management - Skin Diseases - Viral Skin Infections

- \* Aciclovir is effective, if started early, but very expensive and only of use if started in first 24 hours.
- \* Use zinc paste to dry lesions.
- \* Treat secondary infections with amoxicillin if necessary.

##### *HERPES ZOSTER (shingles)*

Caused by the reactivation of the chickenpox virus (*Varicella zoster*), which can remain dormant in the dorsal root ganglion of a sensory nerve in the spinal cord for life. Shingles is uncommon in children unless immunosuppressed. Presentation is characteristic, with pain over the dermatome for several days preceding the appearance of vesicles in the distribution of a sensory nerve (thus almost always unilateral). Children are less severely affected than adults.

- \* Aciclovir is useful if given from the first day, but expensive and not effective if started after the first 24 hours.
- \* Treat with calamine lotion in the acute phase (to dry lesions).
- \* Give adequate analgesia. Paracetamol is usually sufficient, but pethidine may be needed.

##### *WARTS*

These are very common in children, and can be of several types: flat skin-coloured lesions on the face; in-grown painful lesions on the soles; raised lesions on hands; or, large fleshy lesions around the anus. They cause few clinical problems, and disappear on their own.

- \* Treatment is not essential (95% get better on their own) and problems are mainly cosmetic. Warts often recur if treated.
- \* Freezing with liquid nitrogen is probably the most effective method of treatment, if available.
- \* Scraping and cautery is not effective, and surgical removal leaves scars which are often worse than the original warts.
- \* If there are genital warts, consider the possibility of sexual abuse and ask about the child's caretaker. Consider HIV testing.

##### *MOLLUSCUM CONTAGIOSUM*

This presents as small skin coloured raised round lesions, with a depressed centre (umbilication). They spread easily with scratching, but cause no clinical problems and disappear on their own. They may be profuse with immunosuppression.

- \* No treatment required. If persistent, curettage is effective.

##### *PITYRIASIS ROSEA*

The initial lesion is a small patch with fine scaling (the "herald patch"),

followed several days later by many more small lesions along the lower abdomen and on the upper limbs. There is a mild itch, and lesions disappear spontaneously within 6-8 weeks without scarring.

\* No treatment apart from calamine lotion is required.

#### 4.15.7 Eczema (dermatitis)

Eczema and dermatitis are considered together as inflammatory responses of the skin to many different kinds of exogenous and endogenous agents, though frequently the cause is unknown.

##### ***First try to establish stage of eczema***

- \* Acute eczema - a sudden eruption with erythema, vesicles and sometimes bullae, often with a serous exudate (wet appearance).
- \* Subacute eczema - lesions take several days to erupt, are red but not wet, and there are no vesicles or bullae.
- \* Chronic eczema - develops after months or years, and is characterised by thickened dry and scaly skin, with criss-cross lines (lichenification), deep cracks that can bleed, scratch marks, and sometimes infection.

##### ***Then try to determine type of eczema***

- \* Irritative contact eczema - the commonest variety, presenting mainly on the hands, due to the effect of certain chemicals on the skin, e.g. soaps, solvents, detergents.
- \* Allergic contact eczema - is a relatively rare allergic reaction to something in the diet or on the skin, after prior sensitisation (weeks/months/ or years before). It is important to try and find the cause (e.g. soaps, creams, and certain foods especially fruits and fish) as avoiding it is curative.
- \* Atopic eczema - a common eczema associated with asthma, hay fever and urticaria, which tends to run in families. In infants (under 18 months), there is weeping eczema, usually on the face, and spreading to the upper trunk and limbs. Patches dry up and become itchy and scaly. In children over 18 months only the creases of the knees, elbows, wrists and ankles tend to be involved. The itching is severe, and constant scratching leads to lichenification. 95% of all children with atopic eczema grow out of it, usually by 4 years of age.
- \* Seborrhoeic eczema - presents as greasy, scaly lesions on scalp, face, armpits, groin, and skinfolds, often with secondary bacterial infection or candidiasis. It may look similar to atopic eczema.

### Principles of eczema treatment

These principles are the same for all types of eczema.

- \* It is important to tell the parents not to expect total recovery, as most of the eczemas are likely to recur.
- \* Remove any obvious causes (allergic or contact eczema), though in most cases there are more than one cause. Ask about detergents, soaps, vaseline, cosmetic substances, etc.
- \* It is most important to avoid scratching which aggravates the condition. Cover itchy areas with dressings, and cut the nails in children.
- \* In the ACUTE STAGE use wet dressings with cooled boiled water for the first few days, then use calamine or phenol zinc lotion. Never apply any ointment to acute oozing eczema. Give antihistamines for itching which is usually severe in this stage.
- \* In the SUBACUTE STAGE use a zinc cream.
- \* In the CHRONIC STAGE use zinc paste with coal tar and an emulsifying ointment.
- \* For SEBORRHOEIC ECZEMA zinc cream with sulphur is helpful.
- \* For ATOPIC ECZEMA which is usually very dry use zinc paste with coal tar if there is lichenification of the skin. Otherwise use emulsifying ointment.
- \* For INFECTED ECZEMA give antibiotics systemically and topically (if available). Treat eczema as above.

#### 4.15.8 Skin Infestations

##### SCABIES

Scabies is the most common skin condition seen in out patients in Malawi. The cause is a mite (*Sarcoptes scabiei*), which is specific to man. The female lays eggs in tunnels in the skin, which hatch after 4-5 days and develop into new mites after 6-7 days. Infection is by direct contact from an infested person, or occasionally from clothes or bed linen. There is no immunity and reinfestation will occur after successful treatment.

Diagnosis is based on finding the mite burrows (look in the web spaces of fingers and toes, flexor aspects of wrists, buttock creases, around the umbilicus and genital area). Itch is the main symptom, and takes 2-4 weeks to become severe. Reinfestation results in immediate appearance of the lesions with itch, as sensitisation has already occurred. There can also be urticarial reactions with papules, vesicles, crusts and, if

secondarily infected, pustules.

- \* Apply benzyl benzoate (BB) paint 25%, 20 ml if < 5 years, and 50 ml if > 5 years. In small infants dilute to 12.5% (adding 10 ml of tap water to 10 ml of 25% BB paint gives 20 ml of 12.5% BB paint solution, enough for the treatment of one child. Alternatively Lindane can be used.
- \* Make sure that the whole family are treated as the asymptomatic ones are probably "incubating" the infection.
- \* With every new family being treated, give these instructions:
  - \* In the evening take a bath or wash the whole body. Then apply BB paint from neck down covering the whole body, but including the scalp and face in infants.
  - \* 10 hours later (i.e. next morning), wash all the paint off with soap and water.
  - \* wash clothes and linen before using them again.
  - \* repeat the treatment 5 days later.
- \* Itching may continue for 3-4 weeks, so wait that long before giving another treatment. Too many applications will irritate the skin and make the itching worse.
- \* Secondary infection with streptococci is common, and can lead to glomerulonephritis [4.11.2]. Treat with amoxicillin.

#### *PEDICULOSIS CORPORIS*

Body lice are common in Malawi. The lice spend most time in clothes and only attack the body to feed, leaving an itchy insect bite on parts of the body covered by clothes. Look for the lice and their eggs (nits) in the clothes and not the skin.

- \* Advise the guardian to wash all clothes with soap and hot water, and then to iron thoroughly, especially the seams.

#### *PEDICULOSIS CAPITIS*

Head lice are rarer, occur in epidemics in children, and are transmitted by direct contact, combs, brushes and hats. Infection presents as severe itch on the neck and behind the ears, often with secondary infection. Diagnosis is by finding the eggs (nits) cemented to the hair and impossible to remove.

- \* Benzyl benzoate paint (to be used as with scabies) kills the nits and shaving the hair helps.
- \* Alternatively rub Malathion 0.5% or Lindane 1% into the hair and leave for 2 hours before washing and combing. Repeat after 2 weeks.

### *ONCHOCERCIASIS*

A filarial disease transmitted by a small black fly, found living by fast flowing streams, mainly in the Thyolo district. The incubation period is about a year. It presents with itching papules, and later, nodules, usually in one or both legs. Chronic infection causes patchy depigmentation, thickening, and loose inelastic skin, the so called "leopard skin." The main diagnostic clue is the patient's district, as the disease is mainly in Thyolo. Diagnose by seeing microfilariae in a skin snip under the microscope.

- \* Treat with ivermectin (not to children under 5). The dose is 150 micrograms/kg orally as a single dose.

### *LARVA MIGRANS (creeping eruption)*

The cause is the larvae of the dog and cat hookworm (man is not the normal host). The larvae penetrate the skin and create twisting channels in the skin, seen commonly on the feet and buttocks.

- \* Treat with albendazole 400 mg as a single dose (200 mg < 2 years).

### *TUNGA PENETRANS (jiggers)*

Caused by the sand flea, and thus prevalent in sandy areas. The fertilised female penetrates small cracks in the skin usually of the toe webs, and remains in the outermost layer (stratum corneum), where it forms an itchy painful swelling (jigger) after 8-10 days. Scratching helps to burst the jigger liberating the eggs.

- \* Remove the mature female with a sharp needle, taking care not to burst the jigger.
- \* Give a booster dose of tetanus toxoid to prevent tetanus.

### *TUMBU FLY (myasis)*

Female flies lay eggs in sand or drying clothes. The eggs hatch into larvae that penetrate the skin without causing symptoms. Over the following days they grow and form an itchy boil with a characteristic black spot on the top - this is the hole through which they breathe.

- \* Apply vaseline or oil to the boils - this stops the oxygen supply to the larvae, which then try to wriggle out after a few minutes. They can then be removed with forceps.

## 4.15.9 Skin Reactions

### *URTICARIA*

Is the name given to many types of allergic reactions, where the characteristic lesion is the "wheal" (raised skin due to fluid that has

leaked from capillaries as a result of an allergic response). Lesions can either be acute with a single attack lasting for a few days, or chronic with lesions reappearing for more than 6 weeks.

- \* Try to identify and eliminate the cause if possible. Often no cause is found.
- \* Treat itch with calamine lotion and antihistamine.
- \* In severe cases give a course of systemic steroids.

#### *PAPULAR URTICARIA*

An itchy reaction to insect bites or bed bugs, which starts as a wheal, and becomes papular, often with a small vesicle on the top, and bullae on the legs. Secondary infection is common.

- \* Calamine lotion and antihistamines.
- \* Treat secondary infections.

#### *ERYTHEMA MULTIFORME*

A common rash seen in children. It may be due to viruses, but most are unknown cause. Most cases are mild presenting as red/purple papules with central blisters (the so called "target lesions"). The severe type of erythema multiforme is known as Stevens-Johnson syndrome and is usually due to drug reactions (especially sulphonamides, including SP, thiacetazone, an old anti-TB drug (so now very unlikely to be seen) and nevirapine). It involves the mucous membranes and conjunctiva, and cause problems of fluid loss and secondary infection.

- \* Mild cases need only calamine lotion and antihistamines.
- \* Stevens-Johnson syndrome requires hospitalisation, antibiotics, intravenous fluids and correction of electrolyte imbalances. If very severe give steroids, though get advice first.

## 4.16 Blood Diseases

### 4.16.1 Anaemia

See section 3.9

### 4.16.2 Sickle Cell Disease

This is an autosomal recessive inherited disorder (both parents are carriers of the disease). Fetal Hb is normal and persists longer than normal so symptoms seldom start before 6 months old. Common early symptoms from 6-24 months are recurrent multiple painful swellings of the bones of the wrist, hand and foot (hand-foot syndrome), due to bone

infarction from sickled red cells blocking blood vessels. Other features include anaemia, jaundice, pains in limbs or abdomen from infarcts and skull bossing. The spleen enlarges at first, but by 6 years of age has usually shrunk from infarcts. Problems may result from repeated blood transfusions (transfusion reactions, hepatitis and AIDS), susceptibility to infections especially pneumococcal and gram negative bacterial (e.g. salmonellae). Osteomyelitis is quite a common complication, and very difficult to differentiate from aseptic necrosis of bone due to bone infarction. Priapism (penile erection) and aseptic necrosis of the head of the femur, strokes, renal infarcts and acute chest syndromes can occur.

Various "crises" can occur in sicklers: painful crises are caused by infarcts in bone or abdominal organs; aplastic crises are due to cessation of bone marrow function caused by parvovirus; sequestration crises occur in younger children with blood collecting in the liver and spleen; haemolytic crises are due to rapid haemolysis caused by malaria.

- \* As with all chronic conditions, it is important to spend time with the parents, preferably together initially, explaining the genetic cause and nature of the disease, the risk of recurrence, the problems to be expected and the management.
- \* Give proguanil once daily to prevent malaria, preferably with chloroquine once weekly, and explain why you give it.
- \* Supply an antimalarial, in case malaria occurs in spite of prophylaxis.
- \* Give folic acid 2.5-5 mg daily to prevent deficiency arising.
- \* Determine the child's blood group, in case transfusion is needed.
- \* Teach the parents that gastroenteritis and respiratory infections need to be treated early, and instruct them how to use ORS and how to recognise lower respiratory infections.
- \* Review the child regularly and check Hb and MPS. Initially a monthly check is appropriate. When the child is over about 6 years old, after which many sicklers are fairly stable, a check every two to three months, or when the child seems ill, may be frequent enough.
- \* Avoid blood transfusion, both to prevent iron overload and to reduce the risks of infection with hepatitis B and HIV, unless the Hb falls below 5 g/dl. The child becomes accustomed to living with a Hb of about 6-8 g/dl.
- \* Record growth in weight regularly.
- \* Ensure pentavalent vaccine given to prevent hepatitis B, measles.
- \* Sicklers whose spleen has infarcted are liable to pneumococcal

infections, so be alert to prescribe amoxicillin early if there is cough with fever. Policy may be to give monthly benzathine penicillin (as after splenectomy) to prevent pneumococcal infections, but if available and affordable pneumococcal immunisation is better.

**Management of crises:**

- \* Treat painful crises with analgesics, usually paracetamol, but oral morphine or pethidine IM may be needed, though avoid frequent repeated doses, because of the risk of addiction. In severe persistent cases give a blood transfusion.
- \* Aplastic crises are temporary, due to parvovirus infection, and do not recur, but lead to severe anaemia requiring blood transfusion.
- \* Sequestration crises can lead to shock [3.3], so give IV saline 20 ml/kg to restore blood pressure.
- \* Haemolytic crises may need transfusion. Treat malaria.

#### 4.16.3 Haemophilia

A sex-linked recessive hereditary defect of blood coagulation almost totally confined to boys. New cases may arise by mutation, but often there is a family history of a bleeding tendency in males on the mother's side. The mother may pass on the disease to half her sons and the carrier state to half her daughters. Bleeding may occur soon after birth (e.g. from the cord stump or after circumcision) but is often delayed until the boy becomes mobile and starts to fall, which causes bruising and muscle haematomas. There may be prolonged bleeding from minor cuts, bleeding from the gums, bleeding into joints (often the elbows in children not yet crawling, and knees in those walking). There may also bleed from tooth sockets after teeth are lost naturally, by accident or by dental removal. Severity varies, but most haemophiliacs in Malawi seem to be mild.

Diagnosis rests on finding a normal platelet count, bleeding, clotting and prothrombin times but a prolonged partial thromboplastin time, which returns to normal after cryoprecipitate (Factor VIII, lacking in haemophilia, is now available in this form through the Malawi Blood Transfusion Service [5.9], but may later come as concentrate). Christmas disease is rarer, similarly inherited, and corrected by factor IX, not VIII.

- \* Haemophiliacs are liable to hepatitis B (so ensure pentavalent vaccine given) and AIDS from blood transfusions.
- \* Educate parents about the disease covering points such as risk to future offspring and that bleeding (which can be either internal or



external) can only be controlled by factor VIII, given in hospital under specialist supervision. The child should avoid IM injections, operations (without first receiving factor VIII), contact sports (including football), and should be given paracetamol, not aspirin, because of the risk of gastrointestinal bleeding.

- \* Record in every haemophiliac's health passport his diagnosis and blood group. Write warnings that surgery should only be done in a central hospital with factor VIII available, and that IM injections and aspirin must be avoided. Record all blood and factor VIII transfusions.
- \* Encourage education to increase chance of a non-manual job.
- \* If child is bleeding or has a painful joint, give factor VIII fast IV, repeated several times until healing has occurred at the site of bleeding, (expensive, but saving cost in the long term).
- \* Immobilise injured joints during healing with plaster of Paris splints.

#### 4.16.4 Idiopathic Thrombocytopaenic Purpura (ITP)

Idiopathic thrombocytopaenic purpura (ITP) is an acute acquired illness, which follows a minor viral infection, presenting with general purpuric rash and prolonged bleeding from minor cuts. There is a low platelet count but normal red and white cells. The bleeding time is prolonged, but clotting and prothrombin times are normal. The differential includes haemolytic-uraemic syndrome [4.11.6], leukaemia [4.17.5] and meningococcal septicaemia [4.12.1].

- \* Usually no treatment is needed, but if platelets drop below 20,000/c.mm. give prednisolone (2 mg/kg/dose once daily) for 1 week, as there is danger of intracranial bleeding.

#### 4.16.5 Henoch-Schonlein Purpura

A vasculitis, of uncertain, probably immune cause, presenting with erythematous macules or papules, which often become purpuric, on the anterior lower legs and buttocks. There may be arthritis of larger joints, abdominal pain, intestinal bleeding, and nephritis with haematuria. Platelets are normal unlike, in haemolytic-uraemic syndrome [4.11.6]. It usually resolves spontaneously, but occasionally there is persistent renal damage. Intussusception is an important differential diagnosis, but can also occur in Henoch-Schoenlein purpura.

- \* No specific treatment is available.
- \* Give paracetamol for arthritis.

- \* Check urine and BP, and treat nephritis [4.11.2] and hypertension [4.9.4] if present.

## 4.17 Malignancies

### 4.17.1 Burkitt's Lymphoma

Burkitt's lymphoma is the most common malignant tumour seen in Malawian children. It most often affects one or more quadrants of the jaws, giving a rapidly increasing, non-tender and non-painful swelling involving the bones, and with often gross distortion of appearance and loosening of teeth. Other common sites for the tumour are in the abdomen, the orbits (proptosis), and the spine, leading to paraplegia. It may also involve the scalp, bones, lymph nodes, testes, brain and thyroid.

- \* Refer patients to a central hospital for initial assessment and treatment. Treatment requires repeated doses of cytotoxics (mainly cyclophosphamide, perhaps methotrexate and vincristine) over some months, ideally with intrathecal hydrocortisone and methotrexate.
- \* For patients with abdominal or widespread tumours give oral allopurinol for 24 hours before and 4 days after the first dose of cyclophosphamide to prevent tumour lysis syndrome.
- \* Give a rapid IV bolus of cyclophosphamide, 40 mg/kg/dose into a drip, after some 20 ml/kg of normal saline. This ensures a high urinary output, to prevent haemorrhagic cystitis. IV methotrexate and vincristine may be given with IV cyclophosphamide. Currently 6 treatments are given 2 weeks apart. Oral cyclophosphamide is under trial in Blantyre, first at the above dose, then 60 mg/kg/dose for 4 doses 10 days apart, along with intrathecal hydrocortisone and methotrexate, which is to prevent lymphoma spreading to the CNS.
- \* Check Hb, WBC and platelets before each treatment, as cytotoxic therapy suppresses bone marrow, causing anaemia, infective and bleeding complications.
- \* The prognosis is much better when the tumour is confined to one maxilla or lower jaw or even two such sites, than if it is in the abdomen, vertebral canal or elsewhere. Cyclophosphamide alone may cure single jaw tumours, but fewer patients with widespread tumour will survive without combined drug treatment. (Combined treatment will include vincristine and methotrexate as well as cyclophosphamide).

#### 4.17.2 Kaposi's Sarcoma

Since the coming of AIDS this cancer, from being rare has become the second most common malignancy in children in Malawi. It may present as dark red, purple or black lesions of skin, mouth or eye, lymphadenopathy or disease in the chest (breathlessness, cough, haemoptysis) or intestine (bleeding, intussusception). Biopsy of lymph nodes or skin lesions may be needed for diagnosis. HIV infection is almost always present. Chest Xray may show enlarged hilar nodes, pleural effusions, pericardial effusion (best seen by ultrasound) or bilateral hilar infiltrates.

Treatment with vincristine may help, as may ART. Paracetamol and morphine may be needed for palliation of pain.

#### 4.17.3 Retinoblastoma

This is a common unilateral or bilateral malignant tumour of the retina, which spreads down the optic nerve, and may invade the tissues of the orbit causing proptosis. The most common presentation is of an abnormal whitish-yellow opacity appearing in the pupil of the eye ("white reflex").

- \* The treatment in Malawi for retinoblastoma is surgical removal of the affected eye, but carefully rule out Burkitt's lymphoma first.
- \* Without radiotherapy, antitumour drugs alone are unlikely to give cure, and it is probably kinder not to start treating retinoblastoma with cyclophosphamide and vincristine when it has spread out of the orbit.
- \* Relieve severe pain with pethidine if necessary.
- \* Be sure to get histological diagnosis on any eye that is removed, in case it recurs in the other eye, and hard decisions are faced.

#### 4.17.4 Wilm's Tumour

Wilm's tumour is an embryonal tumour of the kidney, which may be unilateral or bilateral. The most common presentation is an abdominal mass. The differential includes Burkitt's lymphoma and neuroblastoma. Ultrasound can give the diagnosis without biopsy in skilled hands.

- \* Refer to Blantyre for several doses of vincristine and actinomycin D (if available) to shrink the tumour, before surgical removal of the kidney and tumour, followed by more chemotherapy. Cure is possible, but in Malawi the tumour usually presents very late so may not be resectable at laparotomy. Treatment is thus often purely palliative i.e. relief of pain with analgesics.

#### 4.17.5 Leukaemia

Leukaemia is a malignant condition of one or other line of the white blood cells. Most common in childhood is acute lymphoblastic leukaemia. The presentation is usually some combination of anaemia, reduced resistance to infections and bleeding.

- \* Acute leukaemias are seldom effectively treated in Malawi. When the patient is a "low risk case" with normal total WBC and no abnormal cells in the peripheral blood, no hepatosplenomegaly or adenopathy, and with parents who understand and are in a position to co-operate with treatment for at least 2 years, remission induction with vincristine and prednisolone may be considered, followed by regular doses of oral methotrexate and 6 mercaptopurine. This requires prolonged attendance at a central hospital. Eventual results are likely to be disappointing.
- \* Chronic leukaemias are very rare, but if found can be treated as in adults.

#### 4.17.6 Histiocytosis X

This is an aggressive malignancy especially in infants, presenting with hepatosplenomegaly, eczematous rash, lymphadenopathy, punched out holes in the skull, fever, and often secondary infections. Young children may have proptosis or pituitary damage from lesions round the skull, while older children may have rather benign isolated eosinophilic granulomata in bones with pain and sometimes pathological fracture.

- \* This disease may respond well to vincristine or cyclophosphamide, but in smaller children may often follow a rapid downhill path to death. Localised bone lesions can be excised.

### 4.18 Poisoning

#### 4.18.1 General Principles of Treatment

- \* Ask: - WHAT was taken
  - WHEN it was taken
  - HOW MUCH was taken
- \* Clear the airway and maintain ventilation.
- \* Treat shock by elevating the legs, usually IV fluids, perhaps a vasopressor drug.
- \* Empty the stomach if seen within 4 hours of poisoning, and the

#### 4 Disease Management - Poisoning - Termic, Paraffin, Iron

patient is conscious, **but do not empty** if corrosive substance (strong acid/alkali) or paraffin (unless you first pass an endotracheal tube) was ingested, or if there is coma

##### METHOD OF EMPTYING STOMACH

##### \* STOMACH WASH-OUT

- Lie the child head down on the left side, and pass a wide gauge soft rubber tube (Ryle's tube) into the stomach, then pour 100-200 mls tap water down the tube, and aspirate, with the patient in the head down position, carefully protecting the airway during the procedure.

\* Treat as necessary hypothermia, hypoglycaemia, convulsions, electrolyte or acid/base disturbance.

#### 4.18.2 Termic (carbamate) and Organophosphate Poisoning

\* Presents with anxiety, restlessness, small pupils, increased secretions, (moist noises in chest) and bradycardia.

\* Empty the stomach if poison was swallowed, or remove clothes and wash skin if poisoning was from skin contact.

\* Maintain the airway.

\* Give atropine 20 microgm/kg/dose every 5-10 minutes until the skin feels and chest sounds dry and there is tachycardia. Pupils dilate later.

#### 4.18.3 Paraffin Poisoning

\* Do not make the child vomit, because of the risks of lipid pneumonitis, which is the main complication if paraffin is inhaled.

\* If the paraffin dose was high (> 10 ml/kg) a stomach wash out only after endotracheal intubation under anaesthesia may be done, as high doses may lead to brain damage.

\* With lower doses give oxygen if necessary.

\* Unabsorbable lipid (seldom available), like medicinal liquid paraffin, given orally is valuable in reducing the absorption of paraffin.

#### 4.18.4 Iron Poisoning

\* A dose of 20 ml/kg of syrup, or 2-3 tablets/kg may be fatal. Abdominal Xray may show how many tablets were swallowed.

\* In severe cases (over 10 tablets ferrous sulphate) there may be vomiting and gut haemorrhage in the acute stage and liver necrosis and shock after 1-2 days. Therefore observe the child for at least 2 days.

- \* Iron tablets should be washed out by gastric lavage, and absorption can be reduced by giving magnesium sulphate, and desferrioxamine (if available) down the nasogastric tube thereafter.

#### 4.18.5 Aspirin Poisoning

- \* ALWAYS EMPTY THE STOMACH as stomach emptying is delayed.
- \* In severe cases IV half Darrow's dextrose with added sodium bicarbonate (30 mmol/l) to increase renal excretion may be needed.
- \* Hypoglycaemia, hypokalaemia, convulsions and metabolic acidosis should be watched for and treated.

### 4.19 Bites

#### 4.19.1 Dog Bite

- \* Fresh dog bites should always be washed well with soap and water or detergent, and should never be sutured.
- \* Tetanus toxoid 0.5 ml IM should be given, and additionally ATS 1500 units IM if the child did not receive pentavalent (or DPT) vaccine.
- \* Antibiotics, such as benzyl penicillin, are advisable in serious bites.
- \* The primary concern is usually whether the dog was rabid. The table below may help you decide the best action to take.
- \* Antirabies vaccine is held at district health office pharmacies.

<u>Nature of exposure</u>	<u>Condition of animal</u>		<u>ACTION</u>
	<u>At first</u>	<u>After 10 days</u>	
Saliva on intact skin	well or not well	rabid or dead	No ARV
Saliva on injured skin and minor bites of limbs or trunk	well well suspect	well rabid/ dead well	No ARV Full course ARV Start ARV, stop 10 days
Saliva on mucosae,	suspect well	rabid/ dead well	Full course ARV Start ARV, stop 10 days
major bites and bites of head and neck	suspect	rabid/dead	Antirabies serum & full course of ARV

#### 4 Disease Management - Bites - Dog Bite, Snake Bite

If the dog is known and under observation, or has been killed and the brain is being submitted for veterinary examination the above recommendations can be followed. Often the dog is unknown and then it must be assumed to be rabid.

- \* If the wound is severe, or affects the head or neck, special antirabies serum is needed as well as ARV.
- \* The modern rabies vaccine is given (1 ml IM) into the deltoid muscle (NOT into the buttock) on days 0, 3, 7, 14, 30 and 90. In view of the expense of the vaccine, when more than one person is being immunised at a time it is good to give instead 0.1 ml intradermally in two sites on days 0 and 3, and into one site on the later days, thus allowing 1 ampoule to be divided amongst about 4 people.

#### 4.19.2 Snake Bite

Effects of snakebite depend on the type of snake and how much venom is injected. Often patient anxiety is important. Tetanus should be prevented.

SNAKE	VENOM TYPE	CLINICAL EFFECTS
Vipers (Puff adder, night adder gaboon viper)	Necrotising occasional bleeding, cardiotoxic	slowly developing oedema, blistering, tissue necrosis, hypotension, arrhythmias, shock
SNAKE Cobras and mambas	Neurotoxic	rapid weakness of eye, swallowing, speech, respiratory muscles, excessive salivation.
Spitting cobra	Necrotising	tissue destruction, eye inflammation
Vine snake, boomslang	Anticoagulant	slow development of generalised bleeding tendency

#### First Aid

- \* DO NOT suck out wound, incise, give alcohol or aspirin.
- \* Reassure, give paracetamol, splint limb, transport to hospital lying on side in case of vomiting, and take dead snake if available.

- \* Only if cobra or mamba bite apply broad bandage as firm (not tight) tourniquet, above bite.
- \* If spitting cobra, wash out eye with water immediately.

### **Hospital Treatment**

- \* History - whether really bitten, what snake.
- \* Look for swelling, drooping eyelids, bleeding gums, hypotension.
- \* If no ill effects after 24 hours, discharge.
- \* If systemic effects occur, or significant limb swelling, give antivenin, but not for vine snake or boomslang bites, as ineffective for them.
- \* Children need the same dose of antivenin as adults - up to 8 vials (80 ml) SAIMR Polyvalent Antivenin for vipers, and up to 10 vials (100 ml) for cobras and mambas. Snake antivenin is not at present available in government hospitals.
- \* Give antivenin IV as follows<sup>1</sup>
  - \* Put dose in 200 ml normal saline.
  - \* Draw up 1/1000 adrenaline injection in a syringe in a dose appropriate to child (0.01 ml/kg).
  - \* Start drip slowly, observing for allergic reaction in first 20 minutes (itching, urticaria, fever, tachycardia, cough or wheeze). If reaction occurs, stop drip and give adrenaline subcutaneously. The reaction should reverse in 5-10 minutes, when the drip can be restarted slowly, with another dose of adrenaline ready.
  - \* Complete the antivenin within 1 hour.

### **Other Treatment for Bites**

#### ***Viper bites***

- \* Elevate bitten limb, give benzyl penicillin if there is swelling, give tetanus toxoid 0.5 ml IM and, if child has not had pentavalent (or DPT) immunisation, ATS 1500 units IM.

#### ***Cobra or mamba bites***

- \* If respiratory paralysis occurs, intubate, ventilate and refer to central hospital. Give anticholinesterase drugs like neostigmine, if available.

#### ***Spitting cobra injury to the eye***

- \* After copious washing, apply antibiotic eye ointment and pad the eye.

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<sup>1</sup> Late reactions with fever and arthralgia (serum sickness) may occur after 10 days and are managed with prednisolone 2 mg/kg/dose once daily for 5 days.



### Vine snake or boomslang

- \* If bleeding occurs, transfuse fresh screened blood.

#### 4.19.3 Bee Stings

These can cause shock either from many stings or anaphylaxis to just one.

- \* Assess for shock [3.3]
- \* If shock is found, give 0.01 ml/kg 1 in 1000 adrenaline (0.01 ml is 10 mcg) IM stat (maximum 0.3 ml). Check heart rate, BP, urine output.
- \* If shock persists, give IV adrenaline diluted in normal saline or 5% dextrose drip, at 0.1 mcg/kg/minute, increased slowly up to 1.0 mcg/kg/minute, according to BP and heart rate response preferably in an ICU situation.
- \* Remove all bee stings by scraping off the skin with a knife.

## 4.20 Surgical Problems

### 4.20.1 Burns

The medical aspects of burns management primarily concern fluid loss and infection. In the initial period the primary concern is to replace fluid loss from the burned surface rapidly to prevent shock.

- \* ASSESS the severity of the burn with the scheme below:<sup>1</sup>

<u>Part of body burned</u>	<u>% Surface area of body</u>
Head	18
Neck	2
Arms (each)	9
Chest and abdomen	18
Back	18
Buttocks	3
Perineum	1
Legs (each)	9
Feet	4

- \* ADMIT if > 5% body surface burn.

### Fluids

- \* MILD BURNS (< 10% of the body surface burn) give oral fluid therapy with as much ORS as the child will tolerate.

---

<sup>1</sup> The relative proportions of children's body surface area changes as they grow, so this scheme is only a very rough guide for young children.

\* MODERATE TO SEVERE BURNS (> 10%, OR > 5% in babies)

Give IV fluids  $2 \text{ ml/kg} \times \% \text{ surface burned} + \text{normal maintenance needs}$ .<sup>1</sup> Give half in the first 8 hrs, and the rest within the next 16 hrs. Use Ringer's lactate or normal saline, adding 100 ml 50% dextrose to a 1 litre bag. Add colloid solution, e.g. haemacel, (if available)  $3.5 \text{ ml/kg} \times \% \text{ burned}$  for plasma expansion. Avoid giving extra potassium.

**Remember that younger children need more vigorous fluid therapy.**

**Relief of pain**

- \* Use pethidine IM in the early stages, changing to paracetamol after 48 hours.

**Infection**

- \* Prophylactic antibiotics for infection are not recommended, but fever is an indication to start antibiotics after taking a wound swab for culture. In the early phase give benzyl penicillin, combining with gentamicin if 48 hours have elapsed since the burn.
- \* Give tetanus immunisation.

**Other points**

Remember to

- \* Weigh the patient on admission.
- \* Check measles immunisation.
- \* Check the Hb and PCV regularly.
- \* Start physiotherapy early (splints & exercises) when skin near joints (especially the hands) is burned.
- \* Pay attention to nutrition in the later course of management of the burned patient.

#### 4.20.2 Acute Abdomen

Features such as pain, tenderness, guarding, abdominal distension, vomiting and constipation are found in patients with illnesses like acute appendicitis, typhoid perforation, and intestinal obstruction caused by hernias, adhesions, volvulus and intussusception, which need surgery. But it must be remembered that medical diseases may present with these symptoms. Abdominal distension (from air swallowing), vomiting and restlessness, suggesting pain, can occur with pneumonia [4.8.14].

<sup>1</sup> If in doubt about calculation treat as for severe dehydration [4.10.1].

Abdominal pain and tenderness, but usually not guarding may be found in gastroenteritis [4.10.2]. Sickle cell disease [4.16.1] and diabetes [4.14.1] can mimic an acute abdomen and need appropriate treatment.

\* Control IV fluids carefully in patients on prolonged drip and suction, to maintain basal needs and give extra to cover losses through vomiting, nasogastric aspiration, diarrhoea, loss of fluid into the intestines in ileus, and extra fluid losses from fever. In general half Darrow's dextrose is the best fluid to use.

**Give glucose during prolonged drip and suction in the infusion fluid to prevent hypoglycaemia. Either use half strength Darrow's dextrose, or add 100 ml 50% glucose to a litre of Ringer's lactate.**

\* Antibiotics may be needed. Combined benzyl penicillin and chloramphenicol is often appropriate, but when bowel has perforated add IV metronidazole 7.5 mg/kg/dose 8 hrly.

#### 4.20.3 Head Injury

\* Keeping a clear airway is essential in all patients with head injuries, when consciousness is depressed.

\* Closed head injuries require careful observation of coma score, pupil size and reaction, pulse rate, BP, and for any convulsions or vomiting.

\* Pethidine should be avoided for pain, paracetamol being preferable, and convulsions are managed with paraldehyde and phenobarbitone (or phenytoin, if oral treatment is possible).

\* Do not give excess fluids, as the patient with a head injury may secrete extra antidiuretic hormone, leading to cerebral oedema.

\* When consciousness deteriorates get expert opinion to decide if intracranial haemorrhage is occurring, and whether it is treatable surgically.

\* When cerebral oedema is suspected, high dose steroids may reduce intracranial pressure temporarily.

\* In compound fractures (externally to the scalp, or internally into the nose or ear) do careful wound debridement and give antibiotics<sup>1</sup>. Give tetanus toxoid 0.5 ml IM, and in addition ATS 1500 units IM in children who have not had pentavalent (or DPT) immunisation.

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<sup>1</sup> Chloramphenicol, unlike benzyl penicillin, penetrates well into the CSF through uninflamed meninges, and is the antibiotic of choice.

#### 4.21 Defilement

Sexual abuse of a child is a serious matter, but since the coming of HIV infection it is potentially life threatening. Equally allegations of sexual abuse have grave consequences for alleged perpetrators. It is therefore important for a senior staff member to take a history and examine the patient, and for a medical or nursing witness to be present. This should be done in privacy. A careful record must be kept of the date and time and the personnel involved. A guardian must also be present and a signed consent obtained if at all possible.

In the history it is important to find out and record the dates and times of alleged episodes, and such details as condom use, subsequent pain, bleeding or discharge, previous and subsequent sexual activity of the patient, any general or local symptoms, past history of blood transfusion, any medications and the family and social background.

Apart from weight and full general examination, careful examination in a good light needs to be made of the genitalia. Normal findings do not exclude abuse.

**A careful record must be kept of who was present, the position and qualifications of the person examining the child, the nature of the allegations, the significant findings, the results of any investigations and the treatment given.**

- \* Tests available to you will probably only be microscopy for sperms and a Gram stain on any discharge and HIV and possibly VDRL testing.
- \* Admit if the child needs surgery or protection from others in the home.
- \* Give all defiled children erythromycin for 7 days and stat benzathine penicillin and gentamicin IM.
- \* Offer post exposure prophylaxis (PEP) for HIV to all defiled children.<sup>1</sup> This requires pre- and post-counselling of guardian and child.
- \* Note children are very liable to anaemia on zidovudine, so follow up for that as well as other drug toxicity.
- \* Only give PEP if:
  - an HIV test is done and is negative.
  - any defilement occurred less than 72 hours before.
  - there are physical signs of trauma or penetration (vaginal or anal).
  - the guardian and child agree to comply with treatment.

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<sup>1</sup> It is very unlikely that the alleged perpetrator will be known to be HIV negative for certain.

#### 4 Disease Management - Defilement

- \* If HIV testing is not immediately available, PEP may be started, but an HIV test must be done within 72 hours. Stop PEP if it is positive.
- \* Post-counsel and follow up any child who is HIV positive.

PEP consists of lamivudine 150 mg with zidovudine 300 mg. Give it for 30 days. Follow up during treatment and then at 1, 3 and 6 months and repeat HIV tests at those return visits. Treatment is given orally b.d. and the dosage is by weight:

<u>Weight</u>	<u>Dose of PEP</u>
< 14 kg	$\frac{1}{4}$ tablet b.d.
15 - 24 kg	$\frac{1}{2}$ tablet b.d.
25 - 34 kg	$\frac{3}{4}$ tablet b.d.
> 35 kg	1 tablet b.d.

# 5 APPENDICES

*5 Appendices - Height & Weight Charts*

**5.1 Height and Weight Charts**

**5.1.1 The Health Passport Weight Chart**

### 5.1.2 Standard Infant Size at Birth<sup>1</sup>

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<sup>1</sup> Based on data from the University of Colorado Health Sciences Center, 1974-80.



*5 Appendices - Height & Weight Charts*

**5.1.3 2-18 Years Height and Weight for Boys<sup>1</sup>**

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<sup>1</sup> Adapted from National Center for Health Research data NCHS Growth Charts, 1976.

### 5.1.4 2-18 Years Height and Weight for Girls<sup>1</sup>

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<sup>1</sup> Adapted from National Center for Health Research data NCHS Growth Charts, 1976.

5 Appendices - Height & Weight Charts

5.1.5 Head Circumferences 0-36 Months, Boys and Girls

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<sup>1</sup> Adapted from National Center for Health Research data NCHS Growth Charts,

## 5.1.6 Weight for Height Table

<u>Length</u> <u>(lying down)</u>	<u>Median</u> <u>weight</u>	<u>80%</u> <u>of median</u>	<u>70%</u> <u>of median</u>
50 cm	3.4 kg	2.7 kg	2.4 kg
51	3.5	2.8	2.5
52	3.7	3.0	2.6
53	3.9	3.1	2.7
54	4.1	3.3	2.9
55	4.3	3.5	3.0
56	4.6	3.6	3.2
57	4.8	3.8	3.4
58	5.1	4.0	3.5
59	5.3	4.3	3.7
60	5.6	4.5	3.9
61	5.9	4.7	4.1
62	6.2	4.9	4.3
63	6.5	5.2	4.5
64	6.7	5.4	4.7
65	7.0	5.6	4.9
66	7.3	5.8	5.1
67	7.6	6.1	5.3
68	7.0	6.3	5.5
69	8.2	6.6	5.7
70	8,5	6.8	5.9
71	8.7	7.0	6.1
72	9.0	7.2	6.3
73	9.2	7.4	6.5
74	9.5	7.6	6.6
75	9.7	7.8	6.8
76	9.9	7.9	6.9
77	10.1	8.1	7.1
78	10.4	8.3	7.2
79	10.6	8.4	7.4
80	10.8	8.6	7.5
81	11.0	8.8	7.7
82	11.2	8.9	7.8
83	11.4	9.1	7.9
84	11.5	9.2	8.1

5 Appendices - Height & Weight Charts

Weight for Height Table (continued)

<u>Height</u> <u>(standing)</u>	<u>Median</u> <u>weight</u>	<u>80%</u> <u>median</u>	<u>70%</u> <u>median</u>
85 cm	12.0 kg	9.6 kg	8.4 kg
86	12.2	9.8	8.5
87	12.4	9.9	8.7
88	12.6	10.1	8.8
89	12.9	10.3	9.0
90	13.1	10.5	9.2
91	13.3	10.7	9.3
92	13.6	10.8	9.5
93	13.8	11.0	9.7
94	14.0	11.2	9.8
95	14.3	11.4	10.0
96	14.5	11.6	10.2
97	14.8	11.8	10.3
98	15.0	12.0	10.5
99	15.3	12.2	10.7
100	15.6	12.4	10.9
101	15.8	12.7	11.1
102	16.1	12.9	11.3
103	16.4	13.1	11,5
104	16.7	13.3	11.7
105	16.9	13.6	11,9
106	17.2	13.8	12.1
107	17,5	14.0	12.3
108	17.8	14.3	12.5
109	18.1	14.5	12.7
110	18.4	14.8	12.9
111	18.8	15.0	13.1
112	19.1	15.3	13.4
113	19.4	15.5	13.6
114	19.8	15.8	13.8
115	20.1	16.1	14.1
116	20.5	16.4	14.3
117	20.8	16.7	14.6
118	21.2	17.0	14.9

## Weight for Height Table (continued)

<u>Height</u> <u>(standing)</u>	<u>Median</u> <u>weight</u>	<u>80%</u> <u>median</u>	<u>70%</u> <u>median</u>
119 cm	21.6 kg	17.3 kg	15.1 kg
120	22.0	17.6	15.4
121	22.4	17.9	15.7
122	22.8	18,3	16.0
123	23.3	18.6	16.3
124	23.7	19.0	16.6
125	24.2	19.4	16.9
126	24.7	19.7	17.3
127	25.2	20.1	17.6
128	25.7	20.6	18.0
129	26.2	21.0	18.4
130	26.8	21.4	18.7

## 5.2 Immunisation

### 5.2.1 Introduction

**Active** Stimulates the body to produce its own antibodies. Immunity is slow to develop but is long-lasting, e.g. diphtheria, pertussis, tetanus, polio, measles, mumps, pneumococci, rotavirus, rubella, hepatitis B, H. influenzae B (HIB).

**Passive** Ready made antibodies are given. Provides instant protection, but only lasts a short time, and can cause allergic reactions, e.g. ATS, diphtheria antitoxin, anti-snake venom, hepatitis A gammaglobulin.

Immunisation from mother to baby through the placenta is an example of naturally occurring passive immunity.

#### Types of active vaccines

**Live** - best for long term protection, and often fewer doses are required e.g. BCG, measles (1 dose), polio (ideally 4 doses).

**Killed** - antibody stimulation not as good, therefore need multiple doses. e.g. pertussis (3 doses).

**Toxoids** - also less strong antibody protection, so need more doses, e.g. tetanus toxoid vaccine (5 doses give lifetime protection).

#### Timing of vaccines

**BCG** Give at birth.

**Polio** Give at birth (zero dose) and then at the same time as DPT, now combined with hepatitis B and HIB in pentavalent vaccine.

**DPT** Start after 1 month and give at monthly intervals, now combined with hepatitis B and HIB in pentavalent vaccine.

**Measles** Maternal antibodies cross the placenta and last about 6-9 months, so vaccinate at over 9 months, EXCEPT when at risk of contact with measles at age 6-9 months (in hospital, or refugees during measles outbreaks). In this case vaccinate at 6 months, repeating at 9 months.

### 5.2.2 Immunisation Schedule

<u>Vaccine</u>	Birth	<u>Weeks</u>			<u>Months</u>
		6	10	14	9
BCG <sup>1</sup>	+				
Pentavalent <sup>2</sup>		+	+	+	
Polio <sup>3</sup>	+	+	+	+	
Measles <sup>4</sup>					+

### 5.2.3 Contraindications & reactions

#### **BCG**

Contraindication	Acute TB, AIDS (but can give to HIV +)
Reaction & treatment	Ulcer - heals spontaneously Local lymphadenitis - heals spontaneously Fistulated nodes – INH for 6 months

#### **Polio**

Contraindication	Nil
Reaction & treatment	Vaccine associated paralysis (very rare)

#### **Pentavalent**

Contraindication	Fever > 38°C - delay giving vaccine History of severe reaction to earlier dose of pentavalent
Reaction & treatment	Fever or rarely febrile convulsions - paracetamol

#### **Measles**

Contraindication	Nil
Reaction & treatment	Fever after 1 week or occasionally mild rash

### 5.2.4 Storage and Shelf Life

#### **The cold chain**

All vaccines are heat sensitive, and from manufacture to use, have to be stored at the right temperatures to preserve their potency. This means

<sup>1</sup> Give BCG intradermally in the R upper arm (0.05 ml if < 12 months, 0.1 ml if > 12 months).

<sup>2</sup> Give pentavalent intramuscularly in lateral thigh (0.5 ml).

<sup>3</sup> Give Polio orally on tongue (2 drops).

<sup>4</sup> Give measles deep sub-cutaneous to lateral thigh (0.5 ml). NB: Give at 6-9 months during measles outbreaks, if admitted to hospital or refugees.



## 5 Appendices - Immunisation

maintaining fridges, freezers and cold boxes. The Expanded Program of Immunisation (EPI) distribute devices that indicate automatically whether the temperature has risen above a safe level. Any vaccines exposed to the wrong conditions will lose efficacy and therefore must be thrown away.

<b><u>VACCINE</u></b>	<b><u>Storage</u></b>	<b><u>Diluent</u></b>
BCG	Fridge	Special diluent provided
Pentavalent	Fridge	Already diluted
Polio	Freezer	Already diluted
Measles	Freezer	Special diluent provided

### Handling of Vaccines

- BCG easily dies if exposed to sunlight.
- BCG can be killed by spirit and other antiseptics - so sterilise syringes and needles by pressure sterilizer or boiling, but NOT by soaking in antiseptics. Clean skin with water NOT spirit.

### 5.2.5 The 10 Commandments of Immunisation

- \* Never use expired vaccines.
- \* Immunise at every opportunity, especially on admission to hospital.
- \* Always ask for the Health Passport.
- \* Don't spread HIV - always use a sterile needle and syringe for each injection.
- \* Monitor the cold chain.
- \* Keep all vaccines at correct temperature.
- \* Never freeze pentavalent/tetanus toxoid.
- \* Never use sterile water for dilution.
- \* It is safe to give all vaccines at the same time at different body sites.
- \* There are very few contraindications to vaccination [5.2.3].

## 5.3 Radiology

Remember that in general:

- \* Clinical skills often tell you more than Xrays will.
- \* Don't do an Xray unless you or someone else can read it.

### 5.3.1 Chest Xrays

#### Quality

**Is the film central?** Are clavicular heads symmetrical in relation to the spine?

**Is the film under or over penetrated ?** You should just be able to see the vertebral spines as well as the ribs joining the spine, through the heart.

**Is the film in full inspiration?** The diaphragm should cross the anterior end of the sixth rib, or the posterior of the ninth.

#### In the normal chest Xray

**Hilum** The right hilum is lower than the left, and the right diaphragm is higher than left. The horizontal fissure is situated about mid height in the right lung and is horizontal, but not always visible. These positions may be altered by lung or abdominal pathology.

**Heart size** In neonates allow 60% of the thoracic diameter, compared to 50% in older children and adults. Remember that in antero-posterior (AP) views the heart appears larger. (If the child is ill and the chest Xray is taken supine, there will be no stomach gas bubble fluid level as a clue. A supine Xray will exaggerate apparent heart size).

**Mediastinum** This is wider in infants being filled by the thymus, which shrinks and disappears by 2 years.

#### Special Situations

**Cardiac Hypertrophy** The differences between left and right ventricular hypertrophy is shown on the next page.

**Foreign body inhalation**

The right main bronchus is more commonly involved. If child can cooperate ask for inspiratory and expiratory films to see if both lungs

deflate equally on expiration. The foreign body is rarely visible. With a ball valve effect there will be hyper-expansion of one lung with possible shift of mediastinum, and even herniation of the lung to the other hemithorax. Complete blockage of an airway causes lung collapse, so look also for displacement of the horizontal fissure.

### **Pneumothorax**

Air under atmospheric pressure enters the pleural space, which is normally under negative pressure (like a vacuum). This causes collapse of part or all of the lung depending on how much air gets in. Look for the lung margins, comparing one side with another, and for tracheal, or even mediastinal shift.

### **Pyopneumothorax**

There is a collapsed lung, and a horizontal fluid level. If massive this will cause mediastinal shift.

### **Effusion/ empyema**

Fluid in the pleural spaces is seen as a crescent shaped fluid level (because the pleural space is under negative pressure), which collects at the angles and in the fissures. It is impossible to distinguish between an effusion and an empyema on Xray alone.

### **Loculated effusion**

There are lines which do not fit the normal anatomical markings, representing walled off fluid filled spaces that develop in chronic situations.

### **Abscess**

Thick walled circular opacity, often with shaggy margins, sometimes containing fluid levels (air in the abscess). The diameter is the same in lateral and postero-anterior (PA) views, whereas these fluid level diameters differ in pyopneumothorax

### **Pneumatocoele**

Thin walled, air filled circular shadow, usually multiple.

### **Cavitation**

Thin or thick walled, air or fluid filled, asymmetrical lesions, with a fluid level if it contains air at atmospheric pressure.

### **Collapse**

May be of a lung (mediastinal shift), lobe (fissure shift), or segment (horizontal lines). In all cases the shift is towards the lesion.

### **Consolidation**

If consolidation is lobar, it is limited by the fissures but it is more commonly patchy and diffuse (bronchopneumonia). Loss of definition of the cardiac outline indicates consolidation in adjacent lung.

**Air bronchogram**

The smaller airways (beyond the main bronchi) become clearly visible as dark branching lines, shown up by the whiter areas of consolidation around them. In children this is a useful sign of consolidation.

**Pericardial effusion**

Fluid in the pericardial sac changes the normal cardiac outline, which becomes globular. Ultrasound is generally a better investigation to prove a pericardial effusion, but remember a little fluid in the pericardium is normal. There may be air in the pericardium (pneumo-pericardium), in which case you should see a fluid level, and the pericardial sac separate from the heart.

**Tuberculosis**

In miliary TB there is diffuse small nodules and mottling, throughout both lung fields, whilst hilar nodes are commonly not enlarged.

### **Common Errors**

- Missing a pneumothorax or an effusion by not looking for the lung borders, and not comparing one side with the other.
- Mistaking the gastric bubble for lung pathology!
- Diagnosing large hearts in infants.
- Failing to identify or use the position of the horizontal fissure and trachea for diagnostic purposes.
- Missing abnormalities (such as infiltrates, enlarged nodes) in the "difficult areas" of the chest Xray film, which are behind the heart, and behind the domes of diaphragm. If a film looks normal, always look at these special areas before excluding radiological abnormalities.

### **5.3.2 Abdominal Xrays**

#### **Intestinal obstruction**

- dilated loops of large bowel (more peripheral, haustration lines go part way across the loops) or small bowel (more central, haustration lines go all the way across the bowel loops).
- 5 or more fluid levels in an erect film.

#### **Hypertrophic pyloric stenosis<sup>1</sup>**

- large distended stomach.
- "String sign" elongated pyloric canal well visualised with barium.
- little gas in bowel.

#### **Duodenal atresia**

- "double bubble" in erect film (air in stomach and duodenal cap).
- no air in rest of abdomen.

#### **Intestinal perforation**

- air under the diaphragm on an erect chest Xray.

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<sup>1</sup> But expert ultrasound is a better investigation for diagnosis of pyloric stenosis.



### 5.3.3 Bone Xrays

Examine Xrays of bone systematically for the following:

- One or more bones affected (localised or generalised disease).
- Are the soft tissues affected.
- Bone density - normal, porotic, or sclerotic.
- Any periosteal reaction (osteomyelitis, congenital syphilis).
- Shaft (fractures).
- Metaphysis and growth plate (osteomyelitis, fractures, rickets).
- Epiphysis and joint (fractures, rickets, cretinism).

Remember that not all abnormalities can be seen on Xray.

## 5.4 Procedures

Skill in all practical procedures can only be gained through practice, initially under good supervision. Before carrying out any procedure, first make sure that you are comfortably seated, the child is securely held by an assistant on a couch or bed, and there is good lighting.

### 5.4.1 Blood Samples

In older children and for small samples of venous blood in younger children, arm veins can be used with a 21 gauge needle (with a vacuum tube or with or without a syringe). Apply a tourniquet around the upper arm, inspect for suitable veins (usually in the cubital fossa, but occasionally on the back of the hand) and clean skin with spirit. Then, stretching the skin with the thumb not holding the needle/syringe, insert the point of the needle 0.5-1 cm distal and slightly to one side of the place where you wish to enter the vein. Then push the needle firmly into the vein.

When larger volumes of blood are needed in small children use the neck veins. These veins are usually quite large and are not near vital structures. Get an assistant to hold the shoulders of the child lying across the couch on his back with head off the edge. Bend the needle slightly at the hub and with the hand not holding the syringe, turn the child's head to the other side and extend the neck over the side of the couch. Clean the skin with spirit, stretch the skin and push the needle through the skin slightly to one side of the vein to be entered. Then firmly push the needle into the vein and aspirate the blood.

For bilirubin estimations in babies use capillary blood from the heel. Clean the heel with spirit and with a sterile lancet prick the side of the plantar surface to avoid piercing the calcaneus bone (may cause osteomyelitis). Make an adequate stab to avoid excess squeezing and draw blood into two heparinised capillary tubes held tilted up at a slight angle (blood will run in by capillary action). Finally plug one end of the tube with plasticine.

Use a finger stab in older children for capillary blood Hb estimation and blood films (for malaria, trypanosomiasis and relapsing fever). Clean the end of a finger and with a sterile lancet prick the lateral side of the pulp of the finger near the tip. Avoid pricking, and possibly infecting, the bone of the terminal phalanx.

### 5.4.2 IV Drips

In small babies the scalp veins are often very useful for drips. A butterfly needle is highly desirable,<sup>1</sup> though an ordinary needle can be manipulated using artery forceps in emergency. If available, a narrow gauge plastic cannula (24 gauge for infants, 22 gauge older children) will give a longer lasting drip, but is more difficult than a butterfly needle to insert. Oblique illumination will often make the veins more easily seen.

- \* Shave the scalp for a wide area round the vein to give a clear view, and for strapping to be taped on.
- \* Distinguish veins and arteries in the scalp by palpation for a pulse, and by determining the direction of flow in the vessels. This is important because intra-arterial drips are slower and may cause scalp necrosis, if continued for over an hour, as the infused fluid is low in oxygen.
- \* Occlude the vein by pressure from a finger or tourniquet of drip tubing round the baby's scalp, and swab the skin over the vein (reflections from the moist scalp often showing the vein more clearly). Attach the needle to the drip and fill with fluid, and then expel one drop by squeezing the tubing so that a small air bubble is visible at the base of the needle. Movement of this bubble gives warning that the vein has been entered, except in very vasoconstricted babies.
- \* Tension the skin with thumb and finger of the left hand (if you are right-handed) and introduce the needle just lateral and distal to the point where it is to enter the vein.
- \* When the needle is through the skin, detach the drip from the butterfly needle, to allow blood to enter the needle freely, and then push the needle into the vein. Movement of the bubble and appearance of blood in the butterfly tubing indicate success.<sup>2</sup> To check whether the needle is in the vein, the drip can be reattached and the tubing gently squeezed. A small swelling (air bubble/fluid) under the skin indicates failure.
- \* In difficult cases you have to try several veins at different sites in the scalp, and both sides of the head, including the occipital, as well as temporal, regions will need to be shaved.
- \* Once the drip is in place, fix it firmly with a thin strip of strapping. Use a further strip to secure a coil of butterfly tubing to the scalp, to

<sup>1</sup> Gauge 21 for blood, gauge 23 for clear fluids, gauge 25 for neonates

<sup>2</sup> Though sometimes in very shocked babies venous pressure is too low, even when the vein has been entered.

give safety in case the drip is jerked. When keeping the drip in place is particularly important, strips of plaster of Paris are a useful and more durable alternative to adhesive strapping.

- \* You can also use veins of forearm and back of the hand, but need splints (tongue depressors, or drip set cardboard boxes, covered thinly with gauze bandage) to prevent movement at wrist or elbow.
- \* When none of these is possible, the external jugular, or less easily, the anterior jugular veins can be used, though an IV cannula is needed or the needle will soon tissue. There is the added disadvantage that neck movements alter the drip rate of flow.
- \* To put a cannula in the external jugular, lay the baby flat on his back, the arms and shoulders being firmly controlled by the assistant, and use a technique similar to taking blood from a neck vein [5.4.1].

### 5.4.3 Intraosseous Drips

When an infusion is urgent but an intravenous drip is impossible, use this method. Choose a point on the medial surface of the upper tibia about 2 cm below the tibial tuberosity (where there is no muscle). Clean the skin well with antiseptic to avoid causing osteomyelitis and insert the needle pointing slightly downwards away from the tibial epiphysis. Preferably use a trochar and cannula, as ordinary needles tend to block with bone: if no trochar and cannula is available, use a wide bore needle (at least gauge 21). Use local anaesthetic, if the child is conscious, as pushing the needle through the periosteum and bone cortex (firm but not too hard pressure to avoid bending the needle tip, with back and forward rotation) is painful. When the marrow is entered there is a give, and the needle becomes fixed. Syringe through the needle with sterile saline (or Ringer's lactate) to clear the needle, and then attach a drip, or for rapid infusions inject IV fluid rapidly with a large sterile syringe. To reduce the risk of osteomyelitis, replace the intraosseous infusion with a venous drip within 4 hours.

### 5.4.4 Venous Cut Down Drips

These are best avoided as they sacrifice a vein, and are prone to infection. However if an IV drip is essential, and no other site possible.<sup>1</sup> a cut down on the long saphenous vein at the ankle may be tried.

- \* Infiltrate local anaesthetic (1-2 ml of 1% lidocaine) intradermally and

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<sup>1</sup> Consider also inserting a drip into the upper tibial marrow.

5 *Appendices - Procedures - Venous Cut Down Drips*

subcutaneously over the long saphenous vein just anterior to the medial malleolus, and make a transverse incision in the overlying skin.

- \* Identify the vein, free it from surrounding fascia by blunt dissection, and lift it up on artery forceps.
- \* Pass two catgut ligatures round the vein, and tie knots in both, leaving the proximal one loose, but tying the distal one tight. Hold both with artery forceps, to hold the vein still for the next stage.
- \* Using sharp pointed scissors make a nick, pointing upwards, in the superficial side of the vein. Introduce the cannula through the hole, securing it by tying the proximal ligature over it. If the cannula has a flange with perforations in it, pass the ligature through these and tie again to hold the cannula in place.
- \* Run the drip, cut the ligatures short and close the skin with interrupted catgut or silk. Make extra sure that the tubing is well secured, as it easily comes adrift in irritable children.

### 5.4.5 IM Injections

Intramuscular injections in babies may be given at three sites, the deltoid muscle, the lateral muscles of the thigh in their upper two thirds, and the upper outer quadrant of the buttock. It is important to make sure that the injections are given in the right areas, and that they do not penetrate down to bone.

#### Risks

- sciatic nerve palsy (with paralysis of the affected leg) if the injection into the buttock is given too far medially,
- osteomyelitis of the femur if the injection in the thigh is too deep and not sterile (usually staphylococci from the skin).

### 5.4.6 Lumbar Puncture

This can be done either with the child lying on one side or sitting upright, and you should use the position you are more familiar with. In the lateral position the assistant holds the trunk of the baby in flexion by grasping the pelvis and shoulders (not the neck which should be left extended to avoid any obstruction of the airway). In the sitting position the baby is again held with the spine flexed but in the vertical plane.

- \* Identify the appropriate intervertebral gap (L3-4) by drawing an imaginary line from the top of the iliac crest to the spine, and mark it by indentation of the skin with a clean finger nail.
- \* Clean the skin well with spirit, which should dry. If care is taken not to touch the skin of the back, after it has been cleaned, and not to touch the metal of the sterile disposable needle at all, more elaborate preparations to ensure sterility of the procedure are not essential. (It is also important not to perform the procedure with wet hands, in case contaminated water runs down the needle shaft during the procedure).
- \* Insert the needle<sup>1</sup> through the skin and slowly push in, aiming at about the umbilicus on the other side, i.e. angled slightly towards the head, until a slight "give" is felt on piercing the dura, and CSF begins to flow.
- \* Collect no more than 20 drops of CSF (unless unusual investigations, like looking for TB bacilli, are to be performed). If the fluid is bloodstained, the first few drops can be discarded, in the hope that the fluid will become clear. If not, the procedure can be performed one space higher up the spine.

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<sup>1</sup> 23 gauge in small infants, 21 gauge in older infants and children.

### 5.4.7 Subdural Tap

If subdural haematoma or subdural effusion (a complication of meningitis) is suspected, the subdural space should be explored. In infants and some children up to the age of 1½ years, this is possible through the lateral angles of the anterior fontanelle.

- \* Shave the scalp over and around the anterior fontanelle, and clean with spirit which should then be allowed to dry.
- \* Insert a sterile gauge 21 injection needle in each lateral angle of the anterior fontanelle in turn, and introduce a short distance, till there is a distinct give as the needle pierces the dura mater. From the subdural space old blood from a haematoma or, in subdural effusion, very yellow fluid may be obtained. Drain as much as possible.
- \* The procedure may need to be repeated daily at first, then on alternate days, till the subdural effusion has dried up. With subdural haematomas, surgery may be needed to remove the whole haematoma.

### 5.4.8 Rectal Snip

This procedure is used in the diagnosis of schistosomiasis.

- \* Explain the procedure is uncomfortable but not painful, and ask the patient to empty the bowel. Sedation is advised for anxious patients.
- \* With the patient lying in the left lateral position, and the right knee flexed, insert a lubricated proctoscope into the rectum, which can be visualised with a bright light source.
- \* Obtain up to 4 samples of rectal mucosa using a sterile pair of biopsy forceps, at 3, 6, 9, and 12 o'clock (as you look down the proctoscope). Be careful not to take too big a "bite" with the forceps. Place the tissue samples obtained in specimen bottles containing formalin.
- \* Complications to this usually safe procedure include perforation of the bowel wall and haemorrhage.

### 5.4.9 Bladder Stab

Use this procedure whenever it is important to collect a clean urine sample in infants and toddlers, (when the more usual mid stream urine technique is not feasible, and the adhesive plastic collecting bag is not available or has not worked). Bag samples in infants are often contaminated despite prior cleansing. The bladder stab is quick, reliable and safe.

- \* Change and feed the infant

- \* After 30 minutes check the bladder is full, and the napkin dry.
- \* Lie the child on his/her back, and ask an assistant to hold the thighs pinned down, with the hips in full flexion and abduction. At this stage proceed quickly before the child empties the bladder with fright!
- \* Insert a 23 g needle (attached to a 5 ml syringe) at a point 2 cm above the symphysis pubis, through the skin for about 3 cm, at right angles to the skin and applying slight suction with the syringe. If the needle is in the bladder, urine should flow freely into the syringe.

#### 5.4.10 Joint Aspiration

The joints most commonly needing aspiration are the shoulder, knee, elbow and ankle. The hip, being very deep to muscle, is difficult, and should be drained by a surgeon if necessary.

The shoulder is usually aspirated laterally, the knee on the medial side, the elbow posteriorly and the ankle either medially or laterally, but be guided by where fluctuation is most obvious.

- \* Before starting the procedure, warn the child that he is going to get an injection to take away pain in the joint.
- \* Clean the skin with spirit and allow to dry, then infiltrate local anaesthetic (2-5 ml 1% lidocaine) intradermally down to the joint capsule, which is very sensitive to pain.
- \* Insert the sterile 21 gauge aspirating needle attached to a sterile syringe, and aspirate the fluid in the joint.

#### 5.4.11 Chest Aspiration

This is usually done posteriorly at about the eighth intercostal space on the affected side, mid way between the spine and the side of the chest. When the effusion is loculated, as shown by Xrays in PA and lateral planes, it may be necessary to aspirate in the axilla or anteriorly in an appropriate intercostal space. A 3-way tap is only necessary for aspiration of large volumes.

- \* Have the child held, usually sitting forward, by an assistant who talks to him to reassure and distract him.
- \* Identify where you are going to do the tap by palpation and percussion, marking the point by pressure with a clean finger nail.
- \* Clean the skin with spirit and allow it to dry, then inject local anaesthetic (2-5 ml of 2% lidocaine) intradermally and then into the intercostal space to block the intercostal nerve, which runs along the



lower edge of the rib above the space, and to anaesthetise the parietal pleura.

- \* Attach the sterile 21 gauge aspirating needle to the sterile syringe (via a sterile 3-way tap, if a large volume is to be aspirated), and introduce it into the pleural space. Normally up to 500 ml of fluid may be aspirated, measured and sampled for testing.

#### 5.4.12 Chest Drain

An under-water-seal drainage system is required, and preferably a tube, like a plain catheter<sup>1</sup> can be used and stitched into the skin. Suitable sterile equipment, including syringe and needle for local anaesthetic, a scalpel for incising the skin and suture material, needle and needle holder should be prepared. For drainage of an empyema, a drain is usually inserted in the mid axilla on the affected side; for a pneumothorax the second intercostal space anteriorly in the mid clavicular line is preferable.

- \* Restrain the child, who may have to be sedated.
- \* Identify and mark the site for the drain, then clean the skin with spirit and iodine if available.
- \* Inject local anaesthetic (2-5 ml of 2% lidocaine) intradermally and then into the intercostal space to block the intercostal nerve, which runs along the lower edge of the rib above the space, and to anaesthetise the parietal pleura.
- \* Incise the skin, make a stab incision down to the pleura, clamp the catheter tube and insert an artery forceps grasping the end of the catheter into the pleural cavity.
- \* Suture the skin round the catheter, and when necessary fasten the catheter to the chest wall with silk or similar non-absorbent suture material. Apply a dressing and attach the catheter to the under-water-seal drainage system. Then unclamp the catheter.

#### 5.4.13 Pericardial Tap

The pericardium may be tapped through a subxiphoid approach, or the 4th left intercostal space close to the sternum, using the "bare area" where pleura does not cover the anterior surface of the pericardium. A pericardial tap is a painful and frightening procedure, and it is advisable to sedate the child well beforehand, to give atropine to prevent vagal <sup>1</sup> and to use local anaesthetic. It is also advisable to use a disposable

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<sup>1</sup> A Foley catheter is not ideal, as the catheter opening is rather far beyond the bulb. bradycardia,

cannula rather than just a sharp needle, so that there is less danger of puncturing the heart as the pericardial effusion is aspirated.

- \* Have the child held by an assistant,
- \* Identify the 4th left intercostal space and sternal edge and mark the skin with a clean finger nail. Clean the skin with spirit and preferably iodine, and inject local (2 to 5 ml of 1% lidocaine) intradermally and then into the 4th left interspace down to the pericardium.
- \* After a suitable pause, insert the sterile cannula on its needle through the anaesthetised area into the pericardial cavity, and, when fluid drains, advance the cannula over the needle and withdraw the needle.
- \* Allow fluid to drain from the cannula, and if there is a large volume, tape the cannula to the chest wall, connect to a drip set and allow to drain into a closed sterile bag.

#### 5.4.14 Ascitic tap.

This is done to diagnose the cause of ascites (which may be TB, pyogenic bacterial infection, nephrotic syndrome, cirrhosis, tumour specially Burkitt's lymphoma or Kaposi's sarcoma) and sometimes to reduce the volume to relieve distension, pain and dyspnoea. Send fluid obtained to the lab to determine cell count and differential, protein level and for Gram and ZN stains.

- \* Percuss the abdomen to demonstrate where the effusion is.
- \* Clean the skin with spirit and inject 1% lidocaine local anaesthetic intradermally and then down to the peritoneum.
- \* Insert a wide bore needle or preferably a cannula and aspirate.
- \* Do not remove more than 500 ml at a time as rapid reaccumulation can cause shock.

#### 5.4.15 Bone Marrow Aspiration

This procedure can be done with a fine needle, but there is a better chance of a good sample if a suitable short-bevelled trochar and cannula are used. In children the iliac crest is the preferred site for biopsy.

- \* Sedate the child if needed. An assistant holds him laid on his side.
- \* Mark a point 2-5 cm behind the anterior superior iliac spine and 1 cm below the iliac crest with a clean finger nail. Clean the skin with spirit and preferably iodine and inject local anaesthetic (2-5 ml of 1% lidocaine) intradermally and down to the periosteum around the area to be biopsied.

- \* After a suitable pause, assemble the sterile trochar and cannula and push through the skin, down to the periosteum. Tighten the movable guard on the cannula at 5 mm from the skin surface, and push trochar and cannula into the marrow cavity. There is a distinct give on entry.
- \* Remove the trochar, attach a sterile 5 or 10 ml syringe, and aspirate marrow by short sharp pulls on the piston of the syringe. Eject the aspirate from the syringe onto a clean watch glass. The presence of rather fatty looking globules amongst the blood indicates marrow.
- \* Remove the cannula and strap a sterile dressing to the puncture site.
- \* Make microscope slide smears from the marrow as for a blood film.

#### 5.4.16 Fine Needle Aspiration Biopsy

This procedure is very useful for diagnosing Burkitt's lymphoma in masses in the jaws, face, abdominal cavity and elsewhere.

#### Preparations for biopsy

Weigh the patient to calculate ketamine dosage. Prepare glass slides for the biopsy (with the patient's initials, biopsy number), syringe of anaesthetic, needles, and spirit before bringing the child into the procedure room to minimise the child's fear.

#### Anaesthesia

Usually in small children use intravenous ketamine anaesthesia. Draw up a total of 3-4 mg of ketamine per kg body weight into a sterile syringe. (Smaller doses should be used in ill patients). Inject the ketamine intravenously in frequent small boluses, while watching the patient carefully till good anaesthesia is obtained.

You may use local anaesthesia with 1 or 2% lidocaine injected intradermally (into the skin) over the area to be biopsied, without IV ketamine, in older or very calm children, or ill children with depressed consciousness or partly obstructed breathing. Take care not to inject the lignocaine subcutaneously round the tumour to be biopsied. Slowly injected intradermal lignocaine will give good skin anaesthesia and ensure that the lignocaine does not enter the biopsy specimen.

#### Sampling procedure

Use a 23 gauge or preferably narrower needle (e.g. 25 or 27 gauge). Insert the needle (not attached to a syringe) into the tumour and rapidly

push it through the tumour in many different directions. If blood appears in the needle hub, stop the biopsy procedure, withdraw the needle from the tumour and make slides from material by ejecting small drops with a syringe onto slides.

### **Slide preparation**

Do not touch slide surface with the fingers, as this will make them greasy, and cells will not stick to them. Squirt small volumes of aspirated tumour material on the slides, and, before the droplets dry, rapidly spread them over the slides with the edge of another slide, using the technique for making fine blood films. Dry the slides to be fixed with methanol rapidly in air and leave them to dry well for 2 to 12 hours. Then fix them in methanol (fresh at the start of each week in a glass fixing jar with vaseline sealed lid). Remove them from the jar, dry rapidly, leave for 10 minutes, and put inside a labeled slide holder, to protect from dust and breakage.

### **5.4.17 Exchange Transfusion**

This procedure should not be performed without previous training. It is used in severely jaundiced neonates when a risk of kernicterus is thought to be high to reduce the indirect bilirubin. [Section 2.4.6]

#### **Dangers of exchange transfusions:**

- sepsis
- over-transfusion (causing pulmonary oedema) and under-transfusion (causing shock)
- cooling
- hypoglycaemia
- hypocalcaemia
- hyperkalaemia (high serum potassium)
- incompatible blood transfusion
- infection with malaria, hepatitis, B, syphilis, or HIV, and bacteria
- vomiting with inhalation

A complete disposable, sterile, closed exchange transfusion set is highly desirable and reduces the risk of infection, but is seldom available. Without it, care should be taken to collect together all the equipment needed for the procedure before starting.

**Suggested list of equipment**

- sterile drapes including one with a 5-7 cm diameter central hole;
  - 1 sterile size 3 scalpel handle
  - 1 pair sterile sponge holding forceps
  - 2 pairs sterile curved mosquito artery forceps
  - 1 pair sterile small toothed dissecting forceps
  - 1 pair sterile small non-toothed dissecting forceps
  - 1 sterile small gallipot
  - 1 sterile large metal container for waste blood (bowl or kidney dish)
  - 2 sterile 20 ml syringes
  - 1 sterile umbilical catheter (or gauge 6 nasogastric feeding tube)
  - 2 sterile disposable 3 way taps
  - 1 sterile small round ended scalpel blade for size 3 handle
  - Sterile gauze swabs
  - Spirit and iodine for swabbing the abdominal wall
  - 1 low reading thermometer
  - 1 stethoscope to be strapped to the baby's chest
  - 2 tubes for clotted blood
  - 2 tubes for anticoagulated blood
  - 1 large splint
  - 1 watch or clock
  - Several gauze bandages
  - Angle-poise or other good source of light
  - 50% dextrose solution and 10% calcium gluconate for IV injection in case of convulsions
  - Means of keeping the baby warm; e.g. incubator, electric blanket, electric heater, insulated hot water bottles.
  - 1 clipboard, with sheets of paper ruled out suitably to record the following: time, volume out, running total of volume of blood removed, volume in, running total of blood injected, heart rate, venous pressure, temperature, comments.
- \* Allow the fresh, HIV Elisa negative, compatible blood to warm up and try not to disturb the cells if they have sedimented. About 190 ml/kg of blood will be needed. Run the drip through with the blood.
  - \* Give the baby IM benzyl penicillin and gentamicin for sepsis risks.
  - \* Prepare the baby by emptying the stomach with a nasogastric tube, and by tying the limbs securely with gauze bandages to the splint.
  - \* Record baseline temperature and heart rate. Keep the baby warm in an incubator, or with electric heating blankets or hot water bottles.

- \* Wash and dry your hands and put on sterile gloves.
- \* Clean the baby's abdomen, lower chest and upper thighs with spirit and iodine, and drape the baby, with the hole in the final drape centred on the umbilicus. Connect the two three-way taps together to give a system that allows syringe, blood from the bottle, outlet to the patient and waste outlet to inter-communicate by turning the two taps. The assistant then passes the end of the drip tube to the operator, and, as it is not all sterile, he receives it in iodine soaked gauze swabs, and attaches it to the three way taps.
- \* The most difficult part of the procedure is usually to identify and cannulate the umbilical vein. This is the largest of 3 vessels, the other 2 being arteries. Amputate the cord close to the skin and identify the umbilical vein in the upper part.
- \* Grasp the vein wall with mosquito forceps, and introduce the umbilical catheter. Finger tip pressure may be needed over the abdomen above the umbilicus to help the catheter enter the inferior vena cava.
- \* Success is recognised when blood flows back freely into the catheter. Take pre-exchange blood samples for Hb and total and direct bilirubin.
- \* Then proceed with the exchange. It should be carried out in deficit to the extent of about 10-20 ml, i.e. always remove blood from the baby before injecting any. After he completes each removal of blood from or injection into the baby, the operator tells the assistant who records the time and volume and whether it has been removed from or injected into the baby, keeping running totals up to date.
- \* In well full term babies 20 ml volumes of blood can be moved, but in smaller and ill babies it is wiser to change 15 or even 10 ml at a time. Removal of blood from the baby and especially injection into the baby should be done rather slowly, but ejection of blood to the waste bowl, or drawing it in from the bottle can be fast.
- \* Count and record the heart rate every five minutes or so.
- \* If there is any sign that the baby is unwell (significant rise or fall in heart rate, restlessness, convulsion, apnoeic attack etc) stop the procedure until the baby is stabilised.
- \* Convulsions may result from hypoglycaemia, hypocalcaemia, or hyperkalaemia (or of course kernicterus). Take specimens for appropriate tests and try the effects of IV 50% glucose (1.0 ml/kg diluted with 5% dextrose 4 ml/kg) and 10% calcium gluconate (1-2 ml/kg) if needed.

## 5 Appendices - Procedures - Exchange Transfusion

- \* Continue the exchange till 180 ml/kg of sedimented red cells have been transfused, over about 1<sup>1</sup>/<sub>2</sub> hours. Then take specimens in two steps, discarding the first 4-5 ml withdrawn, to ensure blood injected towards the baby but still in the catheter is washed out, and testing the second sample for Hb and total and direct bilirubin levels. This will leave the baby with a 10-15 ml deficit which is usually desirable.
- \* At the end of the procedure record the heart rate, temperature and venous pressure (number of cm that the blood runs vertically back up the open catheter in the umbilical vein). If that is over 4 cm, remove more blood, if below 1 cm transfuse a few more ml. Then gradually withdraw the catheter and control bleeding not by suturing, but by strapping a wad of sterile gauze firmly over the umbilical area.
- \* Release the baby from the splint and warm up briefly before the baby is fed and continued on antibiotics and phototherapy.

Steps in the exchange procedure are summarised in the diagram below.

### **Steps to be done in an exchange transfusion**

- Step 1: Aspirate blood from the baby.
- Step 2: Discard baby's blood to waste.
- Step 3: Aspirate fresh blood from the bottle.
- Step 4: Inject fresh blood into the baby.

## 5.5 Fluid Balance

Most childhood diseases lead to either a decreased intake or increased loss of body fluids. Many diseases therefore can cause a fluid imbalance, which may become life threatening in itself. Infants and children have a higher water and electrolyte turnover than adults. They become more easily dehydrated, if needs are not met. On the other hand infants are easily over hydrated, which can lead to brain oedema or heart failure. Careful calculation of fluid needs is therefore essential for treating and preventing dehydration and preventing over hydration.

Maintenance Requirements replace daily losses from the skin, lungs, kidneys and gastrointestinal tract. Healthy children get maintenance requirements from food and drink. Due to anorexia or vomiting the intake of children may not be enough to cover maintenance requirements. This applies especially to unconscious children.

### Maintenance Requirements

<b>1st Week of life (term baby)<sup>1</sup></b>	Day 1	60 ml/kg/day
	Day 2	90 ml/kg/day
	Day 3	120 ml/kg/day
	Day 4	150 ml/kg/day

### **After 1st week of life**

1st 10 kg	100 ml/kg
next 10 kg	+50 ml/kg
> 20 kg	+25 ml/kg
> 10 years	2-3 litres/day

### EXAMPLE 1

Maintenance requirements for a 12 kg child:

1st 10 kg (100 ml x 10)	= 1000 ml
Next 2 kg (50 ml x 2)	= 100 ml
<b>TOTAL</b>	<b>= 1100 ml</b>

### EXAMPLE 2

Maintenance requirements for an 28 kg child:

1st 10 kg (100 ml x 10)	= 1000 ml
Next 10 kg (50 ml x 10)	= 500 ml
Next 8 kg (25 ml x 8)	= 200 ml
<b>TOTAL</b>	<b>= 1700 ml</b>

<sup>1</sup> If the infant is SGA or preterm, increase to 180-210 ml/kg/day after day 5.



### **Replacement Requirement**

These have to be added to maintenance, if the child is dehydrated or losing excess fluids.

A child can lose abnormal volumes of fluid through

- \* diarrhoea
- \* vomiting
- \* burns or skin wounds
- \* draining fluids
- \* excess urine (kidney disease, diabetes)
- \* rapid breathing
- \* fever

If a child has lost an abnormal volume of fluid, this must be estimated and replaced.

### **How to Estimate Replacement Requirements**

Replacement fluids can be calculated by

- \* Measuring intake and output and calculating the difference.
- \* Weighing. Make use of weights recorded in the health passport and always weigh children on admission and regularly while on treatment for dehydration.
- \* Estimating the insensible fluid loss in children with fever (add 10% of maintenance for each 1 degree of fever).
- \* Assessing dehydration.

### **Assessment of Degree of Dehydration**

<b><i>Signs</i></b>	<b><i>Severity of dehydration</i></b>	<b><i>Management</i></b>
2 or more of lethargy or coma sunken eyes unwilling/unable to drink skin pinch goes back v. slowly	severe	Child needs IV or NG tube fluids. Refer to hospital giving ORS on way. If cholera around, give erythromycin.
2 or more of restless, irritable sunken eyes not feeding, drinks eagerly, thirsty skin pinch goes back slowly	some	give 70 ml/kg ORS in 1st 4 hours, and reassess. If breast feeding, continue, if not, give 100-200ml water

<b>Signs</b>	<b>Severity of dehydration</b>	<b>Management</b>
insufficient of these signs to classify as dehydrated	none	teach mother to give extra fluids, to continue to feed, and to look for danger signs.

In babies under 2 months any degree of dehydration shows need for hospital referral and the likely need for antibiotics for sepsis [2.4.7].

If diarrhoea has lasted over 2 weeks with dehydration, correct dehydration and refer to hospital. If there is no dehydration, advise on feeding, give vitamin A, review and consider HIV infection.

### EXAMPLE 3

Fluid needs for a severely dehydrated 7 kg child:

Maintenance fluid 100 ml/kg for weight 7 kg	= 700 ml
Replacement fluid for 10% dehydration (of 7 kg)	= 700 ml
TOTAL needs in 24 hours	= 1400 ml

In moderate or severe dehydration give replacement requirements in 4-6 hours (17-25 ml/kg/hour): in this example 120 - 175 ml/hour. After 4-6 hours reassess the child. If no longer dehydrated, the child needs maintenance plus replacement of current losses over the next 18-20 hours. Persisting dehydration needs to be estimated as before and replaced.

### What Fluids Should be Given?

#### General Rules

- \* Breast milk is best for maintenance, because of its high energy and protein and moderate electrolyte content. Give it whenever possible, by spoon or NGT if the babe will not suck.
- \* Tube feeding is better than IV drip in conscious children who are not vomiting, as there is less hazard of overload.
- \* Give solutions similar in electrolytes to the fluid lost.
- \* Give the highest possible energy content (but no sugar in diabetic coma).
- \* Half strength Darrow's dextrose solution is suitable for maintenance in children without dehydration and to replace fluid in most children dehydrated by diarrhoea and vomiting (though potassium is too high

5 Appendices - Fluid Balance

for rapid replacement in the child shocked from cholera).

- \* If no urine is produced, replace lost fluid and electrolytes, but do not give potassium.

**Special Situations**

- \* Give NEONATES 5% or 10% dextrose. (The electrolyte content of half strength Darrow's is too high).
- \* In PYLORIC STENOSIS use an equal mixture of normal saline and 5% dextrose (i.e. saline diluted to half strength), with KCl added to a concentration of 5 mmol/l. Do not use half strength Darrow's dextrose or normal saline alone.
- \* In ANURIA use 5% dextrose alone for maintenance (no potassium!)
- \* In CHOLERA give Ringer's lactate when fluid is needed fast IV. (Half strength Darrow's has too much potassium). Because of electrolyte problems, change to ORS soon.
- \* In MALNUTRITION try to avoid IV fluids and give special ReSoMal solution orally.

Fluids can be given by mouth, by naso-gastric tube (NGT), intravenously [5.4.2], intraperitoneally [5.4.3], or into the bone marrow [5.4.3]. Fluid replacement by mouth or NGT is preferable, because it is cheaper and the risk of overhydration is less. Intravenous fluids have to be monitored carefully to avoid overhydration and dehydration. If prescribing an intravenous drip, make sure you have enough nursing staff to cope.

**How IV Fluids Differ (contents per litre)**

	<u>Na</u>	<u>K</u>	<u>Ca</u>	<u>Cl</u>	<u>Lactate</u>	<u>Dextrose/l</u>	<u>KCal</u>
<i>Half Darrow's dextrose</i>	61	17	0	51	27	50 gm	200
<i>Ringers' Lactate</i>	130	5	2	111	29	0	0
<i>Normal Saline 0.9%</i>	154	0	0	154	0	0	0
<i>Half Normal Saline 0.45%</i>	77	0	0	77	0	0	0
<i>5% Dextrose</i>	0	0	0	0	0	50 gm	200
<i>Breast milk (mature)</i>		6.5	15	35	12	70 gm	700

### **Risks of IV Fluid Treatment**

- \* **INFECTION:** infection starting at the drip site may lead to septicaemia, and then even osteomyelitis.
- \* **ELECTROLYTE IMBALANCE:** most commonly hypo- or hyperkalaemia from too rapid infusion of potassium free or rich fluid.
- \* **OVERLOAD:** too rapid infusion can cause heart failure (rapid respirations and heart rate, enlarged liver, gallop rhythm), or oedema (commonly seen around the eyes in patients lying down). This is particularly important in our setting, where staff shortages lead to poor infusion supervision. Therefore carefully prescribe both the volume and the time over which it is to be given.
- \* **HYPOGLYCAEMIA:** prolonged IV fluids given without glucose or food.

### **How to Control Fluid Intake:**

- \* Calculate the total fluid needs.
- \* Subtract volume given orally and give only remainder IV.
- \* Mark initial fluid level on bottle on plaster strip, writing in start time. Also mark target level and time.
- \* Teach the guardians about the target level.
- \* Instruct the guardians to call for help if level is falling too fast or slowly.
- \* Special infusion sets with burettes are very helpful, if available, especially in babies.

## **5.6 Special Feeds**

If a refrigerator is available, prepare for 24 hours and store in refrigerator. Otherwise feeds must be made up every 6 hours.

F75 is the name given to a formula providing a suitable combination of calories, carbohydrate, fat and protein for starting feeding severely malnourished children, and F100 for feeding malnourished children once past the initial period and able to tolerate more food (when oedema has gone in kwashiorkor and weight gain has started in marasmic children). When F-75 or F-100 is not available in premixed form, blend the solid components, mineral solution and oil into a paste, then slowly add warm water to make the mixtures shown on the next page.

## 5 Appendices - Special Feeds

<b>F-75</b>	Dried skimmed milk	25 g
	Sugar	100 g
	Vegetable oil	27 g
	Electrolyte/mineral solution	20 ml
	Made up with water to	1000 ml

<b>F-100</b>	Dried skimmed milk	80 g
	Sugar	50 g
	Vegetable oil	60 g
	Electrolyte/mineral solution	20 ml
	Made up with water to	1000 ml

### **High Energy Phala**

- 50 g sugar (4 tablespoons)
- 30 g vegetable oil (2 tablespoons)
- 50 g cereal flour (7 tablespoons)
- 120 g milk powder (16 tablespoons)
- Add to one litre of water and cook

### **Chiponde (trade name high energy food)**

- 68 g (26.1%) peanut paste
- 65 g (25.5%) dry skim milk
- 70 g (26.8%) sugar
- 52 g (20.1%) oil
- 3.64 g (1%) vitamins, minerals
- in one 260 g bottle (1430 kcal/bottle)

### **Lactose-free High Energy Feed (Milk-free)**

- 5 eggs
- 100 g glucose or sugar (8 tablespoons)
- 30 ml vegetable oil (eg. groundnut oil) (2 tablespoons)
- Add water to make one litre

\* When preparing larger quantities it is best to make special measuring containers, rather than weighing or using spoons, to save time.

\* Oil can be added to other feeds to increase calorie content.

## 5.7 Treatment Room Check-List

IV Therapy and Sampling		Tick
* IV Fluids	Half Darrow's and 5% dextrose <sup>1</sup>	[ ]
	5% dextrose	[ ]
	Ringer's lactate <sup>2</sup>	[ ]
	N saline <sup>3</sup>	[ ]
* IV fluid and blood giving sets		[ ]
* Butterfly needles	Size 21	[ ]
	Size 23	[ ]
	Size 25	[ ]
* IV cannulas	Sizes 18, 20, 22, 24	[ ]
* Needles	Gauge 19	[ ]
	Gauge 21	[ ]
	Gauge 23	[ ]
* Trochar and cannula	(for marrow or IO infusions [5.4.3])	[ ]
* Disposable Syringes	5 ml, 10 ml and 20 ml for aspirations of effusions, ascites	[ ]
* Specimen Bottles	Plain tubes for blood x-match, U&E	[ ]
	EDTA tubes for FBC	[ ]
	Sterile bottles for CSF and cultures	[ ]
* Dipsticks	Dextrostix (or similar)	[ ]
	Urine sticks (for blood and protein)	[ ]
* Miscellaneous	Cotton wool and spirit (or Savlon)	[ ]
	Adhesive tape	[ ]
	Blood lancets and clean glass slides	[ ]
	Laboratory forms	[ ]
Dressing Trolley	Dressing packs	[ ]
	Gauze swabs and adhesive tape	[ ]
	GV paint	[ ]
	Eusol	[ ]
	Mercurochrome	[ ]
Emergency Drugs (2 ampoules of each)	Paraldehyde	[ ]
	Phenobarbitone	[ ]
	Diazepam	[ ]
	Frusemide	[ ]

<sup>1</sup> Main all-purpose IV fluid[5.5]

<sup>2</sup> For cholera & hyperglycaemic diabetic coma.

<sup>3</sup> For priming IV giving sets for blood transfusion & for hyperglycaemic diabetic coma.

## 5 Appendices - Treatment Room Check List

Quinine	[ ]
Dexamethasone	[ ]
50% dextrose	[ ]
NaHCO <sub>3</sub>	[ ]
Adrenaline 1:1000	[ ]
2% and 1% Lidocaine	[ ]
Atropine	[ ]
Emergency Equipment	
Suction machine (foot or electric)	[ ]
Ambu bag and masks	[ ]
Suction catheters (Sizes F10, F12)	[ ]
Airways (Sizes 00, 0, 1, 2)	[ ]
Oxygen catheters & NGTs (F8, F6)	[ ]
Oxygen supply (Concentrator or cylinder)	[ ]
Laryngoscope + WORKING batteries	[ ]
ET tubes (Sizes 2 - 7)	[ ]

### **Note:**

#### **Resuscitation**

- Drugs and equipment should be checked DAILY.
- Change water in oxygen humidifiers DAILY to prevent infection spread.
- Staff need regular review sessions to remain familiar with emergency equipment and resuscitation techniques (see [2.3.1] for details).

#### **Tidiness**

- Correct disposal of sharps and general tidiness helps prevent accidents and needle stick injuries - PREVENT HIV INFECTION IN THE WORKPLACE. Make your own catheters.
- IV giving sets used for clear fluids can be saved to make suction catheters. Cut tubing 30 cms from rubber bung and attach rubber to suction machine.
- A butterfly used to give clear fluids can be saved to make an oxygen catheter. Cut off needle and discard (safely!) and attach to oxygen outlet.

#### **Economy - Don't waste limited resources**

- Don't use butterfly needles to take blood.
- Use minimum amount necessary of cotton wool, spirit, adhesive tape etc.

## 5.8 Normal Laboratory Values

Alkaline phosphatase	< 650 U/l
Amylase (serum)	< 90 U/l
ASOT Latex agglutination	> 200 IU ASO/ml
Bilirubin (total)	< 1.0 mg/dl *
Bleeding time	2-7 minutes
Calcium	8.1-10.4 mg/dl *
Chloride	98-110 mg/dl
Cholesterol	< 180 mg/dl
Clotting time	5-8 minutes
Creatine kinase (CK)	24-170 U/l *
Creatinine	< 1.4 mg/dl *
ESR	< 13 mm/hour
Glucose	45-115 mg/dl
G6PD (serum)	< 0.18 mU/ml
G6PD (erythrocyte)	131+/-13 mU/109 RBCs
Osmolality (random urine)	50-1400 mosmol/kg H <sub>2</sub> O
Osmolality (serum)	275-295 mosmol/kg H <sub>2</sub> O
Phosphorus (inorganic)	4.0-7.0 mg/dl
Potassium	3.5-5.5 meq/l
Protein (Albumin)	3.5-5.0 g/dl
Protein (total)	6.0-8.0 g/dl
SGOT	< 37 U/l
SGPT	< 42 U/l
Sodium	135-145 meq/l
Triglycerides	< 150 mg/dl
Urea	10-50 mg/dl
Uric acid	2.4-5.7 mg/dl

### Note:

- 1. Normal values for items marked (\*) are higher in neonates.**
- 2. Normal values may vary with different testing kits.**
- 3. For G6PD test both clotted and EDTA samples are needed.**



## 5.9 Useful addresses

- Ministry of Health, Box 30377, Lilongwe 3. Tel 01 789400  
National AIDS Control Programme  
- Box 30622, Lilongwe 3. Tel 01770022/210/215  
Fax 01 776249
- Malawi College of Health Sciences  
- Box 30368, Lilongwe 3. Tel 01 756777/752028
- Medical Association of Malawi (MAM), Box 30567, Lilongwe 3.  
Medical Council of Malawi  
- Box 30787, Lilongwe 3. Tel 01 727048/727255
- Christian Health Association of Malawi (CHAM)  
- Box 30378, Lilongwe 3. Tel 01 775404/775180
- Nguludi school for the deaf, St. Joseph's Hospital Tel 01 916037  
- Box 5505, Nguludi, Limbe. Tel 01 651420
- EPI Programme, MOH, P.O.Box 30377, Lilongwe, 3 Tel 01 725637
- Malawi Against Polio (MAP)  
- Blantyre Unit, Box 256, Blantyre. Tel 01 677951/674634  
- Lilongwe Unit, Box 30333, Lilongwe 3. Tel 01 727317
- Cheshire homes (Malawi) - Office Tel 01 836329  
- Rehabilitation Centre Tel 01 820810
- Malawi Council for the Handicapped (MACOHA), Box 5971, Limbe.  
All Disease Control Programmes (not AIDS), Community Health Services  
Unit, Box 30377, Lilongwe 3. Tel 01 757506
- Malawi Blood Transfusion Service  
Galaxy House, Glyn Jones/St George Street, Box 2681, Blantyre  
Tel 01 822801/650
- Armando Ramos Building, Paul Kagame Road, Box 1699, Lilongwe  
Tel 01 753819/828
- 24 hour hot lines - 08 206931 (Blantyre), 08 224680 (Lilongwe)  
United Nations Children's Fund (UNICEF)  
- Box 30375, Lilongwe 3. Tel 01 770788/770775  
01 770232/770237
- World Health Organisation (WHO) Tel 01 772755/772350
- Zonal Health Support Offices  
- South West, Box 3, Blantyre Tel 01 878409/878525  
South East, Box 216, Zomba Tel 01 567160  
Central East, Box 53, Salima Tel 01 262499  
Central West, Box 30377, Lilongwe 3 Tel 08 366786  
North, P/Bag 1, Mzuzu Tel 01 312911

# 6

## DRUG LIST

This Drug List is intended as a summary guide to drug dosage and usage. Detailed information should be sought from pharmacology texts and drug literature inserts.

Drugs are referred to by their generic names. Trade names are also included for those drugs that are better known by their trade name, though this in no way recommends the use of one brand over another.

Whereas every attempt has been made to check carefully drug doses and availability, the authors and publishers cannot take responsibility for any inaccuracies.

For further details see Malawi National Formulary (1991).

**Note:** Drugs are coded according to where they are available, at Health Centre (H), District Hospital (D) or Central Hospital (C).

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILTY & REMARKS
ABACAVIR (ABC)	C	AIDS resistant to first line treatment. Target dose is 8 mg/kg/dose twice daily.	O	300 mg tablets. Must only be given in combination and on advice of specialist paediatrician. May cause gastrointestinal upsets. Danger of skin rashes which can give Stevens-Johnson syndrome. Also fatigue, sleep disturbance.
ACICLOVIR	C	Herpes simplex, Herpes zoster, severe chickenpox. For Herpes zoster or chickenpox in the immunocompromised in whom chickenpox can be very severe, give 20 mg/kg/dose 4 times daily for five days. For Herpes simplex half that dose is enough.	O	200 mg tablets. To be effective treatment must be started at the onset of the illness, so in practice will be given to known immunocompromised patients already under care who are infected in an outbreak of chickenpox, or as soon as Herpes zoster appears.
ADRENALINE	H	Anaphylactic, sometimes septic shock, severe bee stings. Start with 0.01 ml/kg/dose (maximum 0.3 ml) IM or subcut. Recurrent or prolonged shock may need IV adrenaline 1 to 10 ml diluted in 1000 ml normal saline. Can also be used in asthma and severe croup.	I.M. subcut or I.V.	Ampoules of 1 ml 1/1000 concentration (1 mg in 1 ml) If used IV adrenaline must be diluted at least 10 times with saline or 5% dextrose and be given slowly under careful monitoring of heart rate, blood pressure and urine output, preferably in an ICU.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
ALBENDAZOLE (Zantel)	H	Ascaris, threadworm, hookworm < 2 yrs 200 mg stat > 2 yrs 400 mg stat. Strongyloides > 2 yrs 400 mg 12 hrly for 5 days	O	200 mg tablets Give once in 6 months
ALLOPURINOL	C	Prevention of tumour lysis syndrome in extensive Burkitt's lymphoma to be given before cyclophosphamide, 5-10 mg/kg/dose b.d. for at least 24 hours before chemotherapy and 4 days after.	O	100 mg tablets. Can cause rashes, Stevens-Johnson syndrome.
AMIKACIN SULPHATE	C	Drug resistant tuberculosis, Dose 15 mg/kg/dose once daily.	IM	1 g vials, Caution if kidney damage present
AMINOPHYLLINE	C	Neonatal apnoea: very preterm neonates 6 mg/kg stat and 1.3 mg/kg/dose 6 hrly Asthma > 1 year 5 mg/kg/dose 6 hrly	O, slow I.V.	50 mg/5 ml suspension made to special order for neonates. 100 mg tablets, IV 25 mg/ml (10 ml ampoule). Can cause tachycardia, nausea, vomiting, diarrhoea
AMITRIPTYLINE	H	Mostly used for depression, but in paediatrics for neuropathic pain induced by anti-retroviral treatment. Dose 0.2-0.5 mg/kg/dose nightly	O	Tablets of 10 or 25 mg. May cause drowsiness, heart arrhythmia.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
AMODIAQUINE		Antimalarial in combination with artesunate for second line treatment if asexual malaria parasites persist in the blood after 48 hours. Dose 10 mg/kg/dose (maximum dose 4 tablets) daily for 3 days.	O	Tablets of 100 mg amodiaquine.
AMOXICILLIN	H	Sepsis all ages 15 mg/kg/dose 8 hrly. Typhoid all ages 30 mg/kg/dose 8 hrly (but not preferred treatment for typhoid).	O	250 mg tablets (film coated) or capsules, or 125 mg/5 ml paediatric suspension (when reconstituted with water). Better absorbed orally than ampicillin. Not penicillinase resistant.
AMPICILLIN	D	Sepsis all ages 12.5-25 mg/kg/dose 6 hrly. Meningitis all ages 50 mg/kg/dose 6 hrly.	I.V., I.M.	250 mg vial for injection. Causes rash with glandular fever. Not penicillinase resistant.
ARTESUNATE		Antimalarial in combination with amodiaquine for second line treatment if asexual malaria parasites persist in the blood after 48 hours. Dose 4 mg/kg/dose (maximum dose 4 tablets) daily for 3 days.	O	Tablets of 40 mg artesunate.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
ASPIRIN	H	Analgesic & antipyretic 10 mg/kg/dose 4 or 6 hrly. Antirheumatic up to 20 mg/kg/dose 4 hrly.	O	300 mg (adult) or 75 mg (paediatric) tablets. Can cause gastric erosions. May be associated with Reye syndrome in children with chickenpox or influenza. Now not used in children under 12, but safe in older children, and especially useful in juvenile rheumatoid arthritis and rheumatic carditis when more expensive alternatives like ibuprofen not available.
ATROPINE	H	Acetylcholine antagonist. In organophosphate poisoning 2 mg every 20-30 minutes. Premedication for anaesthetic, pericardial tap 20 micrograms/kg.	I.V., sub-cut	ampoules of 600 micrograms (0.6 mg).
AZITHROMYCIN (Zithromax)	C	Broad spectrum macrolide antibiotic for respiratory and soft tissue infections (but not intestinal or urinary). Streptococcal tonsillitis, especially for those allergic to penicillin. Antibiotic of choice in trachoma. Dose 10 mg/kg/day in single dose for 3 days.	O	250 mg capsules so unsuitable for children under about 20 kg.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
BENZATHINE PENICILLIN	H	Syphilis neonates 50,000 units/kg stat. Rheumatic fever prophylaxis < 30 kg - 600,000 units monthly; > 30 kg - 1.2 MU monthly. Same dose stat can be used for streptococcal tonsillitis.	I.M.	2.4 MU in 5 ml vial. Can cause allergy, anaphylaxis.
BENZYL PENICILLIN (Crystalline peni- cillin, X-penicillin)	H	Bacterial infections & serious sepsis. All ages 50,000 units/kg/dose, 6 hrly. Meningitis 50,000 units/kg/dose 4 hrly IV. In first week of life can be given b.d.	I.V., I.M.	500,000 units, 1 MU or 5 MU vial. Can cause allergy and occasionally anaphylaxis.
CAPREOMYCIN SULPHATE	C	Drug resistant tuberculosis. See product information for dose.	I.M.	1 g vials
CAPTOPRIL	D	Heart failure, hypertension. Test dose of 0.1 mg/kg then 0.1 to 0.3 mg/kg/dose two or three times daily.	O	Tablets of 12.5 mg. An ACE (aldosterone converting enzyme) inhibitor. Start treatment cautiously in hospital with fre- quent blood pressure checks, especially if diuretics are being given. May be used in acute glomerulonephritis with hyper- tension but not in renovascular disease.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
CARBAMAZEPINE (Tegretol)	D	Epilepsy 5-10 mg/kg/dose 12 hrly. Neuropathic pain 2 mg/kg/dose b.d.	0	200 mg tablets
CEFOTAXIME	C	Severe infections, especially meningitis 1st week of life 25 mg/kg/dose b.d. 1-4 weeks 25 mg/kg/dose 8 hourly, > 4 weeks old 50 mg/kg/dose 8 hourly.	I.V., I.M.	Vials of 500 mg Expensive so usually reserved for severe infections, especially meningitis not responding to first choice antibiotics. Broad spectrum cephalosporin active against many gram negative organisms, not so effective against staphylococci.
CEFTRIAXONE	C	Severe infections, especially meningitis. Avoid under 1 month, especially in presence of jaundice. > 1 month old 50-80 mg/kg/dose once daily.	deep I.M., I.V. slow- ly or by drip	Vials of 250 mg. Expensive so usually kept for severe infections, especially meningitis not responding to first choice antibiotics. Broad spectrum cephalosporin active against many gram negative organisms but not so effective against staphylococci. More convenient than cefotaxime because given once daily.



DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
CEFUROXIME	C	Respiratory and staphylococcal infections. Aged 3 months to 2 years 10 mg/kg/dose b.d. Over 2 years 15 mg/kg/dose b.d. to maximum of 250 mg b.d. (except in very severe chest infections when dose may be 500 mg b.d.)	O	Oral cephalosporin more effective against gram positive bacteria and H. influenzae, but less effective against other gram negative bacteria.
CHLORAMPHENI-COL	D	Sepsis including meningitis < 2 weeks 6 mg/kg/dose 6 hrly, 2-4 weeks 12.5 mg/kg/dose 6 hrly, > 1 month 25 mg/kg/dose 6 hrly.	O, I.M. I.V.	125 mg/5 ml suspension, 250 mg tablets and 1 G vials. Can cause bone marrow suppression - check haemoglobin if course is prolonged. Risk of grey syndrome (shock) in overdose in neonates.
CHLOROQUINE	D	Prevention of malaria in combination with proguanil in a few selected categories of patients such as expatriates from non-malarial countries, sicklers, patients without spleens or immuno-suppressed by drugs, or with frequent recurrent febrile convulsions. Dosage 5 mg/kg/dose weekly. (When used in treatment the dosage is higher, but malaria parasite resistance has lead to its discontinuation).	O	Tablets with 150 mg chloroquine base. Note that some 40% of black skinned patients develop some degree of itch with chloroquine.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
CHLORPHENIRAMINE (Piriton)	H	Allergy, itching all ages 0.1 mg/kg/dose 6 to 12 hrly	0	4 mg tablets. Can cause drowsiness
CHLORPROMAZINE (Largactil)	H	Sedation: 0.5 mg/kg/dose 6 hrly	0	25 mg sugar coated tablets and 25 mg/ml (2 ml ampoule). Overdose may produce Parkinsonian syndrome.
CIPROFLOXACIN	D	Shigella dysentery, chloramphenicol resistant typhoid, urinary infections, pneumonia NOT caused by Streptococcus pneumoniae. 5-10 mg/kg/dose in 2 doses, but for dysentery 20 mg/kg as a single dose repeated twice at 24 hour intervals if bloody diarrhoea persists.	0	250 mg and 500 mg tablets. The scheme of daily observed treatment for dysentery is essential to avoid misuse of treatment leading to drug resistance developing.
COTRIMOXAZOLE (Bactrim, Septrin)	H	Respiratory and urinary infections. In neonates (only after any jaundice passed) 18 mg/kg/dose stat of the combined drug & then 6 mg/kg/dose 12 hrly of the combined drug. In older children 18-30 mg/kg/dose 12 hrly of the combined drug. For Pneumocystis carinii pneumonia (PCP) treatment 60 mg/kg/dose b.d. for 3 weeks followed for PCP prevention with 30-50 mg/kg/dose daily for life.	0	Cotrimoxazole contains 1 part of trimethoprim and 5 of sulphamethoxazole i.e. 480 mg combined drug contains 80 mg trimethoprim. 480 mg adult tablets, 240 mg paediatric tablets and 48 mg/ml of syrup. Can cause bone marrow suppression, rashes, erythema multiforme, folate deficiency.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
CYCLOPHOSPHAMIDE	C	Burkitt's lymphoma 40 mg/kg/dose as bolus into running IV drip, after rapid infusion of 20-30 ml/kg fluid. Hodgkin's disease same dose. Repeatedly relapsing nephrotic syndrome 2.5 mg/kg/day orally, up to 2 months.	I.V., O	200 mg and 500 mg vials and 50 mg tablets. Can cause haemorrhagic cystitis, vomiting and diarrhoea, bone marrow suppression, loss of hair. Dose regulated by haematological tests.
CYCLOSERINE	C	Drug resistant tuberculosis. 5 mg/kg/dose twice daily.	O	250 mg tablets
DAPSONE		Leprosy (in combination with rifampicin, clofazimine) and for prophylaxis of pneumocystis carinii pneumonia (PCP) when cotrimoxazole not tolerated. 1-2 mg/kg/dose daily for 6 months in paucibacillary leprosy, at least 2 years in multibacillary leprosy, for life for PCP prophylaxis.	O	100 mg tablets. Can cause haemolysis, skin rash, and in overdose methaemoglobinaemia (looks like cyanosis).
DEXAMETHASONE	D	Laryngitis 0.3 mg/kg/dose repeated 6 hrly up to 24 hours.	I.V., I.M.	4 mg/ml (5 ml vial). Can cause immune suppression, fluid retention, hypertension, mental disturbance.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
DIAZEPAM (Valium)	H	Convulsions 0.2-0.3 mg/kg/dose IV slowly, repeated 1-4 hrly as needed. Rectally give 0.5 mg/kg/dose.	slow I.V., rec- tally	5 mg/ml (2 ml ampoule). Causes respira- tory depression in overdose.
DIDANOSINE (ddI)	C	AIDS resistant to first line treatment. Target dose 3-4 mg/kg/dose daily, taken on an empty stomach.	0	300 mg tablets. Must only be given in combination and on advice of specialist paediatrician. Can cause nausea, diarrhoea, neuropathy, pancreatitis.
DIGOXIN	C	Cardiac failure. Loading dose: 0.005 mg/kg/dose 8 hrly for 3 doses. Maintenance dose: 0.01 mg/kg/dose once daily.	0	62.5 micrograms (0.0625 mg) or 250 micrograms (0.25 mg) tablets, 50 mcg (0.05 mg)/ml elixir. Can cause nausea, vomiting, bradycardia, arrhythmias.
DOXYCYCLINE	H	Cholera 5 mg/kg/dose stat for children over 12. Also can be given in resistant malaria with quinine (same dose daily for 7 days).	0	100 mg tablets or capsules. Can dis- colour unerupted teeth so best avoided in young children.
EFAVIRENZ (EFV)	C	AIDS resistant to first line treatment. Target dose 12 mg/kg/dose daily.	0	300 mg tablets. Only give combined on advice of paediatrician. Can cause rash, mental disturbance. (Teratogenic so avoid in pregnant women).

DRUG	CODE	USE AND CAPS	ROUTE	AVAILABILITY & REMARKS
ERYTHROMYCIN	H	Respiratory infections all ages 7.5-12.5 mg/kg/dose 6 hrly. For 3 days in cholera.	0	250 mg tablets, 125 mg/5 ml suspension
ETHAMBUTOL	D	Tuberculosis 15 mg/kg/dose once daily combined with rifampicin, INH & Z for 2 months.	0	400 mg and 100 mg tablets. Can cause optic neuritis (impairment of vision).
ETHAMBUTOL/ ISONIAZID (FATOL)	D	Tuberculosis dose as for ethambutol, but note small children will need extra isoniazid.	0	400 mg ethambutol and 100 mg isoniazid tablets. Dangers as for ethambutol and isoniazid.
ETHIONAMIDE	C	Drug resistant TB, given in combination.	0	125 mg and 250 mg tablets.
ETHOSUXIMIDE	C	Petit Mal epilepsy, starting with 500 mg nightly; preschool start with 250 mg nightly	0	250 mg capsules. Can rarely depress bone marrow.
FACTOR VIII	C	For treatment of haemophilia from Red Cross transfusion service. Dose by paediatrician.	I.V.	I.V. infusion packs.
FERROUS SULPHATE	H	Prevention: 1 mg elemental iron/kg/day Treatment: 0-2 years - 6 mg iron/kg/day (1 ml/kg/dose syrup 12 hrly); 3-7 years - 1/2 adult tablet 12 hrly; > 7 yrs - 1 adult tablet 12 hrly.	0	60 mg ferrous sulphate in 5 ml syrup contains 12 mg elemental iron in 5 ml. 200 mg tablets (coated) contain 60 mg elemental iron.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILTY & REMARKS
FLUCLOXACILLIN	D	Staphylococcal sepsis all ages 12.5-25 mg/kg/dose 6 hrly.	O, I.M., I.V.	25 mg /5 ml syrup, 250 capsules, 250 mg vials. Only for use against penicillinase producing staphylococci. Beware penicillin allergy.
FLUCONAZOLE	D	For treatment of cryptococcal meningitis and invasive candidiasis in single daily dose of 6-12 mg/kg/dose	O	200 mg capsule. 2 mg/ml in 25 ml infusion flask. Can cause nausea, headache, skin rash.
FOLIC ACID	D	Macrocytic anaemia, PEM, Sickle cell disease < 2 yrs - half tablet daily: > 2 yrs - 1 tablet daily. (But thrice weekly is enough).	O	5 mg tablets. Sickle cell disease need treatment for life, PEM for 1 dose, and anaemia for 3 weeks.
FRUSEMIDE (Lasix)	H	Diuretic orally all ages 1-2 mg/kg/dose once daily or 12 hrly. I.V. or I.M. 0.5-1.0 mg/kg stat.	O, I.M. slow I.V.	40 mg tablets and 10 mg/ml (2 ml ampoule). Can cause hypokalaemia or dehydration.
GENTAMICIN	D	Serious gram negative sepsis < 7 days old - 5 mg/kg/dose daily ; over 7 days old - 7.5 mg/kg/dose daily.	I.M., I.V.	10 mg/ml and 40 mg/ml (2 ml vials). Can cause nerve deafness, and renal damage. Avoid with renal impairment.
GRISEOFULVIN	D	Tinea capitis 10 mg/kg/dose daily for 2 week	O	125 mg tablets.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
HALOPERIDOL (Serenace)	C	Rheumatic chorea 0.025-0.05 mg/kg/dose daily.	0	2 mg tablets. Toxic effects include abnormal postures.
HYDROCHLOROTHIAZIDE	H	For mild hypertension 25 to 50 mg once daily.	0	50 mg tablets. Can cause hypokalaemia. (low blood potassium).
IBUPROFEN	D	Pain or fever relief. 5-7 mg/kg/dose 6 hrly.	0	200 mg tablets.
INDOMETHACIN (Indocid)	C	Patent Ductus Arteriosus in neonate 0.2 mg/kg/dose 8 hrly for 3 doses.	0	Specially prepared suspension. Can cause oliguria, anuria and gastrointestinal bleeding.
INSULIN PROTAPHANE	D	Diabetes mellitus once or twice daily, dose depending on response.	sub cut. upper thigh, lower abdo.	10 ml vials of 100 units/ml. Needs to be kept cool (best in a fridge).
INSULIN SOLUBLE	D	Diabetes mellitus 4 - 6 hourly or twice daily combined with protaphane insulin, dose depending on response.	sub cut, or I.V.	10 ml vials of 100 units/ml. Needs to be kept cool (best in a fridge).

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILTY & REMARKS
ISONIAZID (INH)	D	Tuberculosis 10-20 mg/kg/dose once a day. Must be given in combination for treatment, but may be used alone in prophylaxis.	O	300 mg and 100 mg tablets. Can cause hepatitis, & peripheral neuritis, pellagra-like syndrome (treated with pyridoxine).
IVERMECTIN	D	Onchocerciasis 150 micrograms/kg stat, fasting. Strongyloides 150-200 micrograms/kg/dose once.	O	Tablets 3 mg.
KANAMYCIN		Drug resistant tuberculosis 5 mg/kg/dose 8 hrly.	I.M., I.V.	250 mg, 500 mg and 1 G vials. Can cause nerve deafness and renal damage. Avoid with renal impairment.
KETAMINE	C	Anaesthesia 2-4 mg/kg given IV slowly over at least 1 minute, in brief procedures such as fine needle biopsy, bone marrow biopsy, administration of intrathecal chemotherapy. 5-10 mg/kg stat if used I.M.	I.V., I.M.	10 ml vials of 50 mg/ml. Avoid if child is hypertensive. Confusional states during recovery may need diazepam sedation.
KETOCONAZOLE	D	For fungal infections and candidiasis 3 mg/kg/dose daily.	O	200 mg tablets. Can cause liver damage, interact with rifampicin, isoniazid, nevirapine, artemether.



DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
LAMIVUDINE (3TC)	D	<p>Combined with stavudine and nevirapine as Triomune used in first line treatment of AIDS. Also available combined with stavudine for use with efavirenz when nevirapine not tolerated.</p> <p>Combined with zidovudine in post exposure prophylaxis for defiled children [4.20.4].</p>	O	<p>In children use 150 mg lamivudine with 30 mg stavudine with/without 200 mg nevirapine per tablet. For first two weeks of treatment give Triomune in the morning lamivudine with stavudine in the evening. Thereafter give Triomune twice daily. See section on AIDS. Lamivudine may cause nausea, headache, fatigue, muscle pain and less often lactic acidosis, pancreatitis.</p>
LEVOTHYROXINE	C	<p>Hypothyroidism, cretinism. Start with 5 micrograms/kg/dose daily, increase by 25 micrograms every 2-4 weeks till mild toxic effects, then reduce one step.</p>	O	<p>Tablet of 25 or 50 micrograms. Overdose causes tachycardia, increased appetite, weight loss, tremor.</p>
LIDOCAINE (Lignocaine)	D	<p>Local anaesthetic. Maximum dose for a child 3 mg/kg.</p>	intra- der- mal, sub cut	<p>1% solution for injection (10 mg/ml). Avoid intravascular injection which can lead to convulsions, cardiac arrest.</p>

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
LOPINOVIR/RITON- AVIR (LPV/r) (Kaletra)	C	Protease inhibitor recommended for second line treatment of AIDS in children combined with abacavir and didanosine. Dosage aim at 8 mg/kg/dose b.d.	O	Capsules have 133 mg lopinavir and 33 mg ritonavir. Only give in combination and on advice of paediatrician. May cause nausea, pancreatitis, neuropathy, lipodystrophy. Interacts with rifampicin.
LUMEFANTRINE/ ARTEMETHER (LA)	H	Antimalarial for first line treatment. Dose aim at 12-18 mg/kg/dose twice daily for 3 days. Maximum dose 4 tablets. Repeat any dose if child vomits within 1 hour.	O	Tablets of artemether 20 mg and lumefantrine 120 mg (combined 140 mg). Give with milk or other fatty food to improve absorption.
MEBENDAZOLE (Vermox)	H	Ascaris, Hookworm, Trichuris > 2 yrs 100 mg 12 hrly x 3 days; threadworm > 2 yrs 100 mg stat, repeated at 2 & 4 wks. Tape worm, strongyloides > 2 yrs 200 mg 12 hrly x 3 days.	O	100 mg tablets, 100 mg/5 ml syrup. Being discontinued.
MELARSOPROL	C	CNS trypanosomiasis. 3.6 mg/kg/dose daily for 3 days, repeated after 7 days for 3 courses.	I.V.	36 mg/ml (5 ml ampoule). Give with prednisolone first 2 weeks to reduce risk of encephalopathy. Can cause exfoliative dermatitis, kidney and liver damage. Check urine for protein before each dose.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
METHOTREXATE (MTX)	C	Burkitt's lymphoma (with cyclophosphamide and vincristine), 20 mg/sq.m.surface area per dose 2 weekly IV. IT to prevent CNS spread 12.5 mg/dose < 9, 15 mg/dose > 9 years diluted 1:9 with saline. Leukaemia in remission orally 15 mg/sq.m. weekly with 6-mercaptopurine.	I.V., I.T., 0	Vials of 50 mg in 2ml, tablets of 2.5 or 10 mg. Toxicity can be severe with bone marrow suppression, ulceration of the mouth, liver damage.
METHYLDOPA (Aldomet)	D	Hypertension all ages start at 5 mg/kg/dose 12 hrly - 6 hrly, increasing gradually if needed to 10 mg/kg/dose 6 hrly.	0	250 mg tablets. Titrate dose according to response. Can cause postural hypotension and drowsiness.
METRONIDAZOLE (Flagyl)	H	Anaerobic sepsis 7.5 mg/kg/dose 8 hrly for 7 days, giardiasis 7.5 mg/kg/dose 8 hrly for 3, amoebiasis 15 mg/kg/dose 8 hrly for 5 days.	0, I.V.	200 mg or 250 mg tablets, 5 mg/ml (100 ml IV infusion pack). Nausea, vomiting, metallic taste in mouth, alcohol reaction.
MORPHINE	D	For relief of severe pain, especially in malignancies. Under 1 year 0.1 mg/kg/dose, over 1 year 0.2-0.4 mg/kg/dose 6 or 4 hrly, as required to relieve pain.	0	Oral morphine solution strength varied by the pharmacist, so consult label. High doses can depress respirations, prolonged treatment causes constipation, addiction.
256 NALIDIXIC ACID	D	Shigella dysentery, resistant urinary infections. 12.5 mg/kg/dose 6 hrly for 5-7 days.	0	500 mg tablets. Can precipitate convulsion in epileptics, give rashes.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILTY & REMARKS
NEVIRAPINE (NVP)	D	Combined with lamivudine and stavudine as Triomune in first line treatment of AIDS. Available separately to prevent mother to baby transmission of HIV. Aim at 4mg/kg/dose once or twice a day.	O	Tablets for children: 200 mg nevirapine alone/with 150 mg lamivudine & 30 mg stavudine. In first 2 weeks give half dose to reduce skin rash risk. Also can give jaundice. Interacts with rifampicin, ketoconazole.
NITROFURANTOIN	D	Urinary tract infection 1.5 mg/kg/dose 6 hrly.	O	500 mg tablets. Take before meals.
PARACETAMOL (Panadol)	H	Analgesic and antipyretic 10-15 mg/kg/dose 4 or 6 hrly.	O	500 mg tablets or 120 mg/5 ml paediatric elixir. Causes liver damage in over-dosage.
PARALDEHYDE	H	Convulsions 0.2 ml (0.15-0.3 ml)/kg/dose up to every 15 minutes, up to 3 doses.	deep I.M.	5 ml and 10 ml ampoules. Use glass syringe, or if not available, use a plastic one quickly, (then discard it). Painful, can cause necrosis if given subcutaneously, can paralyse sciatic nerve.
PETHIDINE	D	Analgesic for severe pain 1 mg/kg/dose 4 to 6 hrly. (Dose range 0.5-2 mg/kg).	O, I.M., I.V.	50 mg tablets, 50 mg/ml injection (1 ml and 2 ml ampoules). Should not be given for more than 3 days. Causes nausea and vomiting, addictive.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
PHENOBARBITONE	D	Epilepsy 5-8 mg/kg/dose once daily, nocte Convulsions 10 mg/kg stat I.M. (used as a loading dose) but can give 15-20 mg/kg in neonate.	O, I.M.	30 mg tablets, 200 mg/ml in 1 ml ampoule. Can cause drowsiness but also restlessness in a few epileptic children, and allergic skin reactions.
PHENYTOIN (Epanutin)	D	Epilepsy 5-8 mg/kg/dose once daily, Neuropathic pain 2.5-5 mg/kg/dose b.d.	O	100 mg sugar-coated tablets. Can cause ataxia, slurred speech, drowsiness, skin reactions, bone marrow depression. Interacts with rifampicin, anti-fungals.
POTASSIUM CHLORIDE	D	Electrolyte for replacement or prevention of deficit, 1-2 mmol/kg/dose once or twice daily.	O, I.V. in drip	600 mg tablets (8 mmol potassium), 20 mmol/10ml injection (10 ml ampoules). Give orally with long term frusemide and hydrochlorothiazide, or to replace deficits (diabetic ketoacidosis, long diarrhoea). Avoid in renal failure. Dilute IV injection 50 times in a 5% dextrose or saline drip, with checks of electrolytes. High blood potassium can cause cardiac arrest.
PRAZIQUANTEL (Biltricide)	H	Schistosomiasis (S.H. & S.M.) 40 mg/kg/stat dose, to nearest $\frac{1}{4}$ tablet. Tapeworm 10 mg/kg/stat.	O	600 mg tablets Best given before sleep.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
PREDNISOLONE	D	Suppress immunity & inflammation all ages 2 mg/kg/dose once daily in the morning, usually reduced gradually later.	0	5 mg tablets. Can cause immune suppression, hypertension, fluid retention, Cushing's syndrome, adrenal suppression, growth retardation.
PROGUANIL (Paludrine)	D	Prevention of malaria < 1 year 1/4 tablet daily, 1-4 yrs 1/2 tablet daily, 5-8 yrs 1 tablet daily, 9-14 yrs 1 1/2 tablets daily, over 15 yrs 2 tablets daily. (Dose by weight 5 mg/kg daily).	0	Should be used only for certain groups, as expatriates from non-malarial countries, sicklers, patients without spleens or immuno-suppressed by drugs, or with frequent recurrent febrile convulsions. Can cause mouth ulcers. Preferably use along with weekly chloroquine.
PROMETHAZINE (Phenergan)	H	Allergy, vomiting 0.5 mg/kg/dose 12 hrly if > 6 months, or single dose nocte.	0	25 mg tablets, 5 mg/5 ml elixir. Mainly used in allergy, rarely in persistent vomiting. Can cause sedation and abnormal postures (dystonia).
PROPANOLOL (Inderal)	D	Hypertension and in Fallot's Tetralogy for angina, 0.25-1.0 mg/kg/dose 6 hrly, slowly increasing dose till adequate response.	0	40 mg tablets. Slows pulse and can make heart failure worse.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
PYRAZINAMIDE ("Z")	D	Tuberculosis 30 mg/kg/dose once daily. Must be given combined with other drugs.	0	400 mg tablets. Also 150 mg combined with rifampicin 60 mg & INH 30 mg in "RHZ". Can cause hepatitis, arthralgia.
PYRIDOXINE	D	For INH toxicity 10 mg/kg/dose once daily.	0	20 mg tablets.
QUININE	H	Cerebral malaria all ages 20 mg/kg in 10 ml/kg 5% Dextrose IV in 4 hrs, then 10 mg/kg 12 hrly, till taking well orally. Chloroquine and SP resistant malaria all ages 10 mg/kg/dose 8 hrly for 7 days	I.V., I.M., 0	300 mg tablets (dihydrochloride or sulphate), 75 mg/5 ml syrup, 300 mg/ml (1 ml ampoule) for infusion. Can cause tinnitus, deafness, headache, nausea, confusion. May cause hypoglycaemia when given IV without 5% dextrose in children
RIFABUTIN	C	For treatment of drug resistant tuberculosis in combination with other drugs under specialist supervision.	0	150 mg tablets Can cause jaundice, interact with other drugs.
RIFAMPICIN	H	Tuberculosis, Leprosy 10-15 mg/kg/dose once a day. Maximum 600 mg daily. Must be given in combination with other drugs.	0	150 mg or 300 mg capsules. Also 60 mg in combined RHZ tablets, and 150 mg in RHZE tablets. Rifampicin induces liver enzymes that accelerate metabolism of contraceptives, steroids and nevirapine.

DRUG	CODE	USE AND DOHSE	ROUTE	AVAILABILTY & REMARKS
RIFAMPICIN/ ISONIAZID (Rifinah)	H	Tuberculosis rifampicin 10-15 mg/kg/dose once a day. Maximum 600 mg daily. Isoniazid 10-20 mg/kg/dose once a day.	0	150 mg rifampicin with 100 mg or 75 mg isoniazid. Can cause jaundice. Rifampicin accelerates metabolism of contraceptives, steroids and nevirapine. INH can cause jaundice, (so avoid other drugs that can damage liver) and peripheral neuropathy, so give with pyridoxine when using other drugs that can cause peripheral neuropathy, such as d4T.
SALBUTAMOL (Ventolin)	H	Asthma > 1 yr old 0.1 mg/kg/dose 8 hrly Inhaler use 1-2 puffs 6 hrly. Nebuliser use 3-6 hrly as needed.	0	2 mg tablets, 2 mg/5 ml syrup, 100 microgram/puff inhaler (200 doses). 1 ml nebuliser solution. Can cause liquid tremor, restlessness.
SPIRONOLACTONE (Aldactone)	C	Cardiac failure, resistant oedema. 0.5-1.0 mg/kg/dose 8 hrly.	0	25 mg tablets.
STAVUDINE (d4T)	D	Combined with lamivudine and nevirapine as Triomune in first line AIDS treatment. Also available combined with lamivudine for use with efavirenz when nevirapine not tolerated.	0	In children use 30 mg d4T with 150mg lamivudine +/- 200 mg nevirapine. For first 2 weeks give Triomune in morning and d4T with 3TC in evening. Thereafter give Triomune b.d. See section on AIDS.



DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
STREPTOMYCIN	D	Tuberculosis all ages 20 mg/kg/dose once a day. Must be combined with other drugs.	I.M.	1 G vial. Can causes dizziness or deafness
SULFADOXINE-PYRIMETHAMINE (SP - Fansidar)	H	Malaria < 6 months: 1/4 tablet stat. 6 months-4 yrs: 1/2 tablet. 4-8 yrs: 1 tablet stat. 9-14 yrs: 2 tablets stat. over 15 yrs: 3 tablets stat. (Dose by weight 25 mg/kg of sulfadoxine stat.) No longer recommended for treatment of malaria in Malawi.	O	Contains pyrimethamine 25 mg & sulfadoxine 500 mg. Can cause marrow depression with prolonged treatment, and skin rashes. Not for prophylaxis. Now replaced as first line treatment by lumefantrine/artemether.
SURAMIN	D	Trypanosomiasis 20 mg/kg/dose every 5-7 days for 5 doses. Not effective without melarsoprol, if CNS is involved.	I.V.	1 G vial. Rare hypersensitivity shock occurs so give a 10 mg IV test dose. Check urine for proteinuria.
TENOFOVIR	C	For treatment of drug resistant AIDS in adults in combination (usually with AZT and 3TC) but not recommended for children.	O	245 mg tablets. Children specially liable to osteoporosis, also renal damage, lactic acidosis.
TRIOMUNE	D	Combination of lamivudine with stavudine and nevirapine used in first line treatment of AIDS.	O	Tablet contains 150 mg lamivudine and 200 mg nevirapine and 30 mg of stavudine. For dosages consult section on AIDs and for side effects and toxicity the individual drugs.

DRUG	CODE	USE AND DOSE	ROUTE	AVAILABILITY & REMARKS
VALPROATE (Epilim)	?C	Epilepsy, primary generalized, petit mal and atypical epilepsy, but less good in focal epilepsy. Dose initially 10 mg/kg/dose b.d., increased gradually if needed to maximum of 17.5 mg/kg/dose b.d. (or given t.i.d. if more convenient).	O	Tablets of 200 mg. Can cause nausea, increased appetite, occasionally liver damage. (Teratogenic so mothers must avoid).
VINCRIStINE	C	Treatment of Kaposi's sarcoma (KS), or (combined with cyclophosphamide and methotrexate) of Burkitt's lymphoma (BL) and Hodgkin's disease. 1.5 mg/sq.m.surface area/dose given weekly in KS and fortnightly in BL.	I.V.	1 or 2 ml vial of 1 mg/ml. Can cause neuropathy with paraesthesiae, abdominal pain, weakness.
VITAMIN A	H	Prophylaxis (measles and malnutrition) < 1 yr 100,000 units, > 1 yr 200,000 units single dose. Therapeutic (keratomalacia, xerophthalmia) give 3 doses on days 0, 1 and 8.	O	50,000 units and 200,000 units capsules.
ZIDOVUDINE (AZT)	D	Second line treatment of AIDS in adults. Not good in children because of risk of anaemia. If used, aim at 3-4 mg/kg/dose twice daily in combination (usually with lamivudine and tenofovir) but do not give with stavudine.	O	Capsules of 100 mg. Can cause anaemia, gastrointestinal upset, liver damage, neuropathy. (Note use combined with Lamivudine in post exposure prophylaxis after defilement [4.20.4]).



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